Participant Manual

Drug Recognition
Expert Course

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Acknowledgements

Preface Page

Session I: Introduction and Overview
  • Glossary of Terms

Session 2: Drugs in Society and in Vehicle Operation

Session 3: Development and Effectiveness of the DEC Program
  • Frye Decisions Regarding Admissibility of DRE Testimony
  • American Prosecutors Research Institute – HGN Case Law Summary
  • Scientific Publications and Research Reports Addressing Nystagmus

Session 4: Overview of the Drug Recognition Expert Procedures
  • Blank DRE Face Sheet

Session 5: Eye Examinations
  • Pupil Size Chart
  • SFST Proficiency Examination Form

Session 6: Physiology and Drugs: An Overview
  • Physiological Pursuit Questions

Session 7: Examinations of Vital Signs

Session 8: Demonstrations of the Evaluation Sequence

Session 9: Central Nervous System Depressants
  • Drug Exemplar Report – Ludes
  • Drug Exemplar Report – Downers
  • Drug Exemplar Report – Flynn

Session 10: Central Nervous System Stimulants
  • Drug Exemplar Report – Rocke
  • Drug Exemplar Report – Tweetker
  • Drug Exemplar Report – Crank
Session 11: Practice Eye Examinations
Session 12: Alcohol Workshop
Session 13: Physician’s Desk Reference and Other Reference Resources
Session 14: Hallucinogens
  • Drug Exemplar Report – Trumpet
  • Drug Exemplar Report – Tripp
  • Drug Exemplar Report – Flipping
Session 15: Practice: Test Interpretation
  • Drug Exemplar Report – Adams
  • Drug Exemplar Report – Baker
  • Drug Exemplar Report – Charles
  • Drug Exemplar Report – Dodge
  • Drug Exemplar Report – Edwards
Session 16: Dissociative Anesthetics
  • Drug Exemplar Report – Dexing
  • Drug Exemplar Report – Sherms
  • Drug Exemplar Report – Krystal
Session 17: Narcotic Analgesics
  • Drug Exemplar Report – Schmack
  • Drug Exemplar Report – Wynn
  • Drug Exemplar Report – Cottin
Session 18: Practice: Test Administration
  • Drug Exemplar Report – Martinez
  • Drug Exemplar Report – Groves
  • Drug Exemplar Report – Hatos
  • Drug Exemplar Report – Jackson
  • Drug Exemplar Report – Stevens
Mid-Course Review
Session 19: Inhalants
• Drug Exemplar Report – Whippets
• Drug Exemplar Report – Poppers
• Drug Exemplar Report – Huffer

Session 20: Practice: Vital Signs Examinations
• Vital Signs Examination Sheet

Session 21: Cannabis
• Drug Exemplar Report – Blunt
• Drug Exemplar Report – Toker
• Drug Exemplar Report – Duby

Session 22: Overview of Signs and Symptoms
• Comparison of DRE Symptomatology Sources

Session 23: Curriculum Vitae Preparation and Maintenance
• Two sample CV’s

Session 24: Drug Combinations
• Indicators Consistent With Drug Categories Chart
• Drug Combination Charts (5 total)

Session 25: Practice: Test Interpretation
• Drug Exemplar Report – Allen
• Drug Exemplar Report – Brown
• Drug Exemplar Report – Cole
• Drug Exemplar Report – Davis
• Drug Exemplar Report – Elliott

Session 26: Preparing the Narrative Report
• Drug Exemplar Report – Roach

Session 27: Practice: Test Administration

Session 28: Case Preparation and Testimony
• DRE Defense Cross Examination Questions

Review of the DRE School
• Self Quiz
• Answer Key to Self Quiz

Session 29: Classifying a Suspect (Role Play)
• Drug Exemplar Report – Alpha
• Drug Exemplar Report – Bravo
• Drug Exemplar Report – Charlie
• Drug Exemplar Report - Delta
• Drug Exemplar Report – Echo
• Drug Exemplar Report – Foxtrot
• Drug Exemplar Report – Golf
• Drug Exemplar Report – Hotel
• Drug Exemplar Report – India
• Drug Exemplar Report – Juliet
• Drug Exemplar Report – Kilo
• Drug Exemplar Report – Lima

Session 30: Transition to Certification Phase of Training
• DRE Log of Drug Influence Evaluations (Rolling Log)
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The Drug Recognition Expert course is a series of three training phases that, collectively, prepare police officers and other qualified persons to serve as drug recognition experts (DRE). Throughout this manual, the terms “drug recognition expert” and “DRE” are used to designate an individual who is specially trained and has continued training to conduct examinations of drug-impaired drivers. This training, developed as part of the Drug Evaluation and Classification Program (DECP) under the auspices and direction of the National Highway Traffic Safety Administration (NHTSA) and the International Association of Chiefs of Police (IACP) has experienced remarkable success since its inception in the 1980s.

As in any educational training program, an instruction manual is considered a “living document” that is subject to updates and changes based on advances in technology and science. A thorough review is made of information by the DECP Technical Advisory Panel (TAP) of the Highway Safety Committee of the IACP with contributions from many sources in health care science, toxicology, jurisprudence, and law enforcement. Based on this information, any appropriate revisions and modifications in background theory, facts, examination and decision making methods are made to improve the quality of the instruction as well as the standardization of guidelines for the implementation of the Drug Recognition Expert Training Curriculum. The reorganized manuals are then prepared and disseminated, both domestically and internationally, to the DECP state coordinators.

Changes will take effect 90 days after approval by the TAP, unless otherwise specified or when so designated by a state coordinator.
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Session 1

Introduction and Overview
A. Welcoming Remarks and Goals

Welcoming Remarks

Introductions - Representatives of Host Agencies and Other Dignitaries

Faculty Introductions
B. Housekeeping

Paperwork

Attendance
Attendance is mandatory at all sessions of this school.

Breaks

Facility

Interruptions
DRE Certification Phases

You have all completed the DRE Pre-School and we look forward to working with you to successfully complete phase two of the certification process. Upon completion of this course, you will be fully proficient in checking vital signs, conducting careful examinations of the eyes, administering divided attention tests and, in general, carrying out the procedural steps of the DRE’s job.

There is one essential learning experience that this classroom training cannot provide – the opportunity to practice examining subjects who are under the influence of drugs other than alcohol. For this reason, this classroom training only constitutes Phase II in the process of developing DRE skills. Phase III of the training (which commences upon the successful completion of this course) involves hands-on practice in an actual enforcement context, i.e. examining persons who are under the influence of drugs.

Although this DRE School will not conclude with the participant's immediate certification as a DRE, successful completion of this classroom training is highly important. No one can advance to Certification Training until they demonstrate a mastery of basic knowledge of drug categories and their effects on the human mind and body, and of the basic skills in administering and interpreting the examinations in the Drug Evaluation and Classification process.
The ultimate goal of the Drug Evaluation and Classification (DEC) program, and of this course of instruction, is to "help you prevent crashes, deaths and injuries caused by drug-impaired drivers".

No one knows precisely how many people operate motor vehicles while under the influence of drugs, or how many crashes, deaths and injuries these people cause. But even the most conservative estimates suggest that America's drug-impaired drivers kill thousands of people each year, and seriously injure tens of thousands of others. There are numerous studies that illustrate these facts.
Upon successfully completing this session participants will be able to:

- State the objectives and goals of the course.
- Outline the major course content.
- Outline the schedule of major course activities.
- Outline the Participant Manual content and organization.
- Recognize course administrative matters.

CONTENT SEGMENTS ................................................................. LEARNING ACTIVITIES

A. Welcoming Remarks and Goals ........................................... Instructor-Led Presentations
B. Housekeeping ................................................................. Participant-Led Presentations
C. Participant Introductions ..................................................... Knowledge Examination
D. Training Goals ................................................................. Reading Assignments
E. Training Objectives
F. Overview of Content and Schedule
G. Course Activities
H. Overview of Participant Manual
I. Glossary of Terms
J. Course Pre-Test Administration
Fact: A study in California of young male (15-34 years old) drivers killed in crashes in the early 1980’s revealed that more than half (51%) tested positive for drugs other than alcohol. The most prevalent drug (other than alcohol) was Cannabis at 37%. 30% of all cases had both alcohol and Cannabis.


**Maryland Shock Trauma Center study (1985 – 1986)**

- 32% of drivers treated at the Shock Trauma Center had used marijuana prior to their crashes.
University of Tennessee Study (1988)

- 40% of drivers receiving emergency treatment had used drugs prior to the crash.

Washington State (Schwilke, et al., 2006)

The results of tests of blood and/or urine from 370 fatally injured drivers revealed that:

- Marijuana was the most encountered drug (12%), followed by:
  - Benzodiazepines (5%) 
  - Cocaine (4.8%) 
  - Amphetamines (4.8%)
A 2009 study revealed 33% of fatally injured drivers who were tested showed positive for drugs other than alcohol. This 33% represented 18% of all fatally injured drivers. Some drivers were not tested for drugs.

**Drugged Driving Incidence**

- In 2010, more than 19% of high school seniors admitted driving under the influence of marijuana. *(SADD)*
- In 2010, 10.6 million people reported driving under the influence of an illicit drug during the past year. *(NSDUH)*

We can do something to remove drugged drivers from our roads.
The Drug Evaluation and Classification Program (DECP) is based on solid medical and scientific facts.

The validity of the DECP has been tested in carefully controlled research in both the laboratory and the field.

By enrolling in Drug Recognition Expert (DRE) training, you have become part of an elite international program. DREs form one of the tightest knit fraternities in law enforcement.

DREs from many agencies and from many parts of the country work closely together to share information and other resources, and to maintain the highest standards of quality.

C. Participant Introductions
D. Training Goals

The goals of the classroom training, from the viewpoint of the law enforcement agencies participating in it, are three fold:

1. To help police officers acquire the knowledge and skills needed to distinguish individuals under the influence of:
   - Alcohol
   - Other drugs
   - Combinations of alcohol and other drugs
   - Injury and illness
2. To enable police officers to identify the broad category or categories of drugs inducing the observable signs of impairment manifested by an individual.
3. To qualify police officers to progress to Certification Training.
E. Training Objectives

When you successfully complete this school, you will be able to:

- Describe the involvement of drugs in impaired driving incidents
- Name the seven categories of drugs and recognize their effects
- Describe and properly conduct the drug influence evaluation
- Document the results of the drug influence evaluation
- Properly interpret the results of the evaluation
- Prepare a narrative for the Drug Influence Report
- Discuss appropriate procedures for testifying in typical drug evaluation and classification cases
- Prepare and maintain a relevant and up-to-date Curriculum Vitae (C.V.)

Before you can be certified as a DRE, you will have to demonstrate that you can do each of these things.
F. Overview of Course Content and Schedule

The course will cover the following topics:

- Drugs in society and in vehicle operation
- Development and effectiveness of the Drug Evaluation and Classification Program (DECP)
- Overview of the DEC Procedures
- Eye Examinations (a major component of the DEC procedures)
- Physiology and Drugs
- Vital signs examinations (a major component of the DEC procedures)
- The seven categories of drugs
- The Physician’s Desk Reference (PDR) and other reference sources
- Interviewing suspects (a major component of the DEC procedures)
- Curriculum Vitae (C.V.) preparation and maintenance
- Case preparation and testimony
- Classifying a suspect (interpreting and documenting the results of an evaluation)
G. Course Activities

Hands-on practice is the principal learning activity of the course.

Eye Examinations Practice:

- Nystagmus, Lack of Convergence, Pupil Size, and Reaction to Light

Alcohol Workshop:

- Psychophysical testing practice
- Volunteer drinkers from outside the class will be recruited for this session.

Practicing interpretation of the examination results:

- Several sessions will be devoted to this allowing the participants to review drug evaluation reports and identify the probable drug category or combinations of categories.

Vital signs examinations:

- Pulse, Blood Pressure, Body Temperature

Practicing administration of the drug influence evaluation:

- Several sessions will be devoted to this. In each, participants will practice administering the drug influence examinations to each other. No hands-on practice with actual drugged subjects is included in the classroom portion of DRE training.

Simulated drug impaired subject examinations:

- Participants will work in teams to conduct and document examinations of instructors who will be simulating the indicators of drug-impaired subjects.
Schedule

H. Overview of Participant Manual

• The Participant manual is the basic reference document for this course.
• The manual contains thumbnails of each instructor presentation per session that includes key messages for each frame.
• Read each session prior to each day’s classes.
• Use the manual to review the material prior to taking the final exam.

By taking good notes, and by studying the manual carefully, participants should have no trouble in passing the course.

• There will be numerous quizzes during the class.
I. Glossary of Terms

The Glossary of Terms used in the course is located in the Participant Manual.

J. Course Pre-Test Administration

- 10 minutes
- Some questions have more than one correct answer
- Scores not entered in permanent record

QUESTIONS?
ACCOMMODATION REFLEX
The adjustment of the eyes for viewing at various distances. Meaning the pupils will automatically constrict as objects move closer and dilate as objects move further away.

ADDITION
Habitual, psychological, and physiological dependence on a substance beyond one’s voluntary control.

ADDITIVE EFFECT
One mechanism of polydrug interaction. For a particular indicator of impairment, two drugs produce an additive effect if they both affect the indicator in the same way. For example, cocaine elevates pulse rate and PCP also elevates pulse rate. The combination of cocaine and PCP produces an additive effect on pulse rate.

AFFERENT NERVES
See: "Sensory Nerves."

ALKALOID
A chemical that is found in, and can be physically extracted from, some substance. For example, morphine is a natural alkaloid of opium. It does not require a chemical reaction to produce morphine from opium.

ANALGESIC
A drug that relieves or allays pain.

ANALOG (of a drug)
An analog of a drug is a chemical that is very similar to the drug, both in terms of molecular structure and in terms of psychoactive effects. For example, the drug Ketamine is an analog of PCP.

ANESTHETIC
A drug that produces a general or local insensibility to pain and other sensation.

ANTAGONISTIC EFFECT
One mechanism of polydrug interaction. For a particular indicator of impairment, two drugs produce an antagonistic effect if they affect the indicator in opposite ways. For example, heroin constricts pupils while cocaine dilates pupils. The combination of heroin and cocaine produces an antagonistic effect on pupil size. Depending on how much of each drug was taken, and on when they were taken, the suspect's pupils could be constricted, or dilated, or within the DRE Average range of pupil size.
ARRHYTHMIA
An abnormal heart rhythm.

ARTERY
The strong, elastic blood vessels that carry blood away the heart.

AUTONOMIC NERVE
A motor nerve that carries messages to the muscles and organs that we do not consciously control. There are two kinds of autonomic nerves, the sympathetic nerves and parasympathetic nerves.

AXON
The part of a neuron (nerve cell) that sends out a neurotransmitter.

BAC
(Blood Alcohol Concentration) - The percentage of alcohol in a person’s blood.

BrAC
(Breath Alcohol Concentration) - The percentage of alcohol in a person’s blood as measured by a breath testing device.

BLOOD PRESSURE
The force exerted by blood on the walls of the arteries. Blood pressure changes continuously, as the heart cycles between contraction and expansion.

BRADYCARDIA
Abnormally slow heart rate.

BRADYPNEA
Abnormally slow rate of breathing.

BRUXISM
Grinding the teeth. This behavior is often seen in person who are under the influence of cocaine or other CNS Stimulants.

CANNABIS
This is the drug category that includes marijuana. Marijuana comes primarily from the leaves of certain species of Cannabis plants that grow readily all over the temperate zones of the earth. Hashish is another drug in this category, and consists of the compressed leaves from female Cannabis plants. The active ingredient in both Marijuana and Hashish is a chemical called delta-9 tetrahydrocannabinol, usually abbreviated THC.

CARBOXY THC
A metabolite of THC (tetrahydrocannabinol).
CHEYNE-STOKES RESPIRATION
Abnormal pattern of breathing. Marked by breathlessness and deep, fast breathing.

CNS (Central Nervous System)
A system within the body consisting of the brain, the brain stem, and the spinal cord.

CNS DEPRESSANTS
One of the seven drug categories. CNS Depressants include alcohol, barbiturates, anti-anxiety tranquilizers, and numerous other drugs.

CNS STIMULANTS
One of the seven drug categories. CNS Stimulants include Cocaine, the Amphetamines, Ritalin, Desoxyn, and numerous other drugs.

CONJUNCTIVITIS
An inflammation of the mucous membrane that lines the inner surface of the eyelids caused by infection, allergy, or outside factors. May be bacterial or viral. Persons suffering from conjunctivitis may show symptoms in one eye only. This condition is commonly referred to as "pink eye", a condition that could be mistaken for the bloodshot eyes produced by alcohol or Cannabis.

CONVERGENCE
The "crossing" of the eyes that occurs when a person is able to focus on a stimulus as it is pushed slowly toward the bridge of their nose. (See, also, "Lack of Convergence".)

CRACK/ROCK
Cocaine base, appears as a hard chunk form resembling pebbles or small rocks. It produces a very intense, but relatively short duration "high".

CURRICULUM VITAE
A written summary of a person's education, training, experience, noteworthy achievements and other relevant information about a particular topic.

CYCLIC BEHAVIOR
A manifestation of impairment due to certain drugs, in which the suspect alternates between periods (or cycles) of intense agitation and relative calm. Cyclic behavior, for example, sometimes will be observed in persons under the influence of PCP.

DELIRIUM
A brief state characterized by incoherent excitement, confused speech, restlessness, and possible hallucinations.

DENDRITE
The part of a neuron (nerve cell) that receives a neurotransmitter.
**DIACETYL MORPHINE**

The chemical name for Heroin.

**DIASTOLIC**

The lowest value of blood pressure. The blood pressure reaches its diastolic value when the heart is fully expanded, or relaxed (Diastole).

**DIPLOPIA**

Double vision.

**DISSOCIATIVE ANESTHETICS**

One of the seven drug categories. Includes drugs that inhibits pain by cutting off or disassociating the brain's perception of pain. PCP and its analogs are considered Dissociative Anesthetics.

**DIVIDED ATTENTION**

Concentrating on more than one thing at a time. The four psychophysical tests used by DREs require the suspect to divide their attention.

**DOWNSIDE EFFECT**

An effect that may occur when the body reacts to the presence of a drug by producing hormones or neurotransmitters to counteract the effects of the drug consumed.

**DRUG**

Any substance that, when taken into the human body, can impair the ability of the person to operate a vehicle safely.

**DYSARTHIA**

Slurred speech. Difficult, poorly articulated speech.

**DYSPNEA**

Shortness of breath.

**DYSMETRIA**

An abnormal condition that prevents the affected person from properly estimating distances linked to muscular movements.

**DYSPHORIA**

A disorder of mood. Feelings of depression and anguish.

**EFFERENT NERVES**

See: "Motor Nerves".
ENDOCRINE SYSTEM

The network of glands that do not have ducts and other structures. They secrete hormones into the blood stream to affect a number of functions in the body.

EXPERT WITNESS

A person skilled in some art, trade, science or profession, having knowledge of matters not within the knowledge of persons of average education, learning and experience, who may assist a jury in arriving at a verdict by expressing an opinion on a state of facts shown by the evidence and based upon his or her special knowledge. (NOTE: Only the court can determine whether a witness is qualified to testify as an expert.)

FLASHBACK

A vivid recollection of a portion of a hallucinogenic experience. Essentially, it is a very intense daydream. There are three types: (1) emotional -- feelings of panic, fear, etc.; (2) somatic -- altered body sensations, tremors, dizziness, etc.; and (3) perceptual -- distortions of vision, hearing, smell, etc.

GAIT ATAXIA

An unsteady, staggering gait (walk) in which walking is uncoordinated and appears to be “not ordered.”

GARRULITY

Chatter, rambling or pointless speech. Talkative.

GENERAL INDICATOR

Behavior or observations of the subject that are observed and not specifically tested for. (Observational and Behavioral Indicators)

HALLUCINATION

A sensory experience of something that does not exist outside the mind, e.g., seeing, hearing, smelling, or feeling something that isn’t really there. Also, having a distorted sensory perception, so that things appear differently than they are.

HALLUCINOGENS

One of the seven drug categories. Hallucinogens include LSD, MDMA, Peyote, Psilocybin, and numerous other drugs.

HASHISH

A form of cannabis made from the dried and pressed resin of a marijuana plant.

HASH OIL

Sometimes referred to as “marijuana oil” it is a highly concentrated syrup-like oil extracted from marijuana. It is normally produced by soaking marijuana in a container of solvent, such as acetone or alcohol for several hours and after the solvent has evaporated, a thick syrup-like oil is produced with a high THC content.
HEROIN
   A powerful and widely-abused narcotic analgesic that is chemically derived from morphine. The chemical, or generic name of heroin is "diacetyl morphine".

HOMEOSTASIS
   The dynamic balance, or steady state, involving levels of salts, water, sugars, and other materials in the body's fluids.

HORIZONTAL GAZE NYSTAGMUS (HGN)
   Involuntary jerking of the eyes occurring as the eyes gaze to the side.

HORMONES
   Chemicals produced by the body's endocrine system that are carried through the bloodstream to the target organ. They exert great influence on the growth and development of the individual, and that aid in the regulation of numerous body processes.

HYDROXY THC
   A metabolite of THC (tetrahydrocannabinol).

HYPERFLEXIA
   Exaggerated or over extended motions.

HYPERGLYCEMIA
   Excess sugar in the blood.

HYPERPNEA
   A deep, rapid or labored breathing.

HYPERPYREXIA
   Extremely high body temperature.

HYPERREFLEXIA
   A neurological condition marked by increased reflex reactions.

HYPERTENSION
   Abnormally high blood pressure. Do not confuse this with hypotension.

HYPOGLYCEMIA
   An abnormal decrease of blood sugar levels.

HYPOPNEA
   Shallow or slow breathing.
HYPOTENSION
Abnormally low blood pressure. Do not confuse this with hypertension.

HYPOTHERMIA
Decreased body temperature.

ICE
A crystalline form of methamphetamine that produces a very intense and fairly long-lasting "high".

INHALANTS
One of the seven drug categories. The inhalants include volatile solvents (such as glue and gasoline), aerosols (such as hair spray and insecticides) and anesthetic gases (such as nitrous oxide).

INSUFFLATION
See "snorting".

INTEGUMENTARY SYSTEM
The skin and accessory structures, hair and nails. Functions include protection, maintenance of body temperature, excretion of waste, and sensory perceptions.

INTRAOCULAR
"Within the eyeball".

KOROTKOFF SOUNDS
A series of distinct sounds produced by blood passing through an artery, as the external pressure on the artery drops from the systolic value to the diastolic value.

LACK OF CONVERGENCE
The inability of a person's eyes to converge, or "cross" as the person attempts to focus on a stimulus as it is pushed slowly toward the bridge of his or her nose.

MAJOR INDICATORS
Physiological signs that are specifically assessed and are, for the most part, involuntary reflecting the status of the central nervous system (CNS) homeostasis (Physiological Indicators).

MARIJUANA
Common term for the Cannabis Sativa plant. Usually refers to the dried leaves of the plant. This is the most common form of the cannabis category.
MARINOL
A drug containing a synthetic form of THC (tetrahydrocannabinol). Marinol belongs to the cannabis category of drugs, but marinol is not produced from any species of cannabis plant.

MEDICAL IMPAIRMENT
An opinion made by a DRE based on the evaluation that the state of a suspected impaired driver is more likely related to a medical impairment that has affected the subject’s ability to operate a vehicle safely.

METABOLISM
The sum of all chemical processes that take place in the body as they relate to the movements of nutrients in the blood after digestion, resulting in growth, energy, release of wastes, and other body functions. The process by which the body, using oxygen, enzymes and other internal chemicals, breaks down ingested substances such as food and drugs so they may be consumed and eliminated. Metabolism takes place in two phases. The first step is the constructive phase (anabolism) where smaller molecules are converted to larger molecules. The second steps is the destructive phase (catabolism) where large molecules are broken down into smaller molecules.

METABOLITE
A chemical product, formed by the reaction of a drug with oxygen and/or other substances in the body.

MIOsis
Abnormally small (constricted) pupils.

MOTOR NERVES
Nerves that carry messages away from the brain, to be body's muscles, tissues, and organs. Motor nerves are also known as efferent nerves.

MUSCULAR HYPERTONICITY
Rigid muscle tone.

MYDRIASIS
Abnormally large (dilated) pupils.

NARCOTIC ANALGESICS
One of the seven drug categories. Narcotic analgesics include opium, the natural alkaloids of opium (such as morphine, codeine and thebaine), the derivatives of opium (such as heroin, dilaudid, oxycodone and percodan), and the synthetic narcotics.
NERVE

A cord-like fiber that carries messages either to or from the brain. For drug evaluation and classification purposes, a nerve can be pictured as a series of "wire-like" segments, with small spaces or gaps between the segments.

NEURON

A nerve cell. The basic functional unit of a nerve. It contains a nucleus within a cell body with one or more axons and dendrites.

NEUROTRANSMITTER

Chemicals that pass from the axon of one nerve cell to the dendrite of the next cell, and that carry messages across the gap between the two nerve cells.

NULL EFFECT

One mechanism of polydrug interaction. For a particular indicator of impairment, two drugs produce a null effect if neither of them affects that indicator. For example, PCP does not affect pupil size, and alcohol does not affect pupil size. The combination of PCP and alcohol produces a null effect on pupil size.

NYSTAGMUS

An involuntary jerking of the eyes.

"ON THE NOD"

A semi-conscious state of deep relaxation. Typically induced by impairment due to Heroin or other narcotic analgesics. The suspect's eyelids droop, and chin rests on the chest. Suspect may appear to be asleep, but can be easily aroused and will respond to questions.

OVERLAPPING EFFECT

One mechanism of polydrug interaction. For a particular indicator of impairment, two drugs produce an overlapping effect if one of them affects the indicator but the other doesn't. For example, cocaine dilates pupils while alcohol doesn't affect pupil size. The combination of cocaine and alcohol produces an overlapping effect on pupil size: the combination will cause the pupils to dilate.

PALLOR

An abnormal paleness or lack of color in the skin.

PARANOIA

Mental disorder characterized by delusions and the projection of personal conflicts that are ascribed to the supposed hostility of others.
PARAPHERNALIA

Drug paraphernalia are the various kinds of tools and other equipment used to store, transport or ingest a drug. Hypodermic needles, small pipes, bent spoons, etc., are examples of drug paraphernalia. The singular form of the word is "paraphernalium". For example, one hypodermic needle would be called a "drug paraphernalium".

PARASYMPATHETIC NERVE

An autonomic nerve that commands the body to relax and to carry out tranquil activities. The brain uses parasympathetic nerves to send "at ease" commands to the muscles, tissues, and organs.

PARASYMPATHOMIMETIC DRUGS

Drugs that mimic neurotransmitter associated with the parasympathetic nerves. These drugs artificially cause the transmission of messages that produce lower blood pressure, drowsiness, etc.

PDR (Physician's Desk Reference)

A basic reference source for drug recognition experts. The PDR provides detailed information on the physical appearance and psychoactive effects of licitly-manufactured drugs.

PHENCYCLIDINE

A contraction of PHENYL CYCLOHEXYL PIPERIDINE, or PCP. Formerly used as a surgical anesthetic, however, it has no current legitimate medical use in humans.

PHENYL CYCLOHEXYL PIPERIDINE (PCP)

Often called "phencyclidine" or "PCP", it is a specific drug belonging to the Dissociative Anesthetics category.

PHYSIOLOGY

Physiology is the branch of biology dealing with the functions and activities of life or living matter and the physical and chemical phenomena involved.

PILOERECTION

Literally, "hair standing up", or goose bumps. This condition of the skin is often observed in persons who are under the influence of LSD.

POLYDRUG USE

Ingesting drugs from two or more drug categories.

PSYCHEDELIC

A mental state characterized by a profound sense of intensified or altered sensory perception sometimes accompanied by hallucinations.
PSYCHOPHYSICAL TESTS

Methods of investigating the mental (psycho-) and physical characteristics of a person suspected of alcohol or drug impairment. Most psychophysical tests employ the concept of divided attention to assess a suspect's impairment.

PSYCHOTGENIC

Literally, "creating psychosis" or "giving birth to insanity". A drug is considered to be psychotogenic if persons who are under the influence of the drug become insane, and remain so after the drug wears off.

PSYCHOTOMIMETIC

Literally, "mimicking psychosis" or "impersonating insanity". A drug is considered to be psychotomimetic if persons who are under the influence of the drug look and act insane while they are under the influence.

PTOSIS

Droopy eyelids.

PULSE

The expansion and contraction of the walls of an artery, generated by the pumping action of blood.

PULSE RATE

The number of expansions of an artery per minute.

PUPILLARY LIGHT REFLEX

The pupils of the eyes will constrict and dilate depending on changes in lighting.

PUPILLARY UNREST

The continuous, irregular change in the size of the pupils that may be observed under room or steady light conditions.

REBOUND DILATION

A period of pupillary constriction followed by a period of pupillary dilation where the pupil steadily increases in size and does not return to its original constricted size.

RESTING NYSTAGMUS

Jerking of the eyes as they look straight ahead.

SCLERA

A dense white fibrous membrane that, with the cornea, forms the external covering of the eyeball (i.e., the white part of the eye).
SENSORY NERVES
Nerves that carry messages to the brain, from the various parts of the body, including notably the sense organs (eyes, ears, etc.). Sensory nerves are also known as afferent nerves.

SINSEMILLA
The unpollenated female cannabis plant, with a relatively high concentration of THC.

SFST
Standardized Field Sobriety Testing. There are three SFSTs, namely Horizontal Gaze Nystagmus (HGN), Walk and Turn, and One Leg Stand. Based on a series of controlled laboratory studies, scientifically validated clues of impairment have been identified for each of these three tests. They are the only Standardized Field Sobriety Tests for which validated clues have been identified.

SNORTING
One method of ingesting certain drugs. Snorting requires that the drug be in powdered form. The user rapidly draws the drug up into the nostril, usually via a paper or glass tube. Snorting is also known as insufflation.

Sphygmomanometer
A medical device used to measure blood pressure. It consists of an arm or leg cuff with an air bag attached to a tube and a bulb for pumping air into the bag, and a gauge for showing the amount of air pressure being pressed against the artery.

Stethoscope
A medical instrument used, for drug evaluation and classification purposes, to listen to the sounds produced by blood passing through an artery.

Sympathetic Nerve
An autonomic nerve that commands the body to react in response to excitement, stress, fear, etc. The brain uses sympathetic nerves to send "wake up calls" and "fire alarms" to the muscles, tissues and organs.

Sympathomimetic Drugs
Drugs that mimic the neurotransmitter associated with the sympathetic nerves. These drugs artificially cause the transmission of messages that produce elevated blood pressure, dilated pupils, etc.

Synapse (or Synaptic Gap)
The gap or space between two neurons (nerve cells).
SYNESTHESIA
A sensory perception disorder, in which an input via one sense is perceived by the brain as an input via another sense. An example of this would be a person “hearing” a phone ring and “seeing” the sound as a flash of light. Synesthesia sometimes occurs with persons under the influence of hallucinogens.

SYSTOLIC
The highest value of blood pressure. The blood pressure reaches its systolic value when the heart is fully contracted (systole), and blood is sent surging into the arteries.

TACHYCARDIA
Abnormally rapid heart rate.

TACHYPNEA
Abnormally rapid rate of breathing.

THC (Tetrahydrocannabinol)
The principal psychoactive ingredient in drugs belonging to the cannabis category.

TOLERANCE
An adjustment of the drug user’s body and brain to the repeated presence of a drug. As tolerance develops, the user will experience diminishing psychoactive effects from the same dose of the drug. As a result, the user typically will steadily increase the dose he or she takes, in an effort to achieve the same psychoactive effect.

TRACKS
Scar tissue usually produced by repeated injection of drugs, via hypodermic needle, along a segment of a vein.

VEIN
A blood vessel that carries blood back to the heart from the body tissues

VERTICAL GAZE NYSTAGMUS
An involuntary jerking of the eyes (up-and-down) which occurs as the eyes are held at maximum elevation. The jerking should be distinct and sustained.

VOIR DIRE
A French expression literally meaning “to see, to say.” Loosely, this would be rendered in English as “To seek the truth,” or “to call it as you see it.” In a law or court context, one application of voir dire is to question a witness to assess his or her qualifications to be considered an expert in some matter pending before the court.

VOLUNTARY NERVE
A motor nerve that carries messages to a muscle that we consciously control.
WITHDRAWAL

This occurs in someone who is physically addicted to a drug when he or she is deprived of the drug. If the craving is sufficiently intense, the person may become extremely agitated, and even physically ill.
Session 2

Drugs in Society and in Vehicle Operation
Learning Objectives

• Define the term “drug” in the context of this course
• Name the seven drug categories relevant to the DEC program
• State in approximate, quantitative terms the incidence of drug use among various segments of the American public

Upon completion of this session, participants will be able to:

• Define the term “drug” in the context of this course.
• Name the seven drug categories relevant to the Drug Evaluation and Classification program.
• State in approximate, quantitative terms the incidence of drug use among various segments of the American public.
• State in approximate, quantitative terms the incidence of drug involvement in motor vehicle crashes and other driving incidents.
• Correctly answer the “topics for study” questions at the end of this session.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES
A. Definition and Categories of Drugs.......................................................... Instructor Led Presentations
B. Incidence and Characteristics of Drug Use in America.......................... Reading Assignments
C. Incidence of Drug Impaired Driving
A. Definition and Categories of Drugs

- *Medicines? Are all drugs medicines? Are all medicines drugs?*
- *Narcotics? Are all drugs Narcotics?*
- *Habit forming substances? Are all drugs habit forming? Are all habit forming substances drugs.*
- A simple, law enforcement oriented definition.
- This definition is derived from the California Vehicle Code.
  “Any substance that, when taken into the human body, can impair the ability of the person to operate a vehicle safely.”
- Within this simple, law enforcement oriented definition, there are seven categories of drugs.
- Each category consists of substances that impair a person’s ability to drive.
- The categories differ from one another in terms of how they impair driving ability and in terms of the kinds of impairment they cause.
- Because the categories produce different types of impairment, they generate different signs and symptoms.
- With training and practice, you will be able to recognize the different signs of drug influence and determine which category is causing the impairment you observe in a subject.
Central Nervous System Depressants

The category of CNS Depressants includes some of the most commonly abused drugs. Alcohol remains the most familiar drug. In 2014, 52.7% of the population aged 12 and older were current drinkers of alcohol.

Source: National Survey on Drug Use and Health (NSDUH), September 2015.

CNS Depressants:

- Slow down the operation of the Central Nervous System (i.e., the brain, brain stem and spinal cord).
- Cause the user to react more slowly.
- Cause the user to process information more slowly.
- Relieve anxiety and tension.
- Induce sedation, drowsiness and sleep.
- In high doses, CNS Depressants will produce general anesthesia. i.e., depress the brain’s ability to sense pain.
- In very high doses, induce coma and death.
Central Nervous System Stimulants

CNS Stimulants constitute another widely abused category of drugs. According to the 2014 NSDUH Survey, there appears to be approximately 1.5 million current (within the last month) Cocaine users aged 12 and older in the U.S. Estimates of drug use vary widely, especially for illicit drugs such as Cocaine, Methamphetamines, etc.

- In 2014, approximately 1.6 million persons aged 12 or older were current non-medical users of stimulants. Source: NSDUH, September 2015.

CNS Stimulants:

- Speed up the operation of the Central Nervous System, and of the various bodily functions controlled by the Central Nervous System
- Cause the user to become hyperactive, extremely talkative
- Speech may become rapid and repetitive
- Heart rate increases
- Blood pressure increases
- Body temperature rises, user may become excessively sweaty
- Induce emotional excitement, restlessness, irritability
- Can induce cardiac arrhythmia (abnormal beating of the heart), cardiac seizures and death
Hallucinogens

Hallucinogens are also widely abused.

LSD and Peyote are only two examples of Hallucinogens. There are many other Hallucinogens. In recent years, significant increases in the abuse of both LSD and “Ecstasy” (MDMA) have been reported.

Hallucinogens:

• Create perceptions that differ from reality. These perceptions are often very distorted, so that the user sees, hears, and smells things in a way quite different from how they really look, sound, and smell.

• Hallucinogens cause the nervous system to send strange or false signals to the brain.

• Clarification: Hallucinogens confuse the Central Nervous System (as well as speeding it up, like CNS Stimulants).

• Produce sights, sounds, odors, feelings and tastes that aren’t real.

• Induce a temporary condition very much like psychosis or insanity.

• Can create a “mixing” of sensory modalities, so that the user “hears colors,” “sees music.”

This mixing of the senses is called Synesthesia. With all of these false, and distorted perceptions, a person under the influence of hallucinogens would be a very unsafe driver.
Dissociative Anesthetics

Examples:
- Dextromethorphan
- Ketamine
- PCP (Phenyl Cyclohexyl Piperidine)

PCP, its analogs and Dextromethorphan are examples of Dissociative Anesthetics. PCP is considered by the medical community to be a Hallucinogen. However, because of the symptomatology it presents, it is in a separate category.

- Phencyclidine is a short form of the chemical name Phenyl Cyclohexyl Piperidine, from which we get the abbreviation “PCP.”

PCP is a synthetic drug, i.e., it does not occur naturally but must be produced in a laboratory-like setting.

PCP has many analogs, or “chemical cousins” that are very similar to PCP in chemical structure, and that produce essentially the same effects.

- Analogs of PCP include Ketamine, Ketalar and Ketajet.
- PCP is also a very powerful pain killer, or anesthetic.

Dextromethorphan (DXM) is found in many over-the-counter anti-tussive cold medications such as Robitussin, Coricidin Cough and Cold, and Dimetapp. DXM is typically abused by school age children, teenagers or young adults to achieve impairment.

- DXM is normally used in liquid or pill form.
- In high doses, DXM impairment is similar to the effects of PCP or Hallucinogens.
Narcotic Analgesics

There are two subcategories of Narcotic Analgesics:

1. Natural Opiates: are derivatives of Opium.
2. Synthetics: are produced chemically in the laboratory. The synthetics are not derived in any way from Opium, but produce similar effects.

The word “Analgesic” means pain reliever. All of the drugs in this category reduce the person’s reaction to pain.

- Heroin is one of the most commonly abused of the Narcotic Analgesics.
- Heroin is highly addictive.

In addition to reducing pain, Narcotic Analgesics produce euphoria, drowsiness, apathy, lessened physical activity and sometimes impaired vision.

Persons under the influence of Narcotic Analgesics often pass into a semi-conscious type of sleep or near-sleep. This condition is often called being “on the nod”. They often are sufficiently alert to respond to questions effectively. Higher doses of Narcotic Analgesics can induce coma, respiratory failure and death.
Inhalants

Inhalants are the fumes of certain substances. These substances are found in many common products:

- Gasoline
- Oil-based paints
- Various glues
- Aerosol cans
- Varnish remover
- Cleaning fluids
- Etc.

Examples:

- Volatile Solvents (Various Glues, Gasoline, Paint, etc.)
- Aerosols (Hairspray, Insecticides, etc.)
- Anesthetic Gases (Nitrous Oxide, Amyl Nitrite, etc.)

Different Inhalants produce different effects.

- Many produce effects similar to those of CNS Depressants.
- A few produce stimulant-like effects.
- Some produce hallucinogenic effects.

The Inhalant abuser’s attitude and demeanor can vary from inattentive, stuporous and passive to irritable, violent and dangerous. The abuser’s speech will often be slow, thick and slurred.
Cannabis

The category “Cannabis” includes the various forms and products of the Cannabis Sativa plant and other species of Cannabis plants.

The primary active ingredient in Cannabis products is the substance known as “Delta-9 Tetrahydrocannabinol,” or “THC.”

Apart from alcohol, marijuana is the most commonly abused drug in this country.

According to the NSDUH 2014 Survey, marijuana was listed as the most common illicit drug used in the U.S. There were 19.8 million Americans over the age of 12 reporting use in the past month.

Daily or almost daily use of marijuana (used on 20 or more days in the past month) increased from 5.1 million persons in 2005-2007 to 8.1 million persons in 2013.

Cannabis appears to interfere with the attention process. Drivers under the influence of Marijuana often do not pay attention to their driving.

Cannabis also produces a distortion of the user’s perception of time, an increased heart rate (often over 100 beats per minute) and reddening of the eyes.
Drug Combinations

Many drug users appear to be “chemical gluttons.” They often ingest drugs from two or more drug categories.

The term for this is “polydrug use.”

Some very common examples of polydrug use include:

- Alcohol with virtually any other drug
- Marijuana and PCP - A common way to ingest PCP is to sprinkle it on a Marijuana “joint” and smoke it.
- Cocaine and Heroin, sometimes called a “speedball.”
- Heroin and Amphetamine, sometimes called a “poor man’s speedball.”
- Heroin and PCP, sometimes called a “fireball.”
- “Crack” Cocaine and PCP, sometimes called a “space base.”
- “Crack” Cocaine and Marijuana, sometimes called a “primo.”
- “Crack” and Methamphetamine, sometimes called “croak.”

Sometimes, people take two different drugs (such as Heroin and Cocaine) that produce some opposite effects.

Example: Heroin tends to lower blood pressure. Cocaine tends to elevate blood pressure.

Different drug combinations may produce unique, interactive effects.

When a person has ingested multiple drugs, that person will experience multiple drug effects.

Under proper medical supervision, specific drugs often are used to reverse overdose conditions. However, it is important to bear in mind that, in a polydrug situation, some of the signs of a particular drug may not be evident even though the person is under the influence of that drug.
B. Incidence and Characteristics of Drug Use in America

- In 2014, 27.0 million Americans aged 12 years or older were current illicit drug users.
- Marijuana was the most commonly used illicit drug in 2014, with 22.2 million users reporting use in the past month.
- In 2014, there were an estimated 1.5 million Cocaine users aged 12 or older in the U.S.

All stats same Source: National Survey on Drug Use and Health (NSDUH, September 2015)

C. Incidence of Drug Impaired Driving

Accurate data on the frequency with which people drive while under the influence of drugs is somewhat limited.

This is due to the various reasons that include:

- Many impaired drivers are never detected.
- Many drug users also consume alcohol, when they are stopped for impaired driving they may be arrested (and tabulated in statistics) as alcohol impaired drivers only.

Fact: About 9.9 million people aged 12 years and older admitted driving under the influence of illicit drugs in the past year.

Source: National Survey on Drug Use and Health (NSDUH) 2014.

When they are involved in crashes, they may not be tested for drugs.
NHTSA undertook a comprehensive study of the prevalence of potentially-impairing drug use by drivers in 2013 and 2014.

Report: The 2013-2014 National Roadside Survey of Alcohol and Drug Use by Drivers. (NHTSA)

Approximately 30,000 drivers were asked to provide an oral fluid or blood sample. Samples were tested for illegal drugs, prescription medicines, and other-the-counter drugs.

- About 20% of drivers tested positive for at least one drug, up from 16.3% in the 2007 Roadside Study.
- 12.6% of the drivers had evidence of marijuana use in their systems, up from 8.6% in the 2007 Roadside Study.
- More than 15% of drivers tested positive for at least one illegal drug, up from 12% in 2007.

Source: National Roadside Survey Fact Sheet, Jan 2014

The facts are unmistakable: Drug use is common among many Americans. So is drug impaired driving.
**NHSTA Drug and Alcohol Crash Risk Study**

- Largest such study ever conducted to assess the comparative risk of drunk and drugged driving.
- Conducted in Virginia Beach, Va., over a 20-month period.
- Collected data from more than 3,000 drivers involved in a crash, and more than 6,000 non-crash drivers for comparison.
- Drivers were tested for a wide range of drugs, but marijuana was the only drug found in large enough numbers for statistically significant findings.

**NHSTA Drug and Alcohol Crash Risk Study Key Findings**

- Drivers at a BAC level of 0.08 percent were about four times more likely to crash than sober drivers.
- Drivers with a BAC level of 0.15 percent were 12 times more likely to crash than sober drivers.
- Marijuana users were about 25% more likely to be involved in a crash than drivers with no evidence of marijuana use.

*Source: NHSTA Drug and Alcohol Crash Risk Study Fact Sheet, January 2014.*
**Topics for Study Questions / Answers:**

1. What does the term “drug” mean, as it is used in this course?

2. What are the seven categories of drugs? To which category does alcohol belong? To which category does Cocaine belong?

3. What does “polydrug use” mean?

4. What is a “Speedball”? What is a “Space Base”?

5. In the 2013 – 2014 National Roadside Survey of Alcohol and Drug Use by Drivers, more than _____ % of drivers, tested positive for at least one illegal drug.
Participant Manual

Drug Recognition Expert Course

Session 3
Development and Effectiveness of the Drug Evaluation and Classification Program

NHTSA
Learning Objectives

- State the origin and evolution of the Drug Evaluation and Classification program
- Describe research and demonstration project results that validate the effectiveness of the program
- State the impact of legal precedents established by case law
- Correctly answer the "topics for study" questions at the end of this session

Upon successfully completing this session the participant will be able to:

- State the origin and evolution of the Drug Evaluation and Classification Program.
- Describe research and demonstration project results that validate the effectiveness of the program.
- State the impact of legal precedents established by case law.
- Correctly answer the “topics for study” questions at the end of this session.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES

A. Origin and Evolution of Drug Evaluation and Classification Program
   Instructor-Led Presentations

B. Evidence of Program Effectiveness
   Reading Assignments

C. Case Law Review

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A. Origin and Evolution of the Drug Evaluation and Classification (DEC) Program

The DEC program was developed by personnel of the Los Angeles Police Department.

Development of the DEC program began in the early 1970’s, in response to a growing awareness that many people apprehended for impaired driving were under the influence of drugs rather than alcohol.

Dick Studdard (Traffic Officer):

- Sergeant Studdard retired from the LAPD in June, 1990.
- Sgt. Studdard and his fellow officers often encountered many impaired drivers whose BACs were zero or very low.

They occasionally succeeded in having physicians examine some of these low BAC subjects, resulting in diagnosis of drug influence.
Some reasons why doctors may be reluctant:

- They typically receive little training in the recognition of specific signs of drug impairment, particularly at street level doses.
- They may not see the subject until hours after the drugs were used, by which time the signs and symptoms often have changed.

As a result, some drivers whom Stg. Studdard and other officers were certain were impaired were not prosecuted or convicted for DWI.

Stg. Studdard concluded that it was essential to develop appropriate procedures that officers could use when confronted with persons suspected of drugs.

Len Leeds, former LAPD Narcotics Officer:

- Was approached by Sgt. Studdard and asked to collaborate in the development of a program to help identify drug-impaired subjects.
- Initiated some independent research by consulting with physicians, enrolling in relevant classes, studying text books, technical articles, etc.
- Secured management level support within the department to continue research and program development.

As time went on, many other key persons both within and outside LAPD contributed to the development and refinement of the program.

In 1979, the program was officially recognized by LAPD.
B. Evidence of Program Effectiveness

LAPD and the National Highway Traffic Safety Administration (NHTSA) worked together to develop the DRE training as we know it today.

The first step was to develop and validate a battery of standardized field sobriety tests for investigating alcohol impaired driving.

LAPD personnel played a major role in the research that led to the wide spread use of Horizontal Gaze Nystagmus, the Walk and Turn test, and the One Leg Stand test.

By the early 1980’s, NHTSA completed its validation of the standardized tests for DWI enforcement.

At this time, NHTSA began to assist LAPD in validating the Drug Recognition Expert program.
**The DRE Process:**

1. Establishes that the subject is impaired
2. Rules out medical impairment
3. Determines the category or categories of drugs involved

The DRE process evolved into what is essentially a three-part determination.

- First, it establishes the subject is impaired and verifies his or her alcohol level is not consistent with the degree of impairment that is evident.

Inconsistency between the observed impairment and the BAC suggests the presence of some other drug(s), or some other complicating factor such as an illness or injury.

- Second, it uses some simple evaluation procedures to determine whether the impairment may stem from illness or injury, requiring medical attention.
- Third, it uses evaluation procedures to determine what category (or categories) of drugs are the likely cause of the impairment.

**Key Point**

The entire evaluation process is standardized.

- Administered the same way to all subjects.
- Administered the same way by all officers.
The Need for Reliable Standardized Assessment Procedure

- One reason for needing a reliable standardized assessment procedure is that we may be called upon to submit evidence of an articulable suspicion of drug influence to support our request for a chemical test of the subject.

- Some courts or motor vehicle hearings officers may find that a low BAC result, by itself, does not provide adequate basis for requesting the subject to submit to a 2nd chemical test.

- Another reason is that the subject may refuse to submit to the chemical test, denying us of scientific evidence of drug influence. In that case, conviction or acquittal may hinge on the officer’s observations and expertise as a DRE.

- A third reason is that chemical tests usually disclose only that the subject has used a particular drug recently. The chemical test usually does not indicate whether the drug is psychoactive at the present time.

- Thus, the DRE procedures are needed to establish that the subject not only has used the drug, but also that he or she is under the influence.

- A fourth reason is that it can be expensive and require a large sample of blood or urine to perform a broad analysis for any or all drugs. Practical constraints require that we be able to point the laboratory technician toward those types of drugs most likely to be found in the sample.

- It is always possible that a person suspected of drug impairment is actually suffering from some medical problem. If a sample is collected, and the subject is not examined by someone who is qualified, evidence of medical problems may not come to light until it is too late.
Two Stages of Validation

Laboratory Validation Study
• Johns Hopkins University

Field Validation Study
• Los Angeles

NHTSA assisted LAPD in a two-phase validation study.

• Laboratory validation, using volunteers who ingested selected drugs. The Johns Hopkins validation was conducted in 1984.

• Field validation, using persons actually arrested in Los Angeles on suspicion of drug influence. The LAPD Field Validation Study was conducted in 1985.

The research validation studies and their titles were:


1. Laboratory Validation Study

The Laboratory Validation took place at Johns Hopkins University in Maryland. The drug examiners were senior DREs from LAPD. The LAPD participants: Dick Studdard; Jerry Powell; Pat Russell; and Doug Laird.

The laboratory experiments were planned and conducted by researchers from Johns Hopkins. Volunteers each took a “pill” and smoked a “cigarette.”

The “pill” contained either no drug (placebo) or one of the following drugs:
- Secobarbital (CNS Depressant)
- Valium (i.e., Diazepam – CNS Depressant)
- d-amphetamine (CNS Stimulant).

A common brand name for secobarbital is Seconal; a common brand name for diazepam is Valium and a common brand name for d-amphetamine is Dexedrine.

The “cigarette” contained either THC or no drug (placebo). Neither the volunteers nor the LAPD officers knew what the volunteers had taken.

Two different dose levels of Marijuana, Diazepam and d-amphetamine were used.

Clarification: some of the Diazepam and d-amphetamine pills were “weak,” some were “strong.” Similarly, some of the Marijuana cigarettes were “weak,” some “strong.” All of the Secobarbital pills were “strong.”
Normal daily dose for therapeutic purposes:

- Secobarbital: approx. 100 mg.
- Diazepam: 4-40 mg.
- d-amphetamine: 15 mg.

Doses administered for this study:

- Secobarbital: 300 mg.
- Diazepam: weak – 15mg, strong – 30mg.
- d-amphetamine: weak – 15 mg, strong – 30 mg.
- Marijuana: weak – 12 puffs or 1.3% THC cigarettes, strong – 12 puffs of 2.8% THC cigarettes.
Laboratory Study Results

- DRE officers correctly identified 95% of drug-free subjects as “unimpaired”
- DRE officers classified 98.7% of high-dose subjects as “impaired”

Results

- The DREs were excellent in identifying subjects who received only placebo doses: they classified 95% of the drug free subjects as “not impaired.”
- Similarly, they were excellent in identifying the high dose subjects.
- They classified as “impaired” 98.7% of the subjects who received Secobarbital or strong doses of Marijuana, Diazepam or d-amphetamine.
- They correctly identified the category of drug for 91.7% of those strong dose subjects.
- The DREs were less successful in identifying the weak dose subjects.
- Only 17.5% of the subjects who received the weak dose of d-amphetamine were classified as “impaired.”
- Only 32.5% of the subjects who smoked the “weak” Marijuana cigarettes were classified as “impaired.”
- The results of the laboratory validation study were considered to be extremely positive.
- The DRE procedures correctly identified the category of drugs in more than 90% of the subjects who were impaired.
- The procedures only rarely indicated that unimpaired subjects were under the influence of drugs.
- Laboratory studies can only allow certain dose levels of drugs, which are much lower than those seen at street levels. Therefore, participants in laboratory studies may not show many of the signs of impairment that are seen with subjects ingesting street level doses of drugs.
2. Field Validation Study

The field validation study was based on one hundred seventy-three people actually arrested on suspicion of driving under the influence of drugs.

None of the 173 cases involved a crash. In all of the cases, the arrested subjects agreed to submit to a blood test.

Twenty-eight different DREs from LAPD and the L.A. area participated in the examinations of these one hundred seventy-three subjects.

The researchers excluded all cases where the subjects refused to give blood, since it would have been impossible to check the DREs accuracy in those cases. Similarly, they excluded all cases that involved crashes, since the subjects’ injuries could have confounded the drug examination. Also excluded were subjects who were found in possession of drugs or had any charges other than the drugged driving charge.
Results of the Field Study

Based on the independent blood tests, only one of the one hundred seventy-three subjects was found to have no alcohol or other drugs. Another ten subjects were found to have only alcohol in them.

Thirty-seven (21%) of the subjects were found to have only one drug.

Eighty-two had two drugs (47%) and forty-three (25%) had three or more drugs.

This means that one hundred twenty-five of the one hundred seventy-three subjects had ingested two or more drugs: that is more than 72% of the subjects.

PCP was the drug most often found among these one hundred seventy-three subjects: more than half of them (56%) had used PCP.

The key finding of this study was the following:

- For more than nine out of ten of the subjects (92.5%), the blood test confirmed the presence of at least one drug category “predicted” by the DREs.
The confirmation rates for specific categories:

PCP: blood tests confirmed DREs’ predictions in 92% of the cases.
Narcotic Analgesics: blood tests confirmed 85% of the DREs’ predictions.
Cannabis: blood tests confirmed 78% of DREs’ predictions.
CNS Depressants: blood tests confirmed 50% of DREs’ predictions.
CNS Stimulants: blood tests confirmed 33% of DREs’ predictions.

Numerous states have conducted comparisons of laboratory analysis and DRE opinions. The correlation rates exceeded 80% in those studies.

A Study conducted in 1990 by the Arizona Department of Public Safety Central Regional Crime Laboratory compiled records of the toxicological analysis corresponding to Arizona DREs were analyzed showing that a laboratory confirmation rate of 86.5% had been achieved.

The overall conclusion of the laboratory and field studies is that the DEC Program is an effective tool for law enforcement.
C. Case Law Review

Court Rulings

Favorable Court Rulings on DEC Procedures.

Courts in various states have ruled favorably on the DEC Program. American courts employ either the Frye or Daubert Standard for determining the admissibility of scientific evidence.

The Frye standard is the traditional test for admissibility of “new” scientific evidence.

The Frye standard: “Is the procedure or principle espoused, accepted by the relevant scientific community?”

Frye standard was set by the US Supreme Court in 1923.

In Daubert, courts serve as a gatekeeper for all scientific evidence.

Daubert standard requires a showing of reliability before scientific evidence can be admitted.

Courts assess evidence by considering four factors:

- Opinions are testable.
- Methods/principles have been subject to peer review.
- Known error rate can be identified.
- Opinions rest on methodology that is generally accepted within the relevant scientific/technical community.
The traditional standard for scientific admissibility of evidence was the Frye Standard.

- **State of Arizona v. Dayton Johnson and Samuel Rodriguez, et al, NOS 90056865 and 90035883, (1990).** An Arizona court (Tucson Municipal Court) ruled that the Frye Standard was met. However, upon appeal, the Arizona State Supreme Court ruled that the Frye Standard did not apply to the DEC Program.

- **Washington v. Baity, 991P.2d, 1151, 140 Wn. 2d 1 (2000).** A Washington Supreme Court ruled that the DRE protocols are the application of traditional techniques.

- **State of Minnesota, City of Minneapolis v. Larry Michael Klawitter, 518 N.W.2d 577, (1993).** A Minnesota Court (City of Minneapolis) ruled that outside of nystagmus, the DEC Program is not subject to the Frye Standard.

- **State of Colorado v. Daniel Hernandez, 92M 181, (1992).** The Colorado Supreme Court determined that the Frye Standard applies to the protocol because the process has “scientific elements.” A Colorado Court (Boulder County Court) ruled that the procedures used by DREs are not new or novel and the Frye Standard did not apply.

- **New Mexico v. Mariam Aleman, Dona Ana County, 3rd District (2003).** A New Mexico Court ruled the DRE’s opinion was correct and that the DRE protocol is admissible.

- **Nebraska v. Cubrich, Case No. CR03-8203 Sarpy County Court (2004).**

In this case, the court used the Daubert Standard. In many jurisdictions, it will not be necessary to have expert scientific testimony to secure admissibility of a DRE’s examination of a subject. The DEC Program is gaining acceptance in many courts.

In fact, testimony based on DRE investigation have been accepted by courts for years.

Expert testimony regarding drug influence has long been accepted by numerous courts. The components of DRE evaluation are generally accepted in the scientific community.

The DEC Program simply combined those components into a systematic and standardized procedure. Thus, many prosecutors believe that FRYE standards do not apply to DRE evaluations and testimony.
**HGN Case Law**

One key element of DEC – namely, Horizontal Gaze Nystagmus – has been recognized as meeting the Frye standard by several State Supreme Courts. First to do so was Arizona, in the case known as State vs. Blake.

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**Summary of HGN Case Law**

The prevailing trend is for courts to admit HGN as evidence of impairment, with the proper scientific foundation.

But courts consistently reject all attempts to introduce HGN as evidence of a quantitative BAC.

The court ruled that in cases where there is no chemical test to determine a BAC level, HGN test results can be admitted the same as of Standardized Field Sobriety Tests to show a “neurological dysfunction,” one cause of which could be the ingestion of alcohol.
Topics for Study Questions /Answers:

1. State four reasons why it is important **not** to rely simply on a chemical test to establish a subject’s drug impairment.

2. What categories of drugs were included in the Johns Hopkins Laboratory Study?

3. In what percentage of cases in the Los Angeles Field Validation Study did blood tests confirm the DREs’ opinion that PCP was present?

4. What percentage of blood tests in the LAPD Field Validation Study confirmed the presence of at least one drug category predicted by the DRE’s?

5. What was the landmark State Supreme Court case that upheld the use of HGN as evidence of impairment?

6. What do we call the traditional standard for admissibility of scientific evidence, set by the U.S. Supreme Court?
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“Frye” refers to a United States Federal Court opinion dealing with the admissibility of scientific evidence. The court established that new or novel scientific evidence, or the novel application of scientific principles, must be shown to have met with general acceptance in the relevant scientific community before it can be admitted.

1990
Nos 90056865 and 90035883 (Unpublished Opinion).
The Municipal Court of the City of Tucson, County of Pima, State of Arizona

“Virtually all the witnesses agreed that the scientific procedures utilized by trained drug recognition experts are reliable and are generally accepted in the scientific community. The methodology in place, used by trained law enforcement personnel in the field, has been shown to produce reasonably reliable and uniform results that will contribute materially to the ascertainment of the truth.”

On May 7, 1992, the Arizona Supreme Court heard oral arguments in a special proceeding regarding this case. The Justices uniformly rejected the application of “Frye” to the DRE procedures. The Chief Justice observed that the component examination procedures had been established for fifty years.

The prosecutors in this case were Tom Rankin (Tucson) and Cliff Vanell (Phoenix).


1992
County Court, Boulder, Colorado
Case No. 92M181 (Unpublished Opinion)
People of the State of Colorado v. Daniel Hernandez

“The DRE methods are accepted within the scientific community because they have found to be reliable.”

“The Court finds that the expert does have sufficient specialized knowledge to assist the jurors in better deciding whether the defendant drove his car when under the influence of a specific drug. The DRE testimony can be used at trial provided a sufficient foundation is laid.” Overall, this court ruled that the procedures used by DRE’s are not new or novel scientific techniques that must meet the “Frye” standard.

The prosecutor in this case was David Archeluta (Boulder County). Expert witnesses for the prosecution include: Sergeant Thomas Page, LAPD, Zenon Zuk, M.D., Marcelline Burns, Ph.D., Rick Abbott, M.D., and Laurel Farrell (chemist).
“Given proper foundation and subject to other qualifications, opinion testimony by experienced police officers trained in use of so-called drug recognition protocol is generally admissible in evidence in a trial of a defendant for driving while under the influence of a controlled substance.”

The Court determined that the gaze nystagmus test satisfies the requirements of “Frye”.

“We agree with the trial court that the officer should be allowed to give an opinion based on the officer’s training and experience and his or her observations following the 12-step drug recognition protocol, as long as (a) there is sufficient foundation for the specific opinion expressed, (b) the state does not attempt to exaggerate the officer’s credentials by referring to the officer as a “Drug Recognition Expert” or to unfairly suggest that the officer’s opinion is entitled to greater weight than it deserves, and...” “We add only that it should be obvious that the mere fact that such opinion testimony by itself will be sufficient to support a guilty verdict.”

The court also determined that, outside of nystagmus, the components of a DRE examination are not scientifically new and are not subject to the “Frye” test.

The trial court stated, “...there is nothing scientifically new, novel, or controversial about any component of the DRE protocol itself. The symptomatology matrix used by DRE’s to reach their conclusions is not new and is generally accepted in the medical community as an accurate compilation of signs and symptoms or impairment by the various drug categories.”

The prosecutor in this case was Karen Herland (City of Minneapolis). Expert witnesses for the prosecution included: Sgt. Thomas Page, LAPD, Dr. Marcelline Burns (psychologist), Dr. David Peed (optometrist), Dr. Zenon Zuk (medical doctor), Eugene Adler (criminalist), Dr. S.J. Jejurikar (MN Bureau of Criminal Apprehension), and Robert Meyer (toxicologist).
“Given proper foundation and subject to other qualifications, opinion testimony by an experienced police officer trained in the use of the drug recognition protocol is generally admissible in evidence in a trial of a defendant charged with driving under the influence of a controlled or chemical substance. Furthermore, Horizontal Gaze Nystagmus (HGN) test results are generally admissible to establish (1) that the defendant was impaired; and/or (2) that the defendant was over the legal limit; and/or (3) the defendant’s specific breath or blood alcohol level at the time he performed the test.”

This court found that the “Frye” standard is inapplicable to the DRE Protocol because neither the protocol nor any of its subsets (including HGN, VGN, and Lack of Convergence) are “scientific”.

Further, these tests are neither new nor novel. The Court also state that “Frye” is inapplicable to HGN, VGN, and LOC because none of them are new or novel. “None of these tests or the theories and procedures they encompass, are new, novel, or emerging scientific techniques. The medical and psychological professions have acknowledged the tests’ underlying theories and procedures for decades.”

The Court concluded:

“Drug recognition training is not designed to qualify police officers as scientists, but to train them as observers. The training is intended to refine and enhance the skill of acute observation...and to focus that power...in a particular situation.”

This court followed the Klawitter (Minnesota) decision, that it requires the state to “lay a proper predicate before referring to a DRE as anything other than a DRE or Drug Recognition Evaluator or Examiner.”

“The real issue is not the admissibility of the evidence, but the weight it should receive. That is a matter for the jury to decide.”

The prosecutor in this case was Steve Talpins (Dade County). Expert witnesses for the prosecution in this case included: Marcelline Burns, Ph.D., Zenon Zuk, M.D., Robert Dobie, M.D., Sergeant Thomas Page, LAPD, and others.
In this case, the court was asked to determine if a drug recognition protocol, used by trained drug recognition officers to determine if a suspect’s driving is impaired by a drug other than alcohol, meets the requirements of *Frye v. United States*, 293 F. 1013,34 A.L.R. 145 (1923), for novel scientific evidence.

The issue brought before the court was; Is a drug recognition program novel scientific evidence generally accepted in the scientific community, thus satisfying the *Frye* test for admissibility?

The facts in this case were:

The state charged Baity with one count of DUI, in violation of RCW 46.61.502 (l) (b) (c), and one count of driving while license suspended in the third degree, in violation of RCW 46.20.342(l)(c), after he failed roadside SFST’s and showed signs of drug impairments.

In a pretrial motion in Baity’s case, the State sought to qualify the DREs as experts and to obtain a ruling on the admissibility of DRE evidence with respect to the defendant’s drug impairment and the evaluation process used to determine that impairment. Specifically, the State sought to admit testimony that Baity’s impairment was consistent with the symptoms associated with one of seven categories of drugs. Additionally, the state moved to admit testimony regarding the use of the horizontal gaze nystagmus (HGN) test, both for the detection of alcohol and for the detection of drugs. Baity moved to suppress all DRE evidence, including the HGN test, on the basis that the DRE program and protocol constitute novel scientific evidence subject to the *Frye* test for admissibility.

On May 19, 1998, the Pierce County District Court judges issued their opinion titled, “Opinion Regarding Admissibility of HGN and DRE.” In that opinion, they denied the defendants’ motions to suppress the field sobriety tests (SFSTs) as to their alcohol impairment, holding those tests are “reasonably understandable to the ordinary person” and therefore not subject to *Frye*. Clerk’s Papers at 56. The court also noted some features of the DRE protocol were either not of a scientific nature or were scientific, but not novel.

The court ruled that after analyzing the DRE protocol and the approach of other courts to its admissibility, that the DRE protocol and the chart used to classify the behavioral patterns associated with seven categories of drugs have scientific elements meriting evaluation under *Frye*. They also found that the protocol to be accepted in the relevant scientific communities. However, the court ruled that there is confined situations where all 12-steps of the protocol have been undertaken. Moreover, an officer may not testify in a fashion that casts an aura of scientific certainty to the testimony. The officer also may not predict the specific level of drugs present in a suspect. The DRE officer, properly qualified, may express an opinion that a suspect’s behavior and physical attributes are or are not consistent with the behavioral and physical signs associated with certain categories of drugs.
The court also held that the protocol meets the mandate of Frye. An officer may testify concerning such drug impairment, subject to the limitations set forth in this opinion, upon meeting the requirements of ER 702 and 703 for the admission of expert opinion testimony. The court reversed the suppression orders of the Pierce County District Court and remanded the cases for further proceedings consistent with this opinion.

2003
Case No. CR-2003-00025
State of New Mexico vs. Miriam Aleman
State of New Mexico, County of Dona Ana
Third Judicial District
Judge Silvia E. Cano-Garica

Defendant made a motion In Limine to exclude the testimony of the DRE officer. They heard the testimony of various witnesses and reviewed the State’s Brief in support of the DRE testing. Testimony and other applicable documents found that:

The DRE officer was recognized as an expert of DRE testing based upon his specialized knowledge and experience, the DRE evaluation method is generally accepted in the particular scientific field of forensic toxicology, the DRE evaluation provides critical information which assists the toxicologist in forming an opinion as to whether the driver was impaired by the use of drugs at or near the time the driver was driving the motor vehicle.

The DRE protocols are the application or incorporation of traditional techniques in the biology, physiology, anatomy, chemistry, pharmacology and toxicology fields, and the ultimate decision as to the driver’s alleged impairment, based on all of the testimony received, rests with the jury.

2004
Case No. CR 03-8203
State of Nebraska vs. Timothy J. Cubrich
Judge Todd J. Hutton, Sarpy Co. Court

The court was asked to determine the admissibility of the law enforcement officer’s opinion that the defendant was under the influence of a drug, other than alcohol, to the extent that his abilities to safely operate the vehicle were appreciable impaired. To this end the court applied the standards set forth in Schafersman v. Agland Coop, 262 Neb. 215, 631 N.W. 2d 862 (2001), having adopted Daubert v. Merrel Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993), as the controlling authority in determining the admissibility of expert opinion testimony.

The court concluded: Since Daubert, the court now serves in the “gatekeeping” role in which it is called upon to determine the reliability and relevance of expert testimony. There is no Case Law in Nebraska which has specifically addressed the issue of expert testimony relating to impaired drivers suspected of using drugs. Nor is there a statutory procedure by which Drug Recognition Examinations or the opinions derived there from have been codified.
Application of the Daubert standard provided a number of considerations the court used in determining the admissibility of evidence through the testimony of an expert, which included:

The 12-step protocol which relies on determining if a person is drug impaired has been recognized in the scientific community, including physicians, ophthalmologists, and forensic toxicologists, as a dependable methodology by which an officer, properly trained, can identify impairment and the category of drug(s) which are impairing the suspect’s cognitive and physical capabilities.

The methodology is reliable because it is dependent on a fixed set of assessments which are verified by a toxicology test. The evaluation process includes HGN testing which has been found to meet the Frye standard of admissibility. Additionally, the HGN and VGN tests have been subject to peer review and publication. The remaining tests serve to screen the suspect’s mental and physical condition documenting clues explaining why the person may or may not be impaired and if so the source(s) involved.

The drug recognition assessment is a tool by which a specially trained officer can conclude “based on the totality of results” whether or not a person is impaired by a drug other than alcohol.

The court found that the DREs opinion was correct in that the Defendant showed signs of impairment from a drug, other than alcohol, which caused him to seek a toxicological examination. The category of drug is admissible for the limited purpose of establishing foundation for drug screen conducted by the toxicologists.
INTRODUCTION

The following state case law summary contains the seminal cases for each state, the District of Columbia and the Federal courts on the admissibility of HGN. Three main issues regarding the admissibility of the HGN test are set out under each state: evidentiary admissibility, police officer testimony, and purpose and limits of the HGN test results. The case or cases that address each issue are then briefly summarized and cited.

ALABAMA

I. Evidentiary Admissibility

HGN is a scientific test that must satisfy the Frye standard of admissibility. The Supreme Court of Alabama found that the State had not presented “sufficient evidence regarding the HGN test’s reliability or its acceptance by the scientific community to determine if the Court of Criminal Appeals correctly determined that the test meets the Frye standards.”


II. Police Officer Testimony Needed to Admit HGN Test Result

The Court did not address this issue.

III. Purpose and Limits of HGN

The Court did not address this issue.

ALASKA

I. Evidentiary Admissibility

HGN is a scientific test. It is generally accepted within the relevant scientific community.


II. Police Officer Testimony Needed to Admit HGN Test Result

A police officer may testify to the results of HGN testing as long as the government establishes a foundation that the officer has been adequately trained in the test.

Ballard, 955 P.2d at 941.
III. Purpose and Limits of HGN

HGN testing is “a reliable indicator of a person’s alcohol consumption and, to that extent, HGN results are relevant.” The court cautioned that the HGN test could not be used to correlate the results with any particular blood-alcohol level, range of blood-alcohol levels, or level of impairment. *Ballard*, 955 P.2d at 940.

ARIZONA

I. Evidentiary Admissibility

HGN is a scientific test that needs to satisfy the *Frye* standard of admissibility. State has shown that HGN satisfies the *Frye* standard. *State v. Superior Court (Blake)*, 718 P.2d 171, 181 (Ariz. 1986) (seminal case on the admissibility of HGN).

II. Police Officer Testimony Needed to Admit HGN Test Result

“The proper foundation for [admitting HGN test results] . . . includes a description of the officer’s training, education, and experience in administering the test and showing that proper procedures were followed.”


III. Purpose and Limits of HGN

HGN test results are admissible to establish probable cause to arrest in a criminal hearing.

*State v. Superior Court (Blake)*, 718 P.2d at 182.

“Where a chemical analysis has been conducted, the parties may introduce HGN test results in the form of estimates of BAC over .10% to challenge or corroborate that chemical analysis.” *Ricke*, 778 P.2d at 1361.

When no chemical analysis is conducted, the use of HGN test results “is to be limited to showing a symptom or clue of impairment.” *Hamilton*, 799 P.2d at 858.

ARKANSAS

I. Evidentiary Admissibility

Novel scientific evidence must meet the *Prater* (relevancy) standard for admissibility. Because law enforcement has used HGN for over thirty-five years, a *Prater* inquiry is not necessary as the test is not “novel” scientific evidence. *Whitson v. Arkansas*, 863 S.W.2d 794, 798 (Ark. 1993).

II. Police Officer Testimony Needed to Admit HGN Test Result

The Court did not address this issue.
III. Purpose and Limits of HGN

HGN may be admitted as evidence of impairment, but is not admissible to prove a specific BAC. *Whitson*, 863 S.W.2d at 798.

**CALIFORNIA**

I. Evidentiary Admissibility

HGN is a scientific test and the *Kelly/Frye* “general acceptance” standard must be applied.


“…[A] consensus drawn from a typical cross-section of the relevant, qualified scientific community accepts the HGN testing procedures….”

*Joehnk*, 35 Cal. App. 4th at 1507, 42 Cal. Rptr. 2d at 17.

II. Police Officer Testimony Needed to Admit HGN Test Result


Police officer can give opinion, based on HGN and other test results, that defendant was intoxicated. Furthermore, police officer must testify as to the administration and result of the test. *Joehnk*, 35 Cal. App. 4th at 1508, 42 Cal. Rptr. 2d at 18.

III. Purpose and Limits of HGN

HGN may be used, along with other scientific tests, as some evidence that defendant was impaired. *Joehnk*, 35 Cal. App. 4th at 1508, 42 Cal. Rptr. 2d at 17.

HGN test results may not be used to quantify the BAC level of the defendant.


**CONNECTICUT**

I. Evidentiary Admissibility


Also see, *Connecticut v. Merritt*, 647 A.2d 1021, 1028 (Conn. App. Ct. 1994). HGN must meet the *Frye* test of admissibility. In this case, the state presented no evidence to meet its burden under the *Frye* test.

HGN satisfies the *Porter* standards and is admissible. (In *State v. Porter*, 698 A.2d 739 (1997), the Connecticut Supreme Court held the *Daubert* approach should govern the admissibility of scientific evidence and expressed factors to be considered in assessing evidence.) *Connecticut v. Carlson*, 720 A.2d 886 (Conn. Super. Ct. 1998).

II. Police Officer Testimony Needed to Admit HGN Test Result
Must lay a proper foundation with a showing that the officer administering the test had the necessary qualifications and followed proper procedures.


**III. Purpose and Limits of HGN**

HGN test results can be used to establish probable cause to arrest in a criminal hearing.


**DELAWARE**

**I. Evidentiary Admissibility**

HGN evidence is scientific and must satisfy the Delaware Rules of Evidence standard.


HGN evidence is acceptable scientific testimony under the Delaware Rules of Evidence.

*Ruthardt*, 680 A.2d at 362.

**II. Police Officer Testimony Needed to Admit HGN Test Result**

Police officer may be qualified as an expert to testify about the underlying scientific principles that correlate HGN and alcohol. Delaware police receiving three-day (twenty-four hour) instruction on HGN test administration are not qualified to do this.

*Ruthardt*, 680 A.2d at 361-62.

Police officer testimony about training and experience alone, without expert testimony, is not enough foundation to admit HGN test results.


**III. Purpose and Limits of HGN**

HGN test results admissible to show probable cause in a criminal hearing.

*Ruthardt*, 680 A.2d at 355.

HGN test results admissible to show probable cause in a civil hearing.


HGN test results cannot be used to quantify the defendant’s BAC. However, they can be used as substantive evidence that the defendant was “under the influence of intoxicating liquor.” *Ruthardt*, 680 A.2d at 361-62.
DISTRICT OF COLUMBIA

I. Evidentiary Admissibility
The Court does not address this issue.

II. Police Officer Testimony Needed to Admit HGN Test Result
The Court used the case law of other jurisdictions to come to the conclusion that the Officer in the case could testify as an expert on the administration and the results of the HGN test. Therefore, in this case, the evidence was properly admitted using the Officer as the expert. See Karamychev v. District of Columbia, 772 A. 2d 806 (D.C. App. 2001).

III. Purpose and Limits of HGN
The Court has not yet addressed this issue.

FLORIDA

I. Evidentiary Admissibility
The 3rd District Court found HGN to be a “quasi-scientific” test. Its application is dependent on a scientific proposition and requires a particular expertise outside the realm of common knowledge of the average person. It does not have to meet the Frye standard because HGN has been established and generally accepted in the relevant scientific community, and has been Frye tested in the legal community. The court took judicial notice that HGN is reliable based on supportive case law from other jurisdictions, numerous testifying witnesses and studies submitted. It is “no longer ‘new or novel’ and there is simply no need to reapply a Frye analysis.” Williams v. Florida, 710 So. 2d 24 (Fla. Dist. Ct. App. 1998).

The 4th District Court found HGN to be a scientific test. However, because it is not novel, the Frye standard is not applicable. However, “[e]ven if not involving a new scientific technique, evidence of scientific tests is admissible only after demonstration of the traditional predicates for scientific evidence including the test’s general reliability, the qualifications of test administrators and technicians, and the meaning of the results.” Without this predicate, “the danger of unfair prejudice, confusion of issues or misleading the jury from admitting HGN test results outweighs any probative value.” The state did not establish the appropriate foundation for the admissibility of HGN test results.


II. Police Officer Testimony Needed to Admit HGN Test Result
“We take judicial notice that HGN test results are generally accepted as reliable and thus are admissible into evidence once a proper foundation has been laid that the test was correctly administered by a qualified DRE [Drug Recognition Expert].”

Williams, 710 So. 2d at 32.
Also see *Bown v. Florida*, 745 So. 2d 1108 (Fl. Dist. Ct. App. 1999) which expands *Williams*. Allows trooper to explain HGN, but district requires confirmatory blood, breath or urine test before admitting HGN into evidence.

No evidence presented as to the police officer’s qualifications nor administration of the HGN test in this case. *Meador*, 674 So. 2d at 835.

**III. Purpose and Limits of HGN**

The HGN test results alone, in the absence of a chemical analysis of blood, breath, or urine, are inadmissible to trigger the presumption provided by the DUI statute, and may not be used to establish a BAC of .08 percent or more. *Williams*, 710 So. 2d at 36.

**GEORGIA**

**I. Evidentiary Admissibility**


HGN testing is judicially noticed as a scientifically reliable test and therefore expert testimony is no longer required before the test results can be admitted.


**II. Police Officer Testimony Needed to Admit HGN Test Result**

Police officer, who received specialized training in DUI detection and worked with a DUI task force for two years, was permitted to testify that, in his opinion, defendant was under the influence. *Sieveking v. Georgia*, 469 S.E.2d 235, 219-20 (Ga. Ct. App. 1996).

A Police officer who testifies to the results, administration, and procedure of HGN may be cross-examined about those areas even if the state only offers him as a POST-certified officer. This is because the analysis and expertise needed for HGN go far beyond those needed by a lay person who observes the walk and turn or one leg stance tests.


**III. Purpose and Limits of HGN**

HGN test can be admitted to show that the defendant “was under the influence of alcohol to the extent that it was less safe for him to drive.” *Sieveking*, 469 S.E.2d at 219.

**HAWAII**

**I. Evidentiary Admissibility**

HGN is a scientific test. The HGN test is reliable under the Hawaii Rules of Evidence and admissible as “evidence that police had probable cause to believe that a defendant was DUI.” Judicial notice of the “validity of the principles underlying HGN testing and the reliability of HGN test results” is appropriate. HGN test results can be admitted into evidence if the officer administering the test was duly qualified to conduct the test and the test was performed properly. *Hawaii v. Ito*, 978 P.2d 191 (Haw. Ct. App. 1999).
II. Police Officer Testimony Needed to Admit HGN Test Result

Before HGN test results can be admitted into evidence in a particular case, however, it must be shown that (1) the officer administering the test was duly qualified to conduct and grade the test; and (2) the test was performed properly in the instant case. Hawaii v. Ito, 978 P.2d 191 (Haw. Ct. App. 1999), See also Hawaii v. Toyomura, 904 P.2d 893, 911 (Haw. 1992) and Hawaii v. Montalbo, 828 P2d. 1274, 1281 (Haw. 1992).

III. Purpose and Limits of HGN

HGN test can be admitted as “evidence that police had probable cause to believe that a defendant was DUI.” Hawaii v. Ito, 978 P.2d 191 (Haw. Ct. App. 1999).

IDAHO

I. Evidentiary Admissibility


II. Police Officer Testimony Needed to Admit HGN Test Result

Officer may testify as to administration of HGN test, but not correlation of HGN and BAC.


III. Purpose and Limits of HGN

“HGN test results may not be used at trial to establish the defendant’s blood alcohol level. Although we note that in conjunction with other field sobriety tests, a positive HGN test result does supply probable cause for arrest, standing alone that result does not provide proof positive of DUI….“ Garrett, 811 P.2d at 493.

HGN may be “admitted for the same purpose as other field sobriety test evidence -- a physical act on the part of [defendant] observed by the officer contributing to the cumulative portrait of [defendant] intimating intoxication in the officer's opinion.”

Gleason, 844 P.2d at 695.

ILLINOIS

I. Evidentiary Admissibility

HGN meets Frye standard of admissibility.


Despite the ruling of the Buening appellate court, the Fourth District Court of Appeals declined to recognize HGN’s general acceptance without a Frye hearing. The court criticized the Buening court for taking judicial notice of HGN’s reliability based on the decisions of other jurisdictions. People v. Kirk, 681 N.E.2d 1073, 1077 (Ill. App. Ct. 1997).
The state supreme court held that the state was no longer required to show than an HGN test satisfied the Frye standard before introducing the results of the test into evidence. Absent proof by the defense that the HGN test was unsound, the State only had to show that the officer who gave the test was trained in the procedure and that the test was properly administered. *The People of the State of Illinois v. Linda Basler*, 740 N.E.2d 1 (Ill. 2000), 2000 Ill. LEXIS 1698 (Ill. 2000). (Plurality Opinion) According to Fourth Circuit, a Frye hearing must be held for HGN to be admitted. *People v. Herring*, 762 N.E.2d 1186.

**II. Police Officer Testimony Needed to Admit HGN Test Result**

“A proper foundation should consist of describing the officer’s education and experience in administering the test and showing that the procedure was properly administered.”

*Buening*, 592 N.E.2d at 1227.

**III. Purpose and Limits of HGN**

HGN test results may be used to establish probable cause in a criminal hearing.


HGN test results admissible to show probable cause in a civil hearing.


HGN test results may be used “to prove that the defendant is under the influence of alcohol.” *Buening*, 592 N.E.2d at 1228.

**INDIANA**

**I. Evidentiary Admissibility**

Results of properly administered HGN test are admissible to show impairment which may be caused by alcohol and, when accompanied by other evidence, will be sufficient to establish probable cause to believe a person may be intoxicated. *Cooper v. Indiana*, 751 N.E.2d 900, 903 (Ind. Ct. App. Feb. 2002)

**II. Police Officer Testimony Needed to Admit HGN Test Result**

The proper foundation for admitting HGN evidence should consist of describing the officer’s education and experience in administering the test and showing that the procedure was properly administered. *Cooper*, 751 N.E.2d at 903.

The question of whether a trained officer might express an opinion that defendant was intoxicated based upon the results of field sobriety tests was not before the court, and thus, the court expressed no opinion concerning the admissibility of such testimony.

*Cooper*, 751 N.E. 2d at 902, n. 1.

**III. Purpose and Limits of HGN**

HGN test results, when accompanied by other evidence, will be sufficient to establish probable cause that the person may be intoxicated. *Cooper*, 751 N.E.2d at 903.

**IOWA**
I. Evidentiary Admissibility

HGN admissible as a field test under the Iowa Rules of Evidence. “[T]estimony by a properly trained police officer with respect to the administration and results of the horizontal gaze nystagmus test are admissible without need for further scientific evidence.”

State v. Murphy, 451 N.W.2d 154, 158 (Iowa 1990).

II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer may testify about HGN test results under Rule 702 if the officer is properly trained to administer the test and objectively records the results.

Murphy, 451 N.W.2d at 158.

III. Purpose and Limits of HGN

HGN test results may be used as an indicator of intoxication.

Murphy, 451 N.W.2d at 158.

KANSAS

I. Evidentiary Admissibility

HGN must meet Frye standard of admissibility and a Frye hearing is required at the trial level. There was no Frye hearing conducted and the appellate court refused to make a determination based on the record it had. State v. Witte, 836 P.2d 1110, 1121 (Kan. 1992).

HGN test has not achieved general acceptance within the relevant scientific community and its exclusion was appropriate. State v. Chastain, 960 P.2d 756 (Kan. 1998).

II. Police Officer Testimony Needed to Admit HGN Test Result

The Court did not address this issue.

III. Purpose and Limits of HGN

The Court did not address this issue.

KENTUCKY

I. Evidentiary Admissibility

HGN test results admitted due to defendant’s failure to object.


II. Police Officer Testimony Needed to Admit HGN Test Result

The Court did not address this issue.

III. Purpose and Limits of HGN

The Court did not address this issue.

LOUISIANA
I. Evidentiary Admissibility

HGN meets Frye standard of admissibility and with proper foundation my be admitted as evidence of intoxication.


The standard of admissibility for scientific evidence is currently the Louisiana Rules of Evidence. State v. Foret, 628 So. 2d 1116 (La. 1993).

II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer may testify as to training in HGN procedure, certification in the administration of HGN test and that the HGN test was properly administered.

Armstrong, 561 So. 2d at 887.

III. Purpose and Limits of HGN

The HGN test may be used by the officer “to determine whether or not he [needs] to ‘go any further’ and proceed with other field tests.” Breitung, 623 So. 2d at 25.

HGN test results may be admitted as evidence of intoxication.

Armstrong, 561 So. 2d at 887.

MAINE

I. Evidentiary Admissibility

Because the HGN test relies on greater scientific principles than other field sobriety tests, the reliability of the test must first be established. Either Daubert or Frye standard must be met. State v. Taylor, 694 A.2d 907, 912 (Me. 1997).

The Maine Supreme Court took judicial notice of the reliability of the HGN test to detect impaired drivers. Taylor, 694 A. 2d at 912.

II. Police Officer Testimony Needed to Admit HGN Test Result

“A proper foundation shall consist of evidence that the officer or administrator of the HGN test is trained in the procedure and the [HGN] test was properly administered.”

Taylor, 694 A.2d at 912.

III. Purpose and Limits of HGN

HGN test results may only be used as “evidence of probable cause to arrest without a warrant or as circumstantial evidence of intoxication. The HGN test may not be used by an officer to quantify a particular blood alcohol level in an individual case.”

Taylor, 694 A.2d at 912.
MARYLAND

I. Evidentiary Admissibility


II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer must be properly trained or certified to administer the HGN test. [NOTE: In *Schultz*, the police officer failed to articulate the training he received in HGN testing and the evidence was excluded.]

*Schultz*, 664 A.2d at 77.

III. Purpose and Limits of HGN

HGN testing may not be used to establish a specific blood alcohol level.


MASSACHUSETTS

I. Evidentiary Admissibility

HGN is scientific and is admissible on a showing of either general acceptance in the scientific community or reliability of the scientific theory. *See Commonwealth v. Lanigan*, 641 N.E.2d 1342 (Mass. 1994). HGN test results are inadmissible until the Commonwealth

introduces expert testimony to establish that the HGN test satisfies one of these two standards. *Commonwealth v. Sands*, 675 N.E.2d 370, 373 (Mass. 1997).

II. Police Officer Testimony Needed to Admit HGN Test Result

“There must be a determination as to the qualification of the individual administering the HGN test and the appropriate procedure to be followed.” In this case there was no testimony as to these facts, thus denying the defendant the opportunity to challenge the officer’s qualifications and administration of the test. *Sands*, 675 N.E.2d at 373.

III. Purpose and Limits of HGN

The Court did not address this issue.

MICHIGAN

I. Evidentiary Admissibility

II. Police Officer Testimony Needed to Admit HGN Test Result

Only foundation necessary for the introduction of HGN test results is evidence that the police officer properly performed the test and that the officer administering the test was qualified to perform it. Berger, 551 N.W.2d at 424.

III. Purpose and Limits of HGN

HGN test results are admissible to indicate the presence of alcohol. Berger, 551 N.W.2d at 424 n.1.

MINNESOTA

I. Evidentiary Admissibility

Court found that HGN meets the Frye standard of admissibility.

State v. Klawitter, 518 N.W.2d 577, 585 (Minn. 1994).

II. Police Officer Testimony Needed to Admit HGN Test Result

Police officers must testify about their training in and experience with the HGN test.

See generally Klawitter, 518 N.W.2d at 585-86.

III. Purpose and Limits of HGN

HGN admissible as evidence of impairment as part of a Drug Evaluation Examination in the prosecution of a person charged with driving while under the influence of drugs.

See generally Klawitter, 518 N.W.2d at 585.

MISSISSIPPI

I. Evidentiary Admissibility

HGN is a scientific test. However, it is not generally accepted within the relevant scientific community and is inadmissible at trial in the State of Mississippi.

Young v. City of Brookhaven, 693 So.2d 1355, 1360-61 (Miss. 1997).

II. Police Officer Testimony Needed to Admit HGN Test Result

Police officers cannot testify about the correlation between the HGN test and precise blood alcohol content. Young, 693 So.2d at 1361.

III. Purpose and Limits of HGN

HGN test results are admissible only to prove probable cause to arrest.

Young, 693 So.2d at 1361.

HGN test results cannot be used as scientific evidence to prove intoxication or as a mere showing of impairment. Young, 693 So.2d at 1361.
MISSOURI

I. Evidentiary Admissibility

Court found that HGN test meets the Frye standard of admissibility. State v. Hill, 865 S.W.2d 702, 704 (Mo. Ct. App. 1993), rev’d on other grounds, State v. Carson, 941 S.W.2d 518, 520 (Mo. 1997).

II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer must be adequately trained and able to properly administer the test. Hill, 865 S.W.2d at 704.

See also, Duffy v. Director of Revenue, 966 S.W. 2d 372 (Mo. Ct. App. 1998). HGN not admitted at trial because the administering officer was not aware of how to properly score the test and interpret its results.

III. Purpose and Limits of HGN

HGN can be admitted as evidence of intoxication. Hill, 865 S.W.2d at 704.

MONTANA

I. Evidentiary Admissibility

Court found that HGN is neither new nor novel; thus, Daubert does not apply. Court still finds that HGN must meet the state’s rules of evidence that are identical to the Federal Rules of Evidence. Hulse v. DOJ, Motor Vehicle Div., 961 P.2d 75, 88 (Mont. 1998).

II. Police Officer Testimony Needed to Admit HGN Test Result

The court held that before an arresting officer may testify as to HGN results, a proper foundation must show that the officer was properly trained to administer the HGN test and that he administered the test in accordance with this training. Before the officer can testify as to the correlation between alcohol and nystagmus, a foundation must be established that the officer has special training in the underlying scientific basis of the HGN test.

Hulse, 961 P.2d 75 (Mont. 1998).

See Also, State v. Crawford, 315 Mont. 480, 68 P.3d 848 (2003), in which the court ruled that the officer’s credentials were sufficient to establish his expertise, along with evidence that he was previously qualified as an expert. They relied on Russette (2002 MT 200), stating that to establish an expert’s qualifications, the proponent of the testimony must show that the expert has special training or education and adequate knowledge on which to base an opinion.

III. Purpose and Limits of HGN

HGN test results admissible as evidence of impairment.

State v. Clark, 762 P.2d 853, 856 (Mont. 1988).
NEBRASKA

I. Evidentiary Admissibility

HGN meets the *Frye* standard for acceptance in the relevant scientific communities, and when the test is given in conjunction with other field sobriety tests, the results are admissible for the limited purpose of establishing impairment that may be caused by alcohol. *State v. Baue*, 607 N.W.2d 191 (Neb. 2000)

II. Police Officer Testimony Needed to Admit HGN Test Result

A police officer may testify to the results of HGN testing if it is shown that the officer has been adequately trained in the administration and assessment of the HGN test and has conducted the testing and assessment in accordance with that training. *State v. Baue*, 607 N.W.2d 191 (Neb. 2000)

III. Purpose and Limits of HGN

“Testimony concerning HGN is admissible on the issue of impairment, provided that the prosecution claims no greater reliability or weight for the HGN evidence than it does for evidence of the defendant's performance on any of the other standard field sobriety tests, and provided further that the prosecution makes no attempt to correlate the HGN test result with any particular blood-alcohol level, range of blood-alcohol levels, or level of impairment.” *State v. Baue*, 607 N.W.2d 191 (Neb. 2000) (quoting *Ballard v. State*, 955 P.2d 931, 940 (Alaska App. 1998))

NEW HAMPSHIRE

I. Evidentiary Admissibility

In *State v. Dahood* (Dec. 20, 2002), the N.H. Supreme Court ruled that the HGN test is admissible under N.H. Rule of Evidence 702 and *Daubert* for the limited purpose of providing circumstantial evidence of intoxication. HGN test is a scientifically reliable and valid test.

N.H. Supreme Court ruled their findings binding in *Dahood* and that courts “will not be required to establish the scientific reliability of the HGN.”

II. Police Officer Testimony Needed to Admit HGN Test Result

“Since we have already determined that the scientific principles underlying the HGN test are reliable, a properly trained and qualified police officer may introduce the HGN test results at trial.” *State v. Dahoo*, 2002 N.H. LEXIS 179.

III. Purpose and Limits of HGN

“HGN results cannot be introduced at trial for the purpose of establishing a defendant’s BAC level…. [T]he results are not sufficient alone to establish intoxication.”

*State v. Dahoo*, Id.
NEW JERSEY

I. Evidentiary Admissibility

In New Jersey, the party offering the results of a scientific procedure into evidence must comply with Frye and show that the procedure is generally accepted in the relevant scientific communities. A party may prove this general acceptance via “(1) testimony of knowledgeable experts[,] (2) authoritative scientific literature[, or] (3) [p]ersuasive judicial decision.” Based on the testimony of Dr. Marcelline Burns and Dr. Jack Richman, the Court found the HGN test to be generally accepted and the results thus admissible. The Court also noted the “significant number” of jurisdictions that have accepted the HGN test as admissible scientific evidence. State v. Maida, 2000 N.J. Super. LEXIS 276 (N.J. Super. Ct. Law Div. 2000).

*But See, State v. Doriguzzi, 760 A.2d 336 (N.J. Super. 2000), which held that HGN is scientific evidence that must meet Frye Standard. However, in each trial, sufficient foundation evidence must be laid by expert testimony to assure defendants that a conviction for DUI, when based in part on HGN testing, is grounded in reliable scientific data. In this case, the appellate court reversed defendant’s conviction because at trial no such foundation was presented. The court found that because HGN testing has not achieved general acceptance in the community, it is not a matter of which a court can take judicial notice.

II. Police Officer Testimony Needed to Admit HGN Test Result

The Court did not address this issue.

III. Purpose and Limits of HGN

The Court found the HGN test admissible “as a reliable scientific indicator of likely intoxication.”

NEW MEXICO

I. Evidentiary Admissibility

HGN is a scientific test. New Mexico follows the Daubert standard, which requires a showing of reliability before scientific evidence can be admitted. The court held that a scientific expert must testify to the underlying scientific reliability of HGN and that a police officer cannot qualify as a scientific expert. Because the State failed to present sufficient evidence regarding the HGN test’s reliability, the court remanded the case stating it would be appropriate for the trial court, on remand, to make the initial determination of whether HGN testing satisfies Daubert. In addition, the court found HGN to be “beyond common and general knowledge” and declined to take judicial notice of HGN reliability. State v. Torres, 976 P.2d 20 (N.M. 1999).

State v. Lasworth, 42 P.3d 844 (Ct. App. N.M. 2001), cert. denied (2002). Results of HGN test were inadmissible at trial (State v. Torres, 976 P.2d 20 (N.M. 1999). The State needed to prove that HGN was both valid and reliable.

State called Dr. Marceline Burns as a witness (reliability) but did not call an expert in a discipline such as biology or medicine to explain how the amount of alcohol a person consumes correlates with HGN (validity).
II. Police Officer Testimony Needed to Admit HGN Test Result

Police officers can qualify as non-scientific experts based on their training and experience. Non-scientific experts may testify about the administration of the test and specific results of the test provided another scientific expert first establishes the reliability of the scientific principles underlying the test. In order to establish the “technical or specialized knowledge” required to qualify as an expert in the administration of the HGN test, “there must be a showing: (1) that the expert has the ability and training to administer the HGN test properly, and (2) that the expert did, in fact, administer the HGN test properly at the time and upon the person in question.” State v. Torres, 976 P.2d 20 (N.M. 1999).

State v. Lasworth, 42 P.3d 844 (Ct. App. N.M. 2001), cert. denied (2002). Court believed that state had to show that presence of HGN (BAC above .08) correlates with diminishment of driver’s mental or physical driving skills (which it failed to do) and a correlation between presence of HGN and BAC above or below .08 (which it did through testimony of Dr. Burns). Court did not preclude use of results of HGN to establish probable cause for arrest or to establish grounds for administering a chemical BAC test.

III. Purpose and Limits of HGN

The Court did not address this issue.

NEW YORK

I. Evidentiary Admissibility

Prue holds that HGN test results are admissible under Frye standard of “general acceptance.” People v. Prue, Indictment No. I-5-2001, Franklin County Court (November 2001).

In Gallup, the court said that it was only necessary to conduct a foundational inquiry into the techniques and the tester’s qualifications for admissibility. People v. Gallup, Memorandum and order #13094, 302 A.D.2d 681 (3rd Dept)( 2003).

The Court allowed the introduction of HGN and the results because it was properly administered and the burden of establishing that HGN is a reliable indicator of intoxication is generally accepted in the relevant scientific community was satisfied.

People v. William Miley, NYLJ 12/6/02 p.30 col. 6 (Nassau Co. Ct 2002).

II. Police Officer Testimony Needed to Admit HGN Test Result

The People must lay a proper evidentiary foundation in order for HGN results to be admissible at trial.
III. Purpose and Limits of HGN

The Court held that HGN is generally accepted in the relevant scientific community as a reliable indicator of intoxication.

NORTH CAROLINA

I. Evidentiary Admissibility

HGN is a scientific test. It “does not measure behavior a lay person would commonly associate with intoxication but rather represents specialized knowledge that must be presented to the jury by a qualified expert.” As a result, “until there is sufficient scientifically reliable evidence as to the correlation between intoxication and nystagmus, it is improper to permit a lay person to testify as to the meaning of HGN test results.”


II. Police Officer Testimony Needed to Admit HGN Test Result

Testimony of one police officer, whose training consisted of a “forty hour training class dealing with the HGN test”, was inadequate foundation for admission of HGN test results.


III. Purpose and Limits of HGN


NORTH DAKOTA

I. Evidentiary Admissibility

Court found that HGN test is admissible as a standard field sobriety test.

*City of Fargo v. McLaughin*, 512 N.W.2d 700, 706 (N.D. 1994).

II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer must testify as to training and experience and that the test was properly administered. *City of Fargo*, 512 N.W.2d at 708.

III. Purpose and Limits of HGN

“ . . . HGN test results admissible only as circumstantial evidence of intoxication, and the officer may not attempt to quantify a specific BAC based upon the HGN test.”

*City of Fargo*, 512 N.W.2d at 708.

OHIO

I. Evidentiary Admissibility

HGN test is objective in nature and does not require an expert interpretation.

Court determined that HGN was a reliable indicator of intoxication without specifically ruling on whether HGN meets Frye or some other standard of admissibility.


Court held that SFSTs, including HGN, must be administered in *strict compliance* with NHTSA’s directives in order for the test results to be admissible.


II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer need only testify to training in HGN procedure, knowledge of the test and ability to interpret results. *Bresson*, 554 N.E.2d at 1336.

III. Purpose and Limits of HGN

HGN can be used to establish probable cause to arrest and as substantive evidence of a defendant's guilt or innocence in a trial for DUI, but not to determine defendant's BAC.

*Bresson*, 554 N.E.2d at 1336.

OKLAHOMA

I. Evidentiary Admissibility


II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer testified to training on how to administer HGN test and how the test was administered in this case. Officer also testified as to his training in analyzing HGN test results. *Yell*, 856 P.2d at 997.

III. Purpose and Limits of HGN

If HGN testing was found to satisfy the *Frye* standard of admissibility, HGN test results would be considered in the same manner as other field sobriety test results. HGN test results are inadmissible as scientific evidence creating a presumption of intoxication.

*Yell*, 856 P.2d at 997.

OREGON

I. Evidentiary Admissibility

HGN test results are admissible under the Oregon Rules of Evidence. HGN test results are scientific in nature, are relevant in a DUI trial, and are not unfairly prejudicial to the defendant. *State v. O'Key*, 899 P.2d 663, 687 (Or. 1995).
II. Police Officer Testimony Needed to Admit HGN Test Result

“Admissibility is subject to a foundational showing that the officer who administered the test was properly qualified, that the test was administered properly, and that the test results were recorded accurately.” O’Key, 899 P.2d at 670.

III. Purpose and Limits of HGN

“… HGN test results are admissible to establish that a person was under the influence of intoxicating liquor, but is not admissible…to establish a person’s BAC….”

O’Key, 899 P.2d at 689-90.

Officer may not testify that, based on HGN test results, the defendant’s BAC was over .10.


PENNSYLVANIA

I. Evidentiary Admissibility

The state laid an inadequate foundation for the admissibility of HGN under the Frye/Topa standard.


Testimony of police officer is insufficient to establish scientific reliability of HGN test.

Moore, 635 A.2d at 692.

Miller, 532 A.2d at 1189-90.

Testimony of behavioral optometrist did not establish general acceptance of HGN test.

Apollo, 603 A.2d at 1027-28.

II. Police Officer Testimony Needed to Admit HGN Test Result

County detective certified as HGN instructor. Court did not comment on whether this would be enough foundation to allow the detective to testify about HGN test results. Moore, 635 A.2d 629.

Police officer had one-day course on HGN. Court did not comment on whether this would be enough foundation to allow the officer to testify about HGN test results. Miller, 603 A.2d at 1189.

III. Purpose and Limits of HGN

Not addressed by court.
SOUTH CAROLINA

I. Evidentiary Admissibility

HGN admissible in conjunction with other field sobriety tests. By implication, HGN is not regarded as a scientific test. State v. Sullivan, 426 S.E.2d 766, 769 (S.C. 1993).

II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer given twenty hours of HGN training. Sullivan, 426 S.E.2d at 769.

III. Purpose and Limits of HGN

HGN test results admissible “to elicit objective manifestations of soberness or insobriety . . . Evidence from HGN tests is not conclusive proof of DUI. A positive HGN test result is to be regarded as merely circumstantial evidence of DUI. Furthermore, HGN test shall not constitute evidence to establish a specific degree of blood alcohol content.” Sullivan, 426 S.E.2d at 769.

SOUTH DAKOTA

I. Evidentiary Admissibility

If it can be shown that a horizontal gaze nystagmus test was properly administered by a trained officer, such evidence should be admitted for a jury to consider at trial along with evidence of the other accepted field sobriety tests administered in South Dakota. STATE v. HULLINGER, 2002 SD 83; 649 N.W.2d 253 (S.D.S.Ct. 2002); 2002 S.D. LEXIS 99

II. Police Officer Testimony Needed to Admit HGN Test Result

Officer may testify if properly trained and test properly administered. At the pretrial hearing, the State presented three witnesses: 1) Monte Farnsworth, training director for the Office of Highway Safety at the Division of Criminal Investigation Law Enforcement Training Academy; 2) Deputy Ludwig; and 3) Dr. Larry Menning, optometrist and expert witness. South Dakota follows a Daubert standard in use of expert witnesses.

III. Purpose and Limits of HGN

The Court did not address this issue.

TENNESSEE

I. Evidentiary Admissibility

HGN is a scientific test. To be admissible at trial, such evidence must satisfy the requirements of Tenn. Rules of Evidence 702 and 703. State provided an inadequate amount of evidence to allow the court to conclude that HGN evidence meets this standard.

State v. Murphy, 953 S.W.2d 200 (Tenn. 1997).
II. Police Officer Testimony Needed to Admit HGN Test Result

HGN must be offered through an expert witness. To qualify as an expert, a police officer must establish that he is qualified by his “knowledge, skill, experience, training or education” to provide expert testimony to “substantially assist the trier of fact to understand the evidence or determine a fact in issue.” Although the court did not rule out the possibility that the officer can be considered an expert, the court set a high level of proof. In this case, the court felt that although the officer had attended law enforcement training in DUI offender apprehension and the HGN test, this training was not enough to establish him as an expert. State v. Grindstaff, 1998 Tenn. Crim. App. Lexis 339 (March 23, 1998).

III. Purpose and Limits of HGN

The Court did not address this issue.

TEXAS

I. Evidentiary Admissibility


II. Police Officer Testimony Needed to Admit HGN Test Result

A police officer must qualify as an expert on the HGN test, specifically concerning its administration and technique, before testifying about a defendant’s performance on the test. Proof that the police officer is certified in the administration of the HGN test by the Texas Commission on Law Enforcement Officer Standards and Education satisfies this requirement. Emerson, 880 S.W.2d at 769.

III. Purpose and Limits of HGN

HGN admissible to prove intoxication, but not accurate enough to prove precise BAC. Emerson, 880 S.W.2d at 769.

UTAH

I. Evidentiary Admissibility


II. Police Officer Testimony Needed to Admit HGN Test Result

Police officer need only testify as to training, experience and observations when HGN admitted as a field test. Garcia, 912 P.2d at 1001.

III. Purpose and Limits of HGN

Admissible as any other field sobriety test. Garcia, 912 P.2d at 1000-01.

WASHINGTON

I. Evidentiary Admissibility
It is “undisputed” in the relevant scientific communities that “an intoxicated person will exhibit nystagmus”. HGN testing is not novel and has been used as a field sobriety test for “decades” and is administered the same whether investigating alcohol impairment or drug impairment. Thus, the use of HGN in drug and alcohol impaired driving cases is acceptable. *State v. Baity*, 140 Wn.2d 1, 991 P.2d 1151 (Wash. 2000).

“The *Frye* standard applies to the admission of evidence based on HGN testing, unless . . . the State is able to prove that it rests on scientific principles and uses techniques which are not ‘novel’ and are readily understandable by ordinary persons.” The state failed to present any evidence to this fact and the court declined to take judicial notice of HGN.


**II. Police Officer Testimony Needed to Admit HGN Test Result**

The Court did not address this issue.

**III. Purpose and Limits of HGN**

The Court did not address this issue.

**WEST VIRGINIA**

**I. Evidentiary Admissibility**

The state did not present evidence for the court to reach “the question of whether the HGN test is sufficiently reliable to be admissible.” However, the court did conclude “that even if the reliability of the HGN test is demonstrated, an expert’s testimony as to a driver’s performance on the test is admissible only as evidence that the driver was under the influence. Estimates of blood alcohol content based on the HGN test are inadmissible.” *State v. Barker*, 366 S.E.2d 642, 646 (W. Va. 1988).


**II. Police Officer Testimony Needed to Admit HGN Test Result**

Police officer's training consisted of a one-day, eight-hour training session conducted by the state police. Officer testified to giving the HGN test about 100 times. Court did not reach question of whether this would be enough to allow the officer to testify about the HGN test results. *Barker*, 366 S.E.2d at 644.
III. Purpose and Limits of HGN

HGN test results admissible to show probable cause in a civil hearing.


“If the reliability of the HGN test is demonstrated, an expert's testimony as to a driver's performance on the test is admissible only as evidence that the driver was under the influence,” the same as other field sobriety tests. _Barker_, 366 S.E.2d at 646.

WISCONSIN

I. Evidentiary Admissibility

The court held that the HGN test results are admissible in this case because the test results were not the only evidence. The results were accompanied by the expert testimony of the officer. _State v. Zivcic_, 598 N.W.2d 565 (Wisc. Ct. App. 1999). See also, _State v. Maxon_, 633 N.W. 2d 278 (Wisc. Ct. App. 2001)

II. Police Officer Testimony Needed to Admit HGN Test Result

A police officer who is properly trained to administer and evaluate the HGN test can testify to the test results. A second expert witness is not needed. _State v. Zivcic_, 598 N.W.2d 565 (Wisc. Ct. App. 1999).

III. Purpose and Limits of HGN

The Court did not address this issue.

WYOMING

I. Evidentiary Admissibility

SFSTs, including HGN, are admissible to establish probable cause when administered in _substantial compliance_ with NHTSA guidelines. Strict compliance is not necessary. The court took judicial notice of the number of states that allow HGN evidence on the basis of the “officer’s training, experience and ability to administer the test”. _Smith v. Wyoming_, 2000 Wyo. LEXIS 202 (Wyo. October 4, 2000).

II. Police Officer Testimony Needed to Admit HGN Test Result

A police officer that is properly trained to administer and evaluate the HGN test can testify to HGN results. _Smith v. Wyoming_, 2000 Wyo. LEXIS 202 (Wyo. October 4, 2000).

III. Purpose and Limits of HGN

HGN test results are admissible to show probable cause.

I. Evidentiary Admissibility

*U.S. V. Eric D. Horn*, 185 F. Supp. 2d 530 (D. Maryland 2002) In this case, U.S. District Court in Maryland made the first application of the newly revised FRE 702 to the HGN and other SFSTs.

Results of properly administered WAT, OLS and HGN, SFSTs may be admitted into evidence in a DWI/DUI case only as circumstantial evidence of intoxication or impairment but not as direct evidence of specific BAC.

Officer must first establish his qualifications to administer the test - training and experience, not opinion about accuracy rate of test or causal connection between alcohol consumption and exaggerated HGN.

Government may prove causal connection by: judicial notice, expert testimony, or learned treatise. Horn may prove other causes by: judicial notice, cross-examination of state’s expert, defense expert, or learned treatise.

*U.S. V. Daras, 1998 WL 726748 (4th Cir. 1998)(Unpublished opinion).* WAT and OLS were not scientific so no expert needed. Court would have applied *Daubert* to HGN test, but there was no need to because breathalyzer, WAT and OLS were sufficient.

HGN test was admitted as part of series of field tests. Its admission was not challenged on appeal. *U.S. v. Van Griffin*, 874 F.2d 634 (9th Cir. 1989).

II. Police Officer Testimony Needed to Admit HGN Test Result

Foundation for HGN must address validity and reliability under FRE 702. In *Horn*, prosecution had a medical doctor and a police officer, but defense used behavioral psychologist to attack HGN literature of Dr. Marceline Burns and others.

III. Purpose and Limits of HGN

SFSTs may be admitted into evidence in a DWI/DUI case only as circumstantial evidence of intoxication or impairment but not as direct evidence of specific BAC. *Horn*.

Properly qualified, Officer may give opinion of intoxication or impairment by alcohol. *Horn*. 
Note: The following states were not listed above due to a lack of case law discussion on HGN: Colorado, Nevada, Rhode Island, Vermont (HGN was mentioned in the context of a refusal being admissible as evidence of probative guilt. State v. Blouin, 168 Vt. 119 (Vt. 1998) Virginia.

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Or

Visit their website www.ndaa-apri.org.
1. Anderson, Schweitz & Snyder, Field Evaluation of Behavioral Test Battery for DWI, U.S. Dept. of Transportation Rep. No. DOT-HS-806-475 (1983) (field evaluation of the Standardized Field Sobriety Test battery (HGN, one-leg stand, and walk and turn) conducted by police officers from four jurisdictions indicated that the battery was approximately 80% effective in determining BAC above and below .10 percent).

2. Aschan, Different Types of Alcohol Nystagmus, 140 ACTA OTOLARYNGOL SUPP. 69 (Sweden 1958) ("From a medico-legal viewpoint, simultaneous recording of AGN (Alcohol Gaze Nystagmus) and PAN (positional alcoholic nystagmus) should be of value, since it will show in which phase the patient's blood alcohol curve is...").


4. Aschan, Bergstedt, Goldberg & Laurell, Positional Nystagmus in Man During and After Alcohol Intoxication, 17 Q.J. OF STUD. ON ALCOHOL, Sept. 1956, at 381. Study distinguishing two types of alcohol-induced nystagmus, PAN (positional alcoholic nystagmus) I and PAN II, found intensity of PAN I, with onset about one-half hour after alcohol ingestion, was proportional to amount of alcohol taken.


8. Burns, The Robustness of the Horizontal Gaze Nystagmus (HGN) Test, U.S. Dept. of Transportation 2004. Concludes that HGN as used by law enforcement is a robust procedure and the data obtained in this report does not support changes or revisions to the current testing or procedure

10. Citek, Ball and Rutledge, *Nystagmus Testing in Intoxicated Individuals*, Vol. 74, No. 11, Nov. 2003, Optometry, established that the HGN test administered in the standing, seated, and supine postures is able to discriminate impairment at criterion BAC’s of 0.08% and 0.10%.

11. Compton, *Use of the Gaze Nystagmus Test to Screen Drivers at DWI Sobriety Checkpoints*, U.S. Dept. of Transportation (1984) (field evaluation of HGN test administered to drivers through car window in approximately 40 seconds: "the nystagmus test scored identified 95% of the impaired drivers" at 2; 15% false positive for sober drivers, id.).


13. Goldberg, *Effects and After-Effects of Alcohol, Tranquilizers and Fatigue on Ocular Phenomena*, ALCOHOL AND ROAD TRAFFIC 123 (1963) (of different types of nystagmus, alcohol gaze nystagmus is the most easily observed).

14. Helzer, *Detection DUIs Through the Use of Nystagmus*, LAW AND ORDER, Oct. 1984, at 93 (nystagmus is "a powerful tool for officers to use at roadside to determine BAC of stopped drivers...(O)fficers can learn to estimate BACs to within an average of 0.02 percent of chemical test readings." Id. at 94).

15. L.R. Erwin, *DEFENSE OF DRUNK DRIVING CASES* (3d ed. 1985) ("A strong correlation exists between the BAC and the angle of onset of (gaze) nystagmus." Id. at 8.15A(3).


17. Misoi, Hishida & Maeba, *Diagnosis of Alcohol Intoxication by the Optokinetic Test*, 30 Q.J. OF STUD. ON ALCOHOL 1 (March-June 1969) (optokinetic nystagmus, ocular adaptation to movement of object before eyes, can also be used to detect central nervous system impairment caused by alcohol. Optokinetic nystagmus is inhibited at BAC of only .051 percent and can be detected by optokinetic nystagmus test. Before dosage subjects could follow a speed of 90 degrees per second; after, less than 70 degrees per second).


ingestion of alcohol and the onset of various kinds of nystagmus "appears to be well documented." Id. "While nystagmus appears to be useful as a roadside sobriety test, at this time, its use to predict a person's blood alcohol level does not appear to be warranted." Id. at 22).


22. Oosterveld, Meineri & Paolucci, Quantitative Effect of Linear Acceleration on Positional Alcohol Nystagmus, 45 AEROSPACE MEDICINE, July 1974, at 695 (G-loading brings about PAN even when subject has not ingested alcohol; however when subjects ingested alcohol, no PAN was found when subjects were in supine position, even with G-force at 3).


26. Savolainen, Riihimaki, Vaheri & Linnoila, Effects of Xylene and Alcohol on Vestibular and Visual Functions in Man, SCAND. J. WORK ENVIRON. HEALTH 94 (Sweden 1980) (abstract available on DIALOG, file 172: Embase 1980-81 on file 5: Biosis Previews 1981-86) (the effects of alcohol on vestibular functions (e.g., positional nystagmus) were dose-dependent).

27. Seelmeyer, Nystagmus, A Valid DUI Test, LAW AND ORDER, July 1985, at 29 (Horizontal Gaze Nystagmus test is used in "at least one law enforcement agency in each of the 50 states" and is "a legitimate method of establishing probable cause." Id.).


29. Tharp, Burns & Moskowitz, Circadian Effects on Alcohol Gaze Nystagmus (paper presented at 20th annual meeting of Society for Psychophysiological Research), abstract in 18 PSYCHOPHYSIOLOGY, March 1981 (highly significant correlation between angle of onset of AGN and BAC).

30. Tharp, Burns & Moskowitz, Development and Field Test of Pschophysical Tests for DWI Arrests, U.S. Dept. of Transportation Rep. No. DOT-HS-805-864 (1981) (standardized procedures for administering and scoring the SCRI three-test battery; participating officers able to classify 81% of volunteers above or below .10).
31. Umeda & Sakata, Alcohol and the Oculomotor System, 87 ANNALS OF OTOLOGY, RHINOLOGY & LARYNGOLOGY, May-June 1978, at 392 (in volunteers whose "caloric eye tracking pattern" (CETP) was normal before alcohol intake, influence of alcohol on oculomotor system appeared consistently in the following order: (1) abnormality of CETP, (2) positional alcohol nystagmus, (3) abnormality of eye tracking pattern, (4) alcohol gaze nystagmus).


Participant Manual

Drug Recognition Expert Course

Session 4
Overview of Drug Recognition Expert Procedures
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Upon successfully completing this session the participant will be able to:

- Name the components of the Drug Evaluation and Classification program drug influence evaluation.
- State the purpose of each component.
- Describe the activities performed during each component.
- Correctly answer the “topics for study” questions at the end of this session.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES

A. Components of the Drug Evaluation and ........ Instructor-Led Presentations / Demonstrations
   Classification Procedure
B. Interview of the Arresting Officer................................. Video Presentations
C. The Preliminary Examination............................................................... Reading Assignments
D. Examinations of the Eyes
E. Divided Attention Psychological Tests
F. Examinations of Vital Signs
G. Dark Room Checks of Pupil Size
H. Examination of Muscle Tone
I. Examination for Injection Sites
J. Subject Statements
K. Opinion of Evaluator
L. Toxicological Examination
M. Video Demonstration
A. Components of the Drug Evaluation and Classification Process

The Drug Influence Evaluation

The Drug Evaluation and Classification Process is a systematic and standardized method of examining a subject to determine:

• Is the subject impaired?
• Is the impairment resulting from an injury, illness, or drug related?
• If drug related, what category (or categories) of drugs is (or are) the likely cause of the subject’s impairment?

The process is systematic in that it is based on a careful assessment of a variety of observable signs and symptoms that are known to be reliable indicators of drug impairment.

• Some of these observable signs and symptoms relate to the subject’s appearance.
• Some of these observable signs and symptoms relate to the subject’s behavior.
• Some relate to the subject’s performance of carefully administered psychophysical tests.
Drugs impair the subject’s ability to control his or her mind and body.

- Psychophysical tests can disclose that the subject’s ability to control mind and body is impaired.
- The specific manner in which the subject performs the psychophysical tests may help indicate the category or categories of drugs causing the impairment.
- Some of the observable signs and symptoms relate to the subject’s automatic responses to the specific drugs that are present.
- All of these reliable indicators are examined and carefully considered before a judgment is made concerning what categories of drugs are affecting the subject.

The evaluation is standardized in that it is administered the same way, every time.

- Standardization helps to ensure that no mistakes are made.
- No examinations are left out.
- No extraneous or unreliable “indicators” are included.
- Standardization helps to promote professionalism among drug recognition experts.
- Standardization helps to secure acceptance in court.

In such cases, the DRE may still be able to form an opinion based upon the evidence obtained. State v. Cammack, 1997 WL 104913 (Minnesota Ct. Appeals, 1997) ruled that a DRE need not complete the entire 12-step evaluation for an opinion to be admissible so long as there is sufficient admissible evidence.
Drug Influence Evaluation Steps

The Drug Evaluation and Classification drug influence evaluation has twelve components or steps.
Breath Alcohol Test

The Breath Alcohol Test is needed to determine Blood Alcohol Concentration (BAC).

The purpose of the breath test is to determine whether the specific drug, alcohol, may be contributing to the impairment observed in the subject.

Obtaining an accurate measurement of BAC enables the DRE to assess whether alcohol may be the sole cause of the observable impairment, or whether it is likely that some other drug or drugs, or other complicating factors are contributing to the impairment.

The Interview of the Arresting Officer.

In most cases, the subjects you will examine will not be people that you arrested.

The arresting officer may have seen or heard things that would be valuable indicators of the kinds of drugs the subject has ingested.

The arresting officer, in searching the subject, may have uncovered drug related paraphernalia, or even drugs themselves.

The arresting officer also may be able to alert you to important information about the subject’s behavior that could be very valuable for your own safety.
The Preliminary Examination

- The preliminary examination is your first opportunity to observe the subject closely and directly.
- A major purpose of the preliminary examination is to determine if the subject may be suffering from an injury or some other medical condition not necessarily related to drugs.
- The preliminary examination will help you decide whether to continue with the drug influence evaluation, pursue a possible medical complication, or proceed with a DWI (alcohol) case.
- Another major purpose of the preliminary examination is to begin systematically assessing the subject’s appearance, behavior and automatic bodily responses for signs of drug induced impairment.

The preliminary examination consists of a series of questions dealing with possible injuries or medical problems; observations of the subject’s face, speech and breath; pupil size and tracking ability; initial checks of the subject’s eyes; and, an initial examination of the subject’s pulse. While you are assessing the subject’s tracking ability, you can also perform a preliminary assessment of whether Horizontal Gaze Nystagmus is present in the subject’s eyes. In particular, if the Nystagmus or “jerking” is observed, an initial estimation of the angle of onset can be made. The approximate angle of onset may help to determine whether the subject has consumed some drug other than alcohol.
Examinations of the Eyes

Certain drugs produce very easily observable effects on the eyes.

One of the most dramatic of these effects is Nystagmus, which means an involuntary jerking of the eyes.

Persons under the influence of alcohol usually will exhibit Horizontal Gaze Nystagmus, which is an involuntary jerking of the eyes occurring as the eyes gaze to the side.

Alcohol is not the only drug that causes Nystagmus.

Horizontal Gaze Nystagmus is not the only observable effect on the eyes that will be caused by various drugs.
Divided Attention Psychophysical Tests

All drugs that impair driving ability will also impair the subject’s ability to perform certain carefully designed divided attention tests.

These tests are familiar to you in the context of examining alcohol impaired subjects.

The same tests are very valuable for disclosing evidence of impairment due to drugs other than alcohol.

The divided attention tests used in the DRE examination include:

- Modified Romberg Balance,
- Walk and Turn,
- One Leg Stand, and
- Finger to Nose.
Examination of Vital Signs

Many categories of drugs affect the operation of the heart, lungs and other major organs of the body.

These effects show up during examination of the subject’s vital signs.

The vital signs that are reliable indicators of drug influence include blood pressure, pulse, and temperature.
Dark Room Examinations

Many categories of drugs affect how the pupils will appear, and how they respond to light. Certain kinds of drugs will cause the pupils to widen dramatically, or dilate. Some other drugs cause the pupils to narrow, or constrict.

By systematically changing the amount of light entering the subject’s eyes, we can observe the pupils’ appearance and reaction under controlled conditions.

We carry out these examinations in a dark room, using a penlight to control the amount of illumination entering the subject’s eyes.

We use a device called a pupillometer to estimate the size of the subject’s pupils. By lining the circles up along side the subject’s pupil, the pupil’s size can be determined.

Other examinations are also conducted in the darkroom, using the penlight: i.e., examination of the nasal area and mouth for signs of drug use and for concealed contraband.
Examination of Muscle Tone

Evidence of muscle tone can also be observed when taking the subject’s pulse, blood pressure or while examining for injection sites.

Certain categories of drugs can cause the user’s muscles to become markedly tense, and rigid. Others may cause flaccidity, or “rubbery-like” muscle tone.

Evidence of this muscle tone may come to light when the subject attempts to perform the divided attention tests.
Examination for Injection Sites

Certain drugs are commonly injected by their users, via hypodermic needles. Heroin is probably most commonly associated with injection, but several other types of drugs also are injected by many users.

Uncovering injection sites on a subject provides evidence of possible drug use.
Subject’s Statements and Other Observations

At this point in the examination, the DRE may have reasonable grounds to believe that the subject is under the influence of a drug or drugs.

The DRE may also have at least an articulable suspicion as to the category or categories of drugs causing the impairment.

The DRE should proceed to interview the subject to confirm their opinion concerning the drug category or categories involved.

The DRE must carefully record the subject’s statements, and any other observations that may constitute relevant evidence of drug induced impairment.
Opinion of Evaluator

Based on all of the evidence and observations gleaned from the preceding steps, the DRE should be able to reach an informed opinion as to:

- Whether the subject is under the influence of a drug or drugs, and if so,
- The probable category or categories of drugs causing impairment.

The DRE must record a narrative summary of the facts forming the basis for their opinion.

Toxicological Examination

The toxicological examination is a chemical test or tests designed to obtain scientific, admissible evidence to substantiate the DRE’s opinion.

Departmental policy and procedures should be followed in requesting, obtaining and handling the toxicological sample.
B. Interview of the Arresting Officer

The purpose of the interview of the arresting officer is to obtain a summary of the subject’s actions, behaviors, etc. that led to the arrest and the suspicion that drugs other than alcohol may be involved.

*Interview Behavior*

Issues concerning the subject’s behavior:

- Was the subject operating a vehicle?
- What actions, maneuvers, etc. were observed?
- Was there a crash? If yes, was the subject injured?
- Was the subject observed smoking, drinking or eating?
- Was the subject apparently inhaling any substance?
- How did the subject respond to the arresting officer’s stop?
- Did the subject attempt to conceal or throw away any items or materials?
- What has been the subject’s attitude and demeanor during contact with the arresting officer and have there been any changes?
Interview Concerning Subject's Statements

- Has the subject complained of an illness or injury?
- Has the subject used any “street terms” or slang associated with drugs or drug paraphernalia?
- How has the subject responded to the arresting officer’s questions?
- Was the subject’s speech slurred, slow, thick, rapid, mumbled, etc.?
- What, specifically, has the subject said?

Interview: Physical Evidence

Issues concerning physical evidence:

- What items or materials were uncovered during the search of the subject or vehicle?
- Were any smoking paraphernalia uncovered?
- Were any injection materials, i.e., needles, syringes, leather straps, rubber tubes, spoons, bottle caps, etc. found?
- Were there any balloons, plastic bags, small metal foil wrappings, etc. found?
- What was the subject’s blood alcohol concentration?
C. Overview of the Preliminary Examination

The preliminary examination consists of:

- Questions.
- Observations of face, breath, and speech.
- Initial checks of the eyes.
- The initial check of the subject’s pulse.

Preliminary Examination Questions

The questions deal with injuries or medical problems the subject may have. They include:

- Are you sick or injured?
- Do you have any physical defects?
- Are you diabetic or epileptic?
- Do you take insulin?
- Are you under a doctor’s or dentist’s care?
- Are you taking any medications or drugs?
Initial Checks of the Eyes

The initial checks of the subject’s eyes include several particularly important items.

Check of the size of each pupil. The initial examination of the eyes may reveal signs of injury or illness. A difference in pupil size of greater than 0.5 mm may indicate an injury or existing medical condition.

Assessment of the ability of the eyes to track a moving object.

The presence of Nystagmus indicates the possible presence of certain categories of drugs.

Initial estimation of the angle of onset of Horizontal Gaze Nystagmus.

The approximate angle of onset may indicate the presence of some drug other than alcohol.

If the subject has also ingested some other drug that also causes Nystagmus, the angle of onset may occur even earlier than the Blood Alcohol Concentration would indicate.

Example: Suppose you are examining a subject who has an angle of onset at 45 degrees.

Based on that alone, you would expect the person's BAC to be in the .05 - .08 percent range. But if that subject has also ingested a Dissociative Anesthetic, the onset could occur much earlier, perhaps as soon as the eyes start to move to the side.

For example: Cannabis, Narcotic Analgesics, CNS Stimulants and Hallucinogens do not cause Nystagmus, and will not affect the angle of onset.
D. Examinations of the Eyes

Eye Examinations

The Examinations of the Eyes consist of three tests:

*Horizontal Gaze Nystagmus (HGN)*

Clue #1 – Lack of smooth pursuit.

Clue #2 – Distinct and sustained Nystagmus at maximum deviation.

Clue #3 – Angle of Onset
Vertical Gaze Nystagmus

Lack of Convergence

Lack of Convergence is checked by first getting the subject to focus on and track the stimulus as it slowly moves in a circle in front of the subject’s face.

Then, the stimulus is slowly pushed in toward the bridge of the subject’s nose and held for approximately one (1) second.

Under the influence of certain types of drugs, the eyes may not be able to converge.
E. Divided Attention Tests

Several Divided Attention tests used for drug examinations are the same familiar tests used for examining alcohol impaired subjects.

- Modified Romberg Balance
- Walk and Turn
- One Leg Stand
- Finger to Nose
Walk and Turn Test Demonstration

Instructions stage:

One-Leg Stand Test Demonstration

Instructions stage:

Finger to Nose Demonstration

Instructions stage:
F. Examinations of Vital Signs

The Vital Signs consist of three things routinely measured in basic physical examinations.

• Pulse
• Blood Pressure
• Temperature

These measurements require some familiar instruments.

• Stethoscope
• Blood pressure cuff and gauge (sphygmomanometer)
• Thermometer
G. Dark Room Checks of Pupil Size

Dark Room Checks for Pupil Size

The principal activity that takes place during the dark room examinations is the estimation of pupil size under three lighting conditions.

- Room light.
- Near total darkness.
- Direct light.

For safety reasons, whenever possible, another officer should always accompany you and the subject into the dark room.

Room Light

Before turning off the lights, you will estimate the size of the subject’s pupils under room light. You must always first estimate the left pupil, then the right.
Dark Room Checks of Pupil Size

• Room light
• Near-total darkness
• Direct light

You must position the pupillometer alongside the eye to ensure an accurate estimation.

After you have completed the room light estimations, turn off the lights and wait at least 90 seconds to allow your eyes and the subject’s eyes to adapt to the darkness.

Near Total Darkness

The next check will be of pupil size under near total darkness.

You will need the bare minimum amount of light necessary to see the subject’s pupils and the pupillometer.

You can create the necessary light by covering the tip of the penlight with your finger or thumb.

The light is then moved near the subjects left eye just until it is possible to distinguish the colored portion of the eye (Iris).

Hold the pupillometer alongside the eye and locate the circle or semi-circle closest in size to the pupil.
Direct Light

The third and final check will be of the pupil size under direct light.

You will shine the full strength of the penlight directly into the subject’s eye for 15 seconds.

Do this by bringing the light in from the side of the subject’s face.

The penlight should be held close enough to the subject’s eye so that its beam fills the eye socket.

When the light is initially shown into the eye, you will check for the pupil’s reaction to light. Then immediately estimate the pupil size under direct light.

Other Activities

Two other activities are conducted while in the darkroom.

- Examination of the nasal area.
- Examination of the oral cavity.
H. Examination of Muscle Tone

Muscle Tone

Starting with the subject’s left arm, examine the arm muscles.

Firmly grasp the upper arm and slowly move down to determine muscle tone.

The muscles should appear flaccid, normal or rigid to the touch.

Examine the right arm in the same fashion.
I. Examination for Injection Sites

Some injection sites may be relatively easy to notice.

Persons who frequently inject certain drugs develop lengthy scars, commonly referred to as “tracks,” from repeated injections in the same veins.

Injection of certain drugs may result in severe caustic action against the skin and flesh, producing easily observable sores.

Often, a fresh injection site may not be readily observable.

Frequently, a DRE will locate the injection site initially by touch, running the fingers along such commonly used locations as the neck, forearms, wrists, back of hand, etc.

When the DRE locates a possible injection site, a light magnifying lens, commonly known as a “ski light” is used to provide a magnified visual examination.

“Ski” – short for schematic

During this step, the third pulse is taken.
J. Subject Statements

Drug Influence Form Questions:

• What medication or drug have you been using? How much?
• Time of use?
• Where were the drugs used? (location)

Be Sure to Record:

• Date/Time of Arrest
• Time DRE Notified
• Evaluation Start Time
• Time Completed
• DRE signature (Include rank)
• ID #
• Reviewed by:
K. Opinion of Evaluator

By this point in the evaluation, the DRE should have formed an opinion of the category or categories of drugs responsible for any observed impairment.

This opinion is based on the totality of the evaluation.
L. Toxicological Examination

Toxicology Samples

Your State’s implied consent statues will dictate the type of sample you can obtain; urine, blood, breath, or saliva.

Departmental policy, state laboratory guidelines and procedures should be followed in requesting, obtaining and handling the toxicology sample.

There may be times when the toxicology sample has to be obtained prior to Step 12 of the DRE protocol. If this occurs, it is recommended that the DRE document that in the narrative portion of the DRE report.

Specimen Containers

The type of container for collecting the sample will be dictated by the type of sample taken and the laboratory requirements where it will be tested.

Containers should be sterile and have a lid that will seal tightly. Make sure the seal is tight to prevent leaks.
Obtaining a Sample

• Urine – normally the officer must witness the collection of the sample.
• Blood – should be drawn by a qualified technician and witnessed by the officer.
• The sample must include a preservative. This is often pre-packaged in the container intended for this use.

Samples should be refrigerated or frozen as soon as possible to minimize degeneration during storage.

Chain of Custody

Establish a policy dictating the chain of custody, if one does not already exist.

Establish a policy for your Department on:

• The sealing of evidence to include officer identification markings; (i.e., initials, labels, tags and packaging).
• Paperwork for the chain of custody and laboratory analysis of your sample.
• Transportation of the sample to the laboratory.
• Return reporting of the laboratory analysis.

These are issues that must be addressed with the individual agencies to insure proper and standardized procedures. Participants should follow-up with the appropriate representatives from their agencies to coordinate this activity.
M. Video Demonstrations (Optional)
Topics for Study Questions:

1. Give three important reasons for conducting drug evaluation and classification evaluations in a standardized fashion.

2. What are the twelve components of the drug evaluation process?

3. How many times is pulse rate measured during the drug influence evaluation?

4. Are the diameters of a pupillometer’s circles/semi-circles indicated in centimeters, millimeters or micrometers?
5. What formula expresses the approximate statistical relationship between blood alcohol concentration and nystagmus onset angle?

6. Which of the seven categories of drugs ordinarily do not cause nystagmus?

7. How many heel-to-toe steps is the subject instructed to take, in each direction, on the Walk and Turn test?

8. What period of time is the subject required to estimate during the Modified Romberg Balance test?

9. What is systolic pressure?

10. What is the name of the instrument used to measure blood pressure?

11. Name the four validated clues of the One Leg Stand test.

12. Name the eight validated clues of the Walk and Turn test.

13. Suppose you have two hypodermic needles, one is 14 gauge, the other is 20 gauge. Which needle has the smaller inside diameter?
# DRUG INFLUENCE EVALUATION

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<th>When?</th>
<th>What have you been drinking?</th>
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<th>No</th>
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<table>
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<th>No</th>
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| Comments: | | |

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<th>How much?</th>
<th>Time of use?</th>
<th>Where were the drugs used? (Location)</th>
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<th>Evaluation completion time:</th>
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<th>DRE #:</th>
<th>Reviewed/approved by / date:</th>
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<th>CNS Depressant</th>
<th>Hallucinogen</th>
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<th>Cannabis</th>
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**Rev 01/15**
Participant Manual

Drug Recognition Expert Course

Session 5
Eye Examinations
Upon successfully completing this session the student will be able to:

- State the purpose of various eye examinations in the DEC Program drug influence evaluation procedure.
- Describe the administrative procedures for the eye examinations.
- Describe the clues for each eye examination.
- Conduct the eye examinations and note the clues observed.
- Prepare complete, clear and accurate records of the eye examinations.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES

A. Purpose of the Examinations ................................................................ Instructor-Led Presentations
B. Procedures and Clues ........................................................................ Instructor-Led Demonstrations
C. Demonstrations .................................................................................... Student-Led Demonstrations
D. Document Procedures .......................................................................... Students’ Hands-On Practice
E. Practice .................................................................................................. Reading Assignments
A. Purposes of the Eye Examinations

- The principal purpose of all of the eye examinations is to obtain articulable facts indicating the presence or absence of specific categories of drugs.
- Certain drug categories usually cause the eyes to react in specific ways. Other drug categories usually do not cause those reactions.
- The tests of Horizontal and Vertical Gaze Nystagmus provide important indicators of the drug categories that may or may not be present.
- If HGN is observed, it is likely that the subject may have ingested alcohol or another CNS Depressant, an Inhalant, a Dissociative Anesthetic, or a combination of those.
- If Vertical Gaze Nystagmus is observed, the implication may be that the subject ingested a large dose of alcohol for that individual, a Dissociative Anesthetic, such as PCP, or high doses of other Depressants or Inhalants.
By comparing the subject’s blood alcohol concentration with the angle of onset of Horizontal Gaze Nystagmus, it may be possible to determine that alcohol is or is not the sole cause of the observed Nystagmus.

**Clarification:** If the angle of onset is significantly inconsistent with the BAC, the implication may be that the subject has also taken a Dissociative Anesthetic, such as PCP, an inhalant, or some CNS Depressant other than alcohol.

The consistency of the angle of onset and BAC can be compared using the following formula:

\[ \text{BAC} = 50 - \text{Angle of Onset} \]

Explanation: BAC = 100 x blood alcohol (i.e., if blood alcohol is 0.10, BAC = 10)

Example: If onset angle is 35 degrees, then: \( \text{BAC} = 50 - 35 = 15 \)

The corresponding blood alcohol concentration would be approximately 0.15.

Keep in mind that this formula is only a statistical approximation. It is not an exact relationship for all subjects at all times.

The formula can easily be “off” by 0.05 or more, even though the subject has consumed no drug other than alcohol.
The purpose of comparing BAC and angle of onset is to obtain a gross indication of the possible presence of another CNS Depressant, Inhalants, a Dissociative Anesthetic, or Cannabis ("DIDC" drugs).

The check for Lack of Convergence can provide another clue as to the possible presence of Depressants, Dissociative Anesthetics, or Inhalants.

Lack of Convergence is also an indicator of the possible presence of Cannabis.

- The checks of pupil size and reaction to light provide useful indicators of the possible presence of many drug categories.
- CNS Depressants, CNS Stimulants, and Inhalants will normally cause the pupils to react slowly. There will generally be little movement with Narcotic Analgesics.
- CNS Stimulants and Hallucinogens normally will cause the pupils to dilate.
- Cannabis normally causes dilation of the pupils, although this isn’t always observed.

Some specific Inhalants may cause pupil dilation.

Narcotic Analgesics will normally cause observable constriction of the pupils.

During the eye examinations you will also check for rebound dilation.
Review of Eye Examinations

- HGN
- VGN
- LOC
- Pupil Size Estimation
- Reaction to Light

B. Procedures and Clues

Three Clues of Horizontal Gaze Nystagmus

- Lack of smooth pursuit
- Distinct and sustained nystagmus at maximum deviation
- Angle of onset of nystagmus

Horizontal Gaze Nystagmus test consists of three separate checks, administered independently to each eye.
First Clue: Lack of Smooth Pursuit

If the subject is wearing contact lenses, note that fact on the report, but don’t have the subject remove them.

If the subject is wearing eyeglasses, have him or her remove them.

- Position the stimulus approximately 12 – 15 inches in front of the subject’s nose.
- Hold the tip of the stimulus slightly above the level of the subject’s eye. Point out that this procedure ensures that the subject’s eyes will be wide open and easy to observe.
- Instruct the subject to hold the head still and follow the stimulus with their eyes.

The first check is for “lack of smooth pursuit.”

- Move the stimulus smoothly, all the way to the subject’s left side and back all the way to the right side.

Make at least two complete passes of the stimulus: to the left side, to the right side, back to the left side, and finally back to the right side.
First Clue:
Lack of Smooth Pursuit

- When doing this, don’t pause at the center of the subject’s face; move all the way to the left, then all the way to the right, then again all the way to the left and back all the way to the right, in a smooth, continuous motion.

- While the eye is moving, examine it for evidence of a lack of smooth pursuit.

- Use the following analogy:

A smoothly pursing eye will move without friction, much the way that a windshield wiper glides across the windshield when it is raining steadily. An eye showing lack of smooth pursuit will move in a fashion similar to a wiper across a dry windshield.

- Also, check to be sure that both eyes are tracking in the same way: if one eye is moving smoothly but the other moves hesitantly or not at all, an illness or injury may be present.
Second Clue: Distinct and Sustained Nystagmus atMaximum Deviation

The second check is for “distinct and sustained nystagmus at maximum deviation.”

- Again position the stimulus as before.
- Move the stimulus all the way to the subject’s left side and hold it there so that the subject’s eye is turned as far to the side as possible.
- Hold the eye at that position for a minimum of 4 seconds, to check carefully for jerking that may be present, and that is distinct.

When you have completed this check for the left eye, repeat the process for the right eye. Then, do it once again for the left eye, and again for the right, to verify that distinct and sustained nystagmus is or is not present.

With this clue, the examiner looks for a very distinct, unmistakable jerking.
Second Clue: Distinct and Sustained Nystagmus at Maximum Deviation

Third Clue: Angle of Onset of Nystagmus

A slight or barely visible tremor is not sufficient to consider this clue present. A definite, sustained jerking must be seen.

Third Clue: Angle of Onset Nystagmus

The final check is for the “angle of onset of nystagmus.”

- Position the stimulus as before.
- Slowly move the stimulus to the subject’s left side, carefully watching the eye for the first sign of jerking.

**Stimulus should be moved at a speed that requires approximately four seconds to travel from center to approximately 45 degrees.**

- When you think that you see the eye jerk, stop moving the stimulus and hold it still.
- Verify that the eye is, in fact, jerking.
- Once you have established that you have located the point of onset, estimate the angle.
- Then, repeat the process for the right eye.
- Then, again check onset for the left eye, and again for the right.
Participants’ Initial Practice of Angle Estimation

- 30 degrees
- 35 degrees
- 40 degrees

Participants will check their accuracy using a template (if available).
Vertical Gaze Nystagmus

- Position the stimulus horizontally, approximately 12 – 15 inches in front of the subject’s nose.
- Instruct the subject to hold the head still and follow the stimulus with the eyes only.
- Raise the stimulus until the subject’s eyes are elevated as far as possible.
- Watch closely for evidence of jerking.

Participants’ Initial Practice of the Vertical Gaze Nystagmus Test

1. Position the stimulus horizontally, approximately 12 – 15 inches in front of the subject’s nose.
2. Instruct the subject to hold the head still and follow the stimulus with the eyes only.
3. Raise the stimulus until the subject’s eyes are elevated as far as possible.
4. Watch closely for evidence of jerking.

1. Practice the test with different subjects to ensure consistency.
2. Record the results for future reference.
3. Review the test procedures with colleagues for feedback.
4. Consider adding additional stimuli to test a wider range of responses.

1. Practice the test in a quiet environment to eliminate distractions.
2. Ensure the lighting is consistent to avoid affecting the subject’s vision.
3. Communicate clearly with the subject to avoid confusion.
4. Document the test results accurately to support future analyses.

1. Review the test results with the subject to discuss any concerns or observations.
2. Provide feedback to the subject on their performance.
3. Offer tips or suggestions for improvement.
4. Encourage the subject to practice the test at home for better familiarity.

1. Practice the test in various settings to assess the subject’s responses under different circumstances.
2. Adjust the stimulus based on the subject’s height and vision to ensure an accurate test.
3. Use a variety of stimuli to test different aspects of the subject’s visual system.
4. Discuss the test results with the subject to explain the significance of the findings.
Lack of Convergence

The test for Lack of Convergence (LOC) is also very simple. But it should be noted that this test may not be as reliable as the other eye tests due to the fact that some people may have an inability to cross their eyes normally.

- Lack of Convergence means an inability to cross the eyes.
- Prior to conducting the check for Lack of Convergence the DRE should determine if the subject to be tested routinely wears eyeglasses during reading and near visual tasks and if so, are they readily available for the test.
- If the subject wears glasses during reading and near visual tasks and they are readily available, ensure that the eyeglasses are worn for the check for Lack of Convergence.

In testing for Lack of Convergence (LOC), the role of clear vision and focusing can have significant effect on the convergence of the eyes. In the clinical setting, the LOC check is routinely conducted with the eyeglasses on if normally worn by the subject during reading and near visual tasks. If the subject’s eyeglasses are not readily available, the DRE should still conduct the test.
This revision to the LOC exam was approved by the IACP Technical Advisory Panel (TAP), November 2008.

- Position the stimulus approximately 12-15 inches in front of the subject’s face.
- Instruct the person to hold their head still and follow the stimulus with the eyes only.
- Keep the object 12-15 inches away from the person’s nose, and start to move the stimulus slowly in a circle, approximately the same size as the subject’s face.
- Once you have verified that the subject is tracking the stimulus, stop moving in a circular manner with the stimulus above eye level, move it slowly and steadily toward the bridge of the nose.
- Hold the stimulus near the bridge of the nose for approximately one (1) second. The stimulus should not come any closer than approximately two (2) inches from the bridge of the nose.
- Carefully observe the subject’s eyes to determine whether both eyes converge.

Participants’ Initial Practice of the Check for the Lack of Convergence
Estimating Pupil Size

The pupils of our eyes continually adjust in size to accommodate different lighting conditions.

The pupillometer is held alongside the subject’s eye, moved up and down until the circle or semi-circle closest in size to the pupil is located.

We use a device called a pupillometer to estimate the size of the subject’s pupils.

Pupil size estimations are recorded as the numeric value that corresponds to the diameter of the circle or semi-circle that is closest in size to the subject’s pupil in each lighting condition.

This should not be confused with pupillary unrest, the continuous, irregular change in the size of the pupils that may be observed under room or steady light conditions or with pupillary light reflex which is the pupil’s normal reaction to the changes in light.
The Three Lighting Conditions

Pupil sizes are estimated under three different lighting conditions:

- Room Light
- Near Total Darkness
- Direct Light
Estimation of Pupil Size under Room Light

- The pupils are examined in room light prior to darkening the room.

Participant’s Initial Practice of Pupil Size Estimation—Room Light

Participant’s Initial Practice of Pupil Size Estimation—Dark Room

- After you have completed the pupil size estimations in room light, you must darken the room, wait approximately 90 seconds (for the officers eyes to adjust to the light), and then proceed with the dark room exam.
Estimation of Pupil Size under Near Total Darkness (NTD)

- For the check under near total darkness completely cover the tip of the penlight with your finger or thumb, so that only a reddish glow and no white light emerges.
- Bring the glowing tip up toward the subject’s left eye until you can just distinguish the pupil from the colored portion of the eye (iris).
- Continue to hold the glowing red tip in that position and bring the pupillometer up alongside the subject’s left eye and locate the circle or semi-circle that is closest in size to the pupil.
- Repeat this procedure for the subject’s right eye.
**Estimation of Pupil Size under Near Total Darkness Using Ultra Violet Light**

Independent research has demonstrated that Ultraviolet (UV) lights are effective tools for assessing pupil size in near total darkness, giving essentially identical results to the standard evaluation regardless of subject eye color. Evaluators found the UV light easier to use, especially when assessing subjects with dark eyes. If this test is used, it should be used after pupil size estimations have been attempted with a finger-covered pen light.

Hold the UV light along the subject’s face at any location from the side of the eye to just below the eye. If the light is held along the cheek, it can be used to illuminate the pupillometer.

Start with the light about parallel to the plane of the subject’s face and slowly increase the angle outward until the light just passes through the cornea, the clear window at the front of the eye.

When using a UV light to assess pupil size, it is important to remember to never shine the light directly into the subject’s eye. In low dosages and for short exposure times, the UV light is not harmful to the subject’s eye. However, the light does emit visible wavelengths in the blue-violet region of the spectrum, otherwise the evaluator would not be able to see that the light is on. Consequently, shining the light directly into the subject’s eye can unintentionally cause the pupil to constrict.
1. Position the light near the subject’s face along the cheek just below the eye starting with the subject’s left eye. Position the tip of the light approximately 6 - 8 inches from the eye (Refer to photo). However, the distance can vary depending on the brightness of the light being used.

2. Start with the light about parallel to the subject’s face and slowly increase the angle outward until the light passes through the cornea (the clear window at the front of the eye) until the yellow-green glow of the crystalline lens is evident.

3. Avoid shining the UV light directly into the subject’s eye. In low dosages and for short exposure times, the UV light is not harmful to the subject’s eye, nor to the evaluator’s eyes.

4. Using a DRE pupillometer, estimate the size of the glowing pupil in NTD.

5. Conduct the same procedure for the right eye.

Using a UV light to estimate pupil size in the NTD lighting condition is an easy, safe, and effective evaluation, especially when assessing subjects with dark eyes. Used properly, there is no potential harm to the subject or the DRE.

Use of the UV light for the NTD pupil estimation is not mandatory and does not replace the current covered penlight procedure. If a DRE uses the UV light for the NTD estimation, it should be documented in the narrative report.
Estimation of Pupil Size under Direct Light

- Bring the penlight from the side of the subject’s face and shine it directly into their left eye.
- Position the penlight so that it illuminates and approximately fills the subject’s eye socket.
- Hold the penlight in that position for 15 seconds, and bring the pupillometer up alongside the left eye.
- Find the circle or semi-circle that is closest in size to the pupil.
- Repeat this procedure for the subject’s right eye.
Pupillary Unrest

Another eye sign that may be observed by the DRE is Pupillary Unrest.

Pupillary Unrest is defined as the continuous, irregular change in the size of the pupils that may be observed under room or steady light conditions.

Pupillary Unrest is not abnormal or a sign of impairment. If observed, it is most likely not related to drug or medical impairment. Its presence can be due to various reasons, e.g., light source fluctuations in focusing, and attention issues of the subject being tested. Pupillary unrest is seen as natural pupillary movements that are active in the presence of light, focusing, and maintaining alertness in normal people.

These movements or oscillations in pupil size changes are typically observed as small amounts of constriction, then dilation, then constriction. These ranges are typically very small in size. They are not rebound dilation.

Pupillary Unrest or sight instability in pupil size are generally related to:

1. Changes in light intensity levels, e.g., movement of the subject’s head, penlight movements, and changes in brightness levels.
2. Changes in focusing (accommodation), e.g., subject changing fixation and not looking at a steady fixed target or stimulus.
3. Other forms of sensory stimulation, e.g., loud noises, irritating questions being asked during the testing, etc.

The unique indicators of Pupillary Unrest are the unevenness and fluctuations in the rate and size of the pupils under lighted conditions and its disappearance in darkness.

There is no current scientific research to support that pupillary unrest is directly related to drug influence at this time.
Rebound Dilation

Rebound dilation is defined as a period of pupillary constriction followed by a period of pupillary dilation where the pupil steadily increases in size and does not return to its original constricted size.

Example: The pupil is estimated at 8.5mm in near total darkness. Once the penlight is shined into the pupil it constricts to 4.0 mm then steadily dilates to 6.0 mm and remains that diameter while the direct light is shined into the eye.

Rebound dilation has been reported with persons impaired by drugs that cause pupillary dilation. Cannabis is most common.
**Pupil Ranges**

For most people, even under very bright light the pupils will not constrict much below a diameter of 2.0 millimeters (mm) or dilate to a diameter of not more than 8.5 mm in near total dark conditions.

Consequently, the use of three distinct pupil size ranges for each of the different testing conditions may be considered more useful in the evaluation to determine impairment vs. non-impairment.

**Pupil Size Technical Terms**

Two key technical terms regarding pupil sizes are: Miosis – abnormally small pupil, i.e., constricted, and Mydriasis – an abnormally large pupil, i.e., dilated.
Non-Impaired Pupil Sizes

With pupil size and range:

Room light
- Approximately 4.0 mm with pupil sizes ranging from 2.5 to 5.0 mm

Near total darkness
- Approximately 6.5 mm with pupil sizes ranging from 5.0 to 8.5 mm

Direct light
- Approximately 3.0 mm with pupil sizes ranging from 2.0 to 4.5 mm

Non-Impaired Pupil Sizes

Room Light
- For a non-impaired person, the average pupil size and range for room light is approximately 4.0 mm, with pupil sizes ranging from 2.5 to 5.0 mm.

Near Total Darkness
- For a non-impaired person, the average pupil size and range for near total darkness is approximately 6.5 mm with pupil sizes ranging from 5.0 to 8.5 mm.

Direct Light
- For a non-impaired person, the average pupil size and range for direct light is approximately 3.0 mm with pupil sizes ranging from 2.0 to 4.5 mm.
Reaction to Light

Assessment of how quickly the pupil constricts to its smallest size during the check of pupil size under direct light when the uncovered light is brought from the side of the subject’s face and the light beam is moved directly into his or her left eye.

- As you bring the beam of light directly into the subject’s eye, note how the pupil reacts.
- Under ordinary conditions, the pupil should react very quickly, and constrict noticeably when the light beam strikes the eye.
- Under the influence of certain categories of drugs, the pupil’s reaction may be slow, or there may be no visible reaction at all.

For DRE purposes, we consider the pupil’s reaction to be slow if it takes more than one second to reach its smallest size.

- Hold the direct light on the subject’s eye for 15 seconds to assess pupil reaction.
- Also check for Rebound Dilation during this 15 second period.
- Caution should be used by the officer so as not to move the light beam or allow the bulb to change in light intensity.
- When you have completed this process for the left eye, repeat it for the right eye.

Participants’ initial practice in assessing the pupil’s reaction to light.
C. Demonstrations

- Check for Lack of Smooth Pursuit
- Check for Distinct and Sustained Nystagmus at Maximum Deviation
- Check for an Onset of Nystagmus prior to 45 degrees

Estimation of Angle of Onset

Demonstration of Vertical Gaze Nystagmus and Lack of Convergence
Demonstration of Pupil Size and Reaction to Light Checks

- Room Light
- Dark room checks of pupil size
- Near total darkness
- Direct light
- Reaction to light
D. Documentation Procedures

A brief examination of the eyes is made during the Preliminary Examination.

- Check for equal pupil size.
- Check for resting nystagmus.
- Assessment of tracking ability.
- Initial assessment of the angle of nystagmus
- *Horizontal Gaze Nystagmus*
- *Vertical Gaze Nystagmus*
- *Lack of Convergence*
- The dark room eye examinations are documented in a subsequent section of the form.
Sample Eye Examination

A brief examination of the eyes is made during the Preliminary Examination.

- Check for equal pupil size.
- Check for resting nystagmus.
- Assessment of tracking ability.
- Initial assessment of Nystagmus angle of onset.

**Horizontal Gaze Nystagmus**

**Vertical Gaze Nystagmus**

"Yes" implies that Vertical Gaze Nystagmus was present, "No" implies that it was not present.

**Lack of Convergence**

The dark room eye examinations are documented in a subsequent section of the form.

**Preliminary Eye Exams**

**Eye Exams**
Pupil Size Estimations

- Room Light
- Near Total Darkness
- Direct Light

Reporting out of Pupil Size Estimations
Tabulations:

Room Light

Repeat this process for each of the other two lighting conditions.

Near Total Darkness Tabulation:

Direct Light Tabulation:
E. Practice

Preliminary Eye Exams

- Check for equal pupil size.
- Check for resting nystagmus.
- Assessment of tracking ability.
- Initial estimation of nystagmus angle of onset.

Eye Exams

- Horizontal Gaze Nystagmus.
- Vertical Gaze Nystagmus.
- Lack of Convergence.
QUESTIONS?
# Pupil Size Chart

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<th>Pupil Size</th>
<th>Room Light</th>
<th>Near Total Darkness</th>
<th>Direct Light</th>
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PARTICIPANT PROFICIENCY EXAMINATION
STANDARDIZED FIELD SOBRIETY TEST BATTERY

Name ___________________________ Date ______ / ______ / ______
Agency ______________________________________________________

I. HORIZONTAL GAZE NYSTAGMUS

1. ___ Have subject remove glasses if worn.
2. ___ Stimulus held in proper position (approximately 12”-15” from nose, just slightly above eye level.
3. ___ Check for equal pupil size and resting nystagmus.
4. ___ Check for equal tracking.
5. ___ Smooth movement from center of nose to maximum deviation in approximately 2 seconds and then back across subject’s face to maximum deviation in right eye, then back to center. Check left eye, then right eye. (Repeat)
6. ___ Eye held at maximum deviation for a minimum of 4 seconds (no white showing). Check left eye, then right eye. (Repeat)
7. ___ Eye moved slowly (approximately 4 seconds) from center to 45 angle. Check left eye, then right eye. (Repeat)
8. ___ Check for Vertical Gaze Nystagmus. (Repeat)

II. WALK AND TURN

1. ___ Instructions given from a safe position.
2. ___ Tells subject to place feet on a line in heel-to-toe manner (left foot behind right foot) with arms at sides and gives demonstration.
3. ___ Tells subject not to begin test until instructed to do so and asks if subject understands.
4. ___ Tells subject to take nine heel-to-toe steps on the line and demonstrates.
5. ___ Explains and demonstrates turning procedure.
6. ___ Tells subject to return on the line taking nine heel-to-toe steps.
7. ___ Tells subject to count steps out loud.
8. ___ Tells subject to look at feet while walking.
9. ___ Tells subject not to raise arms from sides.
10. ___ Tells subject not to stop once they begin.
11. ___ Asks subject if all instructions are understood.
III. ONE LEG STAND

1. ___ Instructions given from a safe position.
2. ___ Tells subject to stand straight, place feet together, and hold arms at sides.
3. ___ Tells subject not to begin test until instructed to do so and asked if subject understands.
4. ___ Tells subject to raise one leg, either leg, approximately 6” from the ground, keeping raised foot parallel to the ground, and gives demonstration.
5. ___ Tells subject to keep both legs straight and to look at elevated foot.
6. ___ Tells subject to count out loud in the following manner: one thousand one, one thousand two, one thousand three, and so on until told to stop, and gives demonstration.
7. ___ Checks actual time subject holds leg up. (Time for 30 seconds.)

Instructor: ________________________________________________________________

Note: In order to pass the proficiency examination, the student must explain and proficiently complete each of the steps listed.
Participant Guide

Drug Recognition Expert Course

Session 6
Physiology and Drugs: An Overview
A. Physiology and Drugs: An Overview

Upon successfully completing this session the participant will be able to:

- Explain in layman’s terms the general concept of human physiology.
- Explain in layman’s terms the purpose and functions of major systems in the body (nervous system, circulatory system, respiratory system, etc.)
- Explain in layman’s terms how drugs work in the body.
- Explain in general terms how the drug evaluation is used to detect signs and symptoms indicative of drug impairment.
- Correctly answer the “topics for study” questions at the end of this session.

CONTENT SEGMENTS..................................................................................... LEARNING ACTIVITIES
A. Physiology and Drugs: An Overview ............................................. Instructor-Led Presentations
B. Body Systems ............................................................................................ Reading Assignments
C. The Concept of Homeostasis
D. A Simplified Concept of the Nervous System
E. How Drugs Work
F. Medical Conditions Which Sometimes Mimic Drug Impairment
A. Physiology and Drugs: An Overview

Before we can understand how drugs work, we must have a basic understanding of how the body works.

We will review general concepts of how the body functions in a “normal” or “standard” human.
“Average” or “Normal” Within the DEC Program

- “Average” is a quantity that represents the “middle” or “typical” value that the majority of healthy, non-impaired people would exhibit or have in a specific test that is measured numerically.

- “Normal” describes both a range of values or results that are “close to” average, but can be above or below the “average” value for the majority of healthy non-impaired people. “Normal” can also be used to describe unremarkable conditions on tests that are not measured numerically such as muscle tone, etc.

Within the DEC Program, normal” means the same thing as “healthy” or “non-impaired” or within the “DRE average ranges.”

For example, the “Average”, or typical value, for pupil size in near total darkness is 6.5 mm. This means that when ALL the sizes were measured using the DRE test protocol, in a large number of pupils in healthy, non-impaired adults, the average pupil size for those was approximately 6.5 mm while the average range, or for normal pupil size was 5.0-8.5 mm.
Average and Normal

• Terms are overused by DREs
• In context of the DEC, it can mean “healthy,” “non-impaired,” or “as expected.”
• “Normal” is commonly used to refer to a result within the DEC average ranges, such as pupil sizes, pulse rate, and BP.

Point out that when using the term “normal” or “normals,” the DRE should understand what these terms refer to, as well as the differences in use between law enforcement, the DEC Program and medical practitioner use of the same terms. Although the term “normal range” was historically used in the DEC Program, we now use the term “average range” to provide a better description of what is observed.

To avoid the defense argument of “what is ‘normal’ for my client?” DRE’s need to be prepared to explain the meaning and use of the term as it relates to the DEC Program.

A Healthcare Practitioner can determine what is “Normal” for a person based on their training, experience and a combination of additional data. They can also determine this through ordered tests and their results, what has been usual for the person over time, if the specific result is getting better, worse, or staying stable, if the disease process being evaluated is getting better, worse, or staying stable, how abnormal the test result is, and if it may represent an error in the test itself. A DRE does not have any of the above healthcare information available during the time the DRE evaluation is performed.
Another way to look at Average versus Normal:

- The *average* length (number) of a pregnancy is 40 weeks.
- The *average* length (range) of a pregnancy is 38 to 42 weeks.
- If a woman is *two weeks* past her “due date,” she is still not necessarily *late*!
- Being past the due date is not itself a problem, but:
  - There is a greater risk of problems.
  - There may be other observable signs and reported symptoms of potential problems.

What DREs Need to Know About “Average”—General Rules:

1. The closer the test finding is to the Average value for the majority of normal people, the more likely the person is not exhibiting impairment in that function.
2. The farther away from the Average and the closer to the edge of the “Average Range” for the majority of people, the more possible the person is going to be exhibiting impairment in that function.
3. The farther outside the Average Range for the majority of normal people in the test, the greater the likelihood that the person is exhibiting impairment in that function.
Opinion versus Diagnosis

- An Opinion is a judgment based on special knowledge and experience.
- A Diagnosis is a legal and medical conclusion reached by someone with medical experience and expertise.
- DREs do not make a diagnosis.
- The DRE’s goal is to determine the presence of impairment and the probable cause(s) of the observed impairment.

1. An Opinion is a judgment based on special knowledge and experience.
2. A Diagnosis is a legal and medical conclusion reached by someone with medical experience and expertise.
3. DREs do not make a diagnosis.
4. The DRE’s goal is to determine the presence of impairment and the probable cause(s) of the observed impairment.

As a DRE, when you complete an evaluation and decide whether the person is impaired, whether the impairment is a result of a medical problem or drugs, and if drugs, what drug category or categories is/are causing the impairment, you are rendering an OPINION. You are NOT making a diagnosis.

A diagnosis is a medical conclusion reached by someone with medical experience and expertise.
**Opinion vs. Diagnosis**

<table>
<thead>
<tr>
<th>Reason for Assessment</th>
<th>DRE</th>
<th>Doctor</th>
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<tbody>
<tr>
<td><strong>LEGAL</strong>: Non voluntary arrest: Impaired driving and need to determine possible reason for impairment.</td>
<td>-LEGAL: Non voluntary Arrest</td>
<td>-MEDICAL: Voluntary visit with symptoms or complaints</td>
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<td><strong>LEGAL</strong>: Unpredictable and may be limited or refused since it involves evidence and rights.</td>
<td>-Unpredictable and may be limited or refused since it involves evidence and rights</td>
<td></td>
</tr>
<tr>
<td><strong>MEDICAL</strong>: Doctor or medical personnel generally get full compliance, a full history, order tests in order to receive the proper diagnosis and treatment. DREs do not provide treatment in regards to the evaluation.</td>
<td>-Full with history and tests to lead to proper diagnosis and treatment</td>
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<tr>
<th>Time</th>
<th>DRE</th>
<th>Doctor</th>
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<tbody>
<tr>
<td><strong>LEGAL</strong>: A single, one-time contact with limited time.</td>
<td>-Single One-Time contact and limited time</td>
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<tr>
<td><strong>MEDICAL</strong>: May involve multiple visits and time.</td>
<td>-Multiple visits and time</td>
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<th>Outcome Goal</th>
<th>DRE</th>
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</tr>
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<td><strong>MEDICAL</strong>: Differential diagnosis leading to multiple tests, leading to the treatment goal(s).</td>
<td>-Differential Diagnosis with multiple tests leading to treatment goal</td>
<td></td>
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**REASON FOR ASSESSMENT:**

LEGAL: Non voluntary arrest: Impaired driving and need to determine possible reason for Impairment

Medical Care is offered and decided in beginning.

MEDICAL: Voluntary visit with symptoms or complaints

**COMPLIANCE:**

LEGAL: Unpredictable compliance, and may be limited or the subject may simply refuse, since it involves rights and evidence.

MEDICAL: Doctor or medical personnel generally get full compliance, a full history, order tests in order to receive the proper diagnosis and treatment. DREs do not provide treatment in regards to the evaluation.

**TIME:**

LEGAL: A single, one-time contact with limited time.

MEDICAL: May involve multiple visits and time.

**OUTCOME GOAL:**

LEGAL: Presence of impairment, and inconsistent with BAC, the opinion is based on probabilities as to the cause of impairment. Treatment is not the outcome goal.

MEDICAL: Differential diagnosis leading to multiple tests, leading to the treatment goal(s).
Primary focus will be on the systems or component parts of those systems that are examined during the drug influence evaluation. They include:

- Central Nervous System
- Eyes
- Blood Pressure and Pulse
- Balance and Coordination
- Body Temperature
B. Body Systems

Physiology is the branch of biology that deals with the functions and activities of life or living matter and the physical and chemical phenomena involved.

For the purposes of this course, physiology is the study of the functions of living organisms and their parts.

*Source: Merriam-Webster’s Medical Dictionary (2008).*
A convenient way of discussing human physiology is to list the ten major systems of the body. The phrase “MURDERS INC” helps us remember the names of the ten systems. Each letter stands for the name of one system. CHANGES in these systems act as the basis for determining IMPAIRMENT.
Muscular System

*M stands for the MUSCULAR SYSTEM*

The body has three different kinds of muscles.

- The heart or cardiac muscle.
- Smooth muscles, which control the body’s involuntary operations.
- Striated muscles, which carry out our voluntary movements.

Examples: Smooth muscles control breathing, the operation of the pyloric valve (a muscle located at the base of the stomach), dilation and constriction of pupils, and all other things that we do not consciously control.

All three types of muscles are examined at various stages of the drug influence evaluation.

Urinary System

*U is for the URINARY SYSTEM.*

The system consists of two kidneys, the bladder, ureters connecting the kidneys to the bladder, and the urethra, which transports the urine out of the body.

Kidneys filter waste or harmful products, such as drugs and their metabolites, from the blood, and dump these waste products into the bladder.

Respiratory System

The first R in “MURDERS INC” stands for the RESPIRATORY SYSTEM.

The major parts of the Respiratory System are the lungs and the diaphragm.

The diaphragm is a smooth muscle that draws the air into the lungs and forces it out.

Lungs take in oxygen and transfer it to the blood, and remove carbon dioxide and some other waste products from the blood, and expel them into the outside air.
Digestive System

D stands for the DIGESTIVE SYSTEM.

Major components of this system are the tongue, teeth, esophagus, stomach, intestines, liver, and pancreas.

The Digestive System breaks down large particles of food, until they are of a size and chemical composition that can be absorbed in the blood.

Endocrine System

E is for the ENDOCRINE SYSTEM.

The Endocrine System is made up of a number of different glands that secrete hormones.

Hormones are complex chemicals that travel through the blood stream and that control or regulate certain body processes.

Some drugs can mimic the effects of certain hormones, or can react with the hormones in ways that alter the hormones’ effects.

Reproductive System

The second R in “MURDERS INC” stands for the REPRODUCTIVE SYSTEM.

The functions of the reproductive system fall into two categories:

• self-producing (cytogenic), and
• hormone producing (endocrinic).

We are primarily concerned with hormone production since the hormones produced by the reproductive system aid the nervous system in its regulatory role.

Skeletal System

S is for the SKELETAL SYSTEM.

Consists of bones, cartilage and ligaments. The Skeletal System provides support to the body, permits movement, and forms blood cells.
Integumentary System

The I in “INC” stands for the INTEGUMENTARY SYSTEM.

Consists of the skin, hair, fingernails and toe nails, and accessory structures.

The chief functions of the Integumentary System include protection of the body, control of the body temperature, excretion of wastes (i.e. through sweat) and sensory perception.

Nervous System

N is for the NERVOUS SYSTEM.

This system consists of the brain, the brain stem, the spinal cord and the nerves.

Nerves keep the brain informed of changes in the body’s external and internal environments.

Nerves also carry messages from the brain to the body’s muscles, tissues and organs.

The nervous system controls, coordinates and integrates all physiological processes, so that normal body functions can be maintained.

Circulatory System

C is for the CIRCULATORY SYSTEM.

For our purposes, the most important parts of the Circulatory System are the heart, the blood vessels (e.g., arteries, veins, capillaries, etc.) and the blood.

Blood is the body’s primary transport mechanism: it carries food, water, oxygen, hormones, antibodies, etc. to the body’s tissues and organs.

Blood is also primarily responsible for carrying heat throughout the body.

Blood is the main transport mechanism for bringing drugs to the brain.

The heart, of course, pumps the blood and causes it to circulate throughout the body.
Homeostasis is indicated in the above slide. It represents average (normative) values for the clinical indicators used by the DRE to assist in making an opinion of impairment, and medical drug related causes.
In the above slide, the indicators listed are common with persons impaired by a drug category or categories, in this case CNS Stimulants, or perhaps are experiencing a medical issue. Whatever the case, they usually will exhibit indicators of impairment.

Individuals that are impaired exhibit numerous indicators of impairment. In other words, they generally do not exhibit the average values (normative) for the related indicators.
Interrelated Body Systems
C. The Concept of Homeostasis

Homeostasis is “The dynamic balance, or steady state, involving levels of salts, water, sugars and other materials in the body’s fluids.”

The human body is exposed to a constantly changing external environment, which influences the internal environment.

Changes are neutralized by the internal environment – the blood. Oxygen, foods, water and other substances are constantly leaving bodily fluids to enter cells, while carbon dioxide and other wastes are leaving the cells to enter these fluids.

Yet, the chemical composition of these fluids remains within very narrow limits.

This phenomenon is called homeostasis.

This involves message sending and actions triggered by the balance within the autonomic nervous system (sympathetic and parasympathetic), hormones and neurotransmitters.

Drugs interfere with the homeostatic mechanisms and produce signs and symptoms that can be recognized by a trained DRE.
Someone who is ill or injured...

- For non-substance-abusing people who are ill (sick), they have signs and symptoms of being “out of balance”

- They want to get back “in balance” to feel better (“like usual”), so doctors give them drugs to put them in balance.

For non-substance-abusing people who are ill, they have signs and symptoms of being “out of balance.” In other words, their homeostasis is “out of balance.”

They want to get their homeostasis back “in balance” to feel better (“like usual”), so doctors give them drugs to help put them in balance.
Drug abusers put themselves “out of balance” in their Nervous System to get “high.” They typically show signs of impairment in the drug influence evaluation. In effect, they want to change their consciousness. This is why we NEED TO KNOW physiology and drug effects.
Each neuron, or “wire segment” has three main parts:

- the cell body; the axon; the dendrite

The axon is the part of the neuron that sends out the neurotransmitter, or chemical messenger.
The dendrite is the part that receives the neurotransmitter.
The gap between two neurons is called a synapse, or synaptic gap.
D. The Nervous System

Clarification: Nerves are often pictured as telephone or telegraph wires.

The nerves that carry messages to and from the brain often are pictured as “wires” that carry electrical signals.

A more accurate, but still simplified concept would envision a nerve as a series of broken wire segments, with the segments separated by short spaces, or gaps.
We can imagine messages running along the “wire segments” in much the same manner that electrical impulses run along telephone wires.

When the message reaches the end of the “wire segment,” it triggers the release of chemicals that flow across the gap, and contact the next “wire segment.”

When the chemical contacts the next wire segment, it generates an electrical impulse which runs along the wire until it reaches the next gap.

At that gap, the message again triggers the release of chemicals that flow across to the next “wire segment,” and the process continues.
Classification of Nerves

Some nerves carry messages away from the brain, to the body’s muscles and organs. These are called motor, or efferent nerves.

The brain uses motor nerves to send commands to the heart to beat, the lungs to breathe, the muscles to contract or expand, and so forth.

Other nerves carry messages to the brain, i.e. from the eyes, ears and other senses, from the muscles, etc. These are called Sensory, or Afferent nerves.

The brain decodes the messages that come along the sensory nerves to monitor the condition of the body and of the outside world.

A fundamental notion: if something interferes with the messages the brain sends along the motor nerves, the brain’s control over the heart, the lungs, the muscles and other organs will be distorted.

Another fundamental notion: if something interferes with the messages the brain receives from the sensory nerves, the brain’s perception of the outside world and of the body’s status will be distorted.
There are two sub-systems of motor nerves:

- The voluntary nerves send messages to the striated muscles that we consciously control.
- The autonomic nerves send messages to the muscles and organs that we do not consciously control, i.e. smooth muscle and cardiac muscle.

- The Autonomic sub-system is divided into two groups.
• The Sympathetic nerves command the body to react in response to fear, stress, excitement, etc.

**CLARIFICATION: Sympathetic nerves control the body’s “fight or flight” responses.**

**EXAMPLES:** Sympathetic nerves carry the messages that cause: blood pressure to elevate, pupils to dilate, sweat glands to activate, hair to stand on end, heartbeat to increase and strengthen, blood vessels of the skin to constrict, the walls of the hollow viscera to relax (inhibiting digestion).

• Parasympathetic nerves carry messages that produce relaxed and tranquil activities.

**EXAMPLES:** Parasympathetic nerves carry messages that cause: pupils to constrict, heartbeat to slow, peripheral blood vessels to dilate, blood pressure to decrease.

Certain neurotransmitters (i.e. chemical messengers) aid in the transmission of messages along sympathetic and parasympathetic nerves.

Drugs that mimic neurotransmitters associated with parasympathetic nerves are called parasympathomimetic drugs.

Some drugs mimic the action of these neurotransmitters: when taken into the body, these drugs artificially cause the transmission of messages along sympathetic or parasympathetic nerves.

Drugs that mimic the neurotransmitter associated with sympathetic nerves are called sympathomimetic drugs.

Sympathomimetic drugs artificially cause the transmission of messages that produce elevated blood pressure, dilated pupils, etc.
Examples: CNS Stimulants, Hallucinogens, and to some extent Dissociative Anesthetics and Cannabis.
The Sympathetic subsystem of the autonomic nervous system controls the stimulating type effects of the body. It can be referred to as “Adrenergic.”

Adrenergic means; relating to nerve cells or fibers of the autonomic nervous system that use norepinephrine as their neurotransmitter.

We can relate this to “adrenaline” which tends to speed up the body’s processes.
What are Adrenergic Drugs Used to Treat?

Bronchodilators

Bronchodilators act directly to improve breathing in patients with respiratory diseases like asthma, chronic obstructive pulmonary and bronchitis.

Epinephrine, Ephedrine, and Albuterol are common examples.

Vasopressors

Vasopressors can act adrenergic receptors and on dopamine receptors. They can act on more than one type of receptor at the same time.

Phenylephrine, Ephedrine, Pseudoephedrine (Sudafed), and Dopamine are examples.

The vasopressors stimulate smooth muscle contraction of the blood vessels and leads to vasoconstriction (Rise in blood pressure)

The increased blood pressure can be used to treat patients with shock.

Drugs in this class may also be used when swelling of the blood vessels in the mucous membranes of the nose blocks up the nasal passage and causes discharge.

Cardiac stimulators

Adrenergic drugs (Epinephrine) are also used to stimulate and restore the heartbeat.
Parasympathomimetic drugs artificially cause the transmission of messages that produce lowered blood pressure, drowsiness, etc.

Cholinergic means; an agent that resembles acetylcholine or simulates its action.

Acetylcholine is released at the ends of nerve fibers in the somatic and parasympathetic nervous systems and is involved in the transmission of nerve impulses in the body.
An **ANTicholinergic** drug can look like a sympathomimetic

Uses include:

- Preoperative Medication - They inhibit salivary and bronchial secretions. They block the vagal slowing of the heart that can occur with general anesthesia.
- Gastrointestinal Disorders - They decrease gastrointestinal motility and can be used to treat ulcers, diarrhea, and hypermotility.
- Ophthalmologic Examinations - Topical use can cause mydriasis which causes a full visualization of the retina.
- Cycloplegia relaxes the lens so that proper prescriptions for glasses can be determined.
- Parkinson Disease - They reduce the tremors and rigidity associated with Parkinson and drug-induced Parkinson disease.
- Motion Sickness - These drugs are used to treat or prevent motion sickness because of their central nervous system depressant action.
A Cholinergic drug can look like a parasympathomimetic.

The vagus nerve is responsible for heart rate, gastrointestinal peristalsis, etc...

The vagus nerve is the parasympathetic innervation of the heart: slows it down... reduces blood pressure... Parasympathomimetic Drugs:

**Cholinergic**

Cardiovascular - Decreased heart rate, decreased blood pressure due to vasodilation

Gastrointestinal - Increases motility of the GI system increasing peristalsis (movement through the intestine) and relaxing sphincter muscles.

Urinary - Stimulates urination by contracting the muscles of the bladder and relaxing the bladder’s sphincter muscles.

Other - Increases salivation, perspiration, and tears.
How a Neurotransmitter Works

1. Neuron makes a neurotransmitter.
2. Synaptic vesicles are small membrane bound structures in the axon that contain neurotransmitters.
3. The vesicles release neurotransmitters into the synaptic gap.
4. Neurotransmitter crosses the gap to bind to receptor site and performs a function.
5. Neurotransmitter is removed or taken up out of synaptic gap.
6. Restore or Remake for future use.

In our simple model of nerves, each “wire segment” corresponds to a nerve cell, called a neuron. The chemical that flows across the gaps separating neurons is called a neurotransmitter.

The body has a number of different neurotransmitters; each carries a different chemical message.

The sequence of how a neurotransmitter works:

1. The neuron makes a neurotransmitter.
2. Synaptic vesicles are small membrane bound structures in the axon terminals of nerve cells that contain neurotransmitters for storage.
3. These vesicles release neurotransmitters into the synaptic gap.
4. The neurotransmitter crosses the synaptic gap and binds to a receptor site on the adjacent neuron to cause the receptor to perform a function, usually generate an electrical impulse to continue onward through that neuron.
5. Removal and Reuptake—the neurotransmitter is either broken down or taken back up into the originating neuron.
6. Restore or Remake—for future reuse.
E. How Drugs Work

In very simple terms, drugs work by artificially creating natural body reactions generally associated with the work of neurotransmitters and hormones.

Therapeutic doses of legitimate prescription and over the counter drugs are designed to produce mild and carefully controlled simulations of the natural action of neurotransmitters and hormones.

Large, abusive doses of drugs may produce greatly exaggerated simulations of the natural action of hormones and neurotransmitters, sometimes with disastrous results.

Example: Cocaine (a sympathomimetic drug) may artificially create a message commanding the heart to beat so rapidly that cardiac arrest results.

When a person ingests a drug and artificially simulates the natural action of hormones and neurotransmitters, the body’s dynamic balance is disrupted.

The body automatically responds to the presence of the drug by producing other hormones and chemicals that can oppose the drug’s effects, and bring the body back into balance.
### How Drugs Work

**Example One:**
Person ingests a drug that speeds up bodily functions.

**Example Two:**
Person ingests a drug that slows down bodily functions.

---

**Example Number One**

If a person ingests a stimulant drug that mimics neurotransmitters associated with the sympathetic nerves, the body may react by excreting hormones that depress the bodily functions that the drug is exciting.

If a person ingested Cocaine, for example, the Cocaine would artificially stimulate the body functions. The body would then produce hormones and neurotransmitters to slow down the body functions to try to maintain homeostasis.

**Example Number Two**

If a person ingests a drug that depresses some bodily function, the body may pour out one of its natural chemicals that stimulate that same function.

An interesting situation can occur when the drug is no longer psychoactive.

The chemicals produced by the body in an effort to counteract the drug may still be active.

These natural chemicals have exactly the opposite effect on the body that the drug had: after all, that is precisely why the body produced those chemicals.

As a result, the person may feel, appear and act in a manner exactly opposite to the way he or she would feel, appear and act when under the influence of the drug.
**Neurotransmitters**

Although there are more than 100 chemicals in the brain, only about two dozen probably are true neurotransmitters.

Among the primary neurotransmitters that have been identified are:

- Norepinephrine (also called Noradrenaline)
- Acetylcholine
- Dopamine

Acetylcholine plays a role in muscle control, and affects neuromuscular or myoneural junctions. Acetylcholine also plays an important role in learning and memory. Produced by cholinergic neurons and bind to either nicotinic receptors (named after one of their most potent activators, nicotine, and the reason tobacco is so addictive) or muscarinic receptors.

- Dopamine

Dopamine plays a role in mood control and is used in treating Parkinson’s Disease. It is necessary for mental concentration, alertness, high energy, motivation, hunger regulation and sex drive. Dopamine functions in the brain’s reward pathway, release making you feel good. It is an excitatory neurotransmitter.
Neurotransmitters ("Chemical Messengers")

• Serotonin
  Serotonin is a vasoconstrictor, thought to be involved in sleep, wakefulness, and sensory perception. Tryptophan is a precursor to serotonin, and has been used to treat insomnia. Serotonin is strongly associated with mood—overall state of mind—and deficiency is associated with depression.

• Gamma Amino Butyric Acid (Abbreviated GABA)
  GABA inhibits various neurotransmitters and also causes a release of growth hormones. GABA is the major INHIBITORY neurotransmitter in the brain and acts like the “brake pedal” in a car.

• Glutamate
  Glutamate functions as an “on switch” in the brain, and is classified as an excitatory neurotransmitter. Glutamate is the most common EXCITATORY neurotransmitter in the brain and acts like the “gas pedal” in a car.
Endorphins and Enkephalins

These are the body’s natural pain relievers. They may be released in response to influences that may cause pain to the person.

There are many drugs that artificially induce the effects of neurotransmitters and hormones.
It is not uncommon for a DRE to encounter someone on the “downside.”

Definition:

“When the body reacts to the presence of a drug by releasing neurotransmitters and hormones to counteract the effects of the drug consumed to return to homeostasis.”

The neurotransmitters and hormones persist in the body longer than the drug they are responding to, resulting in the demonstration of opposite findings after the drug is gone from the body until the hormones and neurotransmitters are eliminated.

We call this situation being on the “downside” of the drug.

One example of the downside effect can be seen with an individual abusing stimulant drugs, such as cocaine or methamphetamine.

Example: with cocaine (a drug that is metabolized, or broken down by the body fairly quickly) the user may be exhibiting drowsiness and general depression by the time the DRE is called to the scene.

The concept of “downside” will be especially important to us when we discuss the effects of CNS Stimulants and drug combinations.

Then the body attempts to “counteract” the stimulant effects. When the effects of the drug diminish, the results may mimic a Narcotic Analgesic, for example.

This is the body’s attempt to reduce the size of the shift away from homeostasis caused by introduction of the drug.
While the drug is present and active in the body—applying the gas pedal in this stimulant example—the body triggers its systems to apply the brakes to try to regain homeostasis.

This involves engagement of the parasympathetic nervous system to attempt to regulate and slow the sympathetic system, as well as release of inhibitory neurotransmitters and hormones into the bloodstream. The hormone system is the slowest to engage and the slowest to disengage.
As time passes, the (stimulant) drug ingested “wears off,” by metabolism to inactivate the foreign chemical and prepare it for elimination from the body. This results in a reduced pressure on the gas pedal. While this is occurring, the body’s effort at “braking” to counter the stimulant’s pressure on the gas pedal is still ramping up and engaging to try to regain homeostasis.
The stimulant drug ingested is now essentially eliminated, or its effect has worn off, so there is no pressure on the gas pedal.

The body’s attempt at braking to regain homeostasis is now in full swing and is UNOPPOSED, so effects the OPPOSITE of the original drug ingested (stimulant) can be seen on evaluation (depressant).
With this example, the downside of CNS Stimulants can mimic Narcotic Analgesics and vice versa.

In effect, the drug(s) have worn off, however, the body is still continuing to produce neurotransmitters and hormones that counteract the effects of the drug(s). With the drug(s) not now having an effect on the body, these neurotransmitters and hormones are adversely affecting the body, causing signs and symptoms opposite of those of the prior drug effects. Keep in mind that a subject may not exhibit all of the opposite indicators. For example, a person on the “downside” of CNS Stimulants may not have ptosis, and/or they may not have facial itching. Like with drug impairment, the DRE may not observe every indicator of a particular drug category.
"Negative Feedback"

When the brain accommodates the routine presence of a drug by turning off the supply of natural chemicals that correspond to the drug

Negative Feedback

Another interesting effect that drugs can produce is called Negative Feedback.

By taking the drug, the person artificially simulates the action of certain hormones and / or neurotransmitters.

If the person continues to take the drug, the body may simply cease producing the natural chemicals that the drug simulates.

In effect, the body comes to rely on the drug to supply itself with those chemicals.

Example of Negative Feedback: when people regularly use heroin, cocaine, or marijuana, their bodies may cease producing the neurotransmitters and hormones known to be crucial for proper pain relief, stress reduction, mental stability and motivation.

One result of this may be increased tolerance to the drug: since the body isn’t producing its own natural chemicals, it can more easily stand the drug.
Tolerance

- May exhibit relatively little evidence of impairment
- Even tolerant drug users, when impaired, usually exhibit clinical evidence (i.e. vital signs, eye signs, etc.)

Even tolerant drug users, when impaired, usually exhibit clinical evidence (i.e., in the vital signs and eye signs – such as HGN).

Physical Dependence

Another result may be physical dependence, or addiction.

In simplest terms, people take drugs because they like the feelings the drugs produce.

The artificial simulation of the natural action of hormones and neurotransmitters appears to permit the user to create any feeling or mood he or she desires.

As time goes on, and negative feedback develops, the user finds that he or she can only achieve those feelings and moods if the drug is taken, and if the drug is not taken, the user does not return to a normal, non-drug-using state. He/she feels much worse in the opposite direction of the substance used. So one additional reason for physical dependence or addiction is to PREVENT WITHDRAWAL SYMPTOMS and ALLOW “NORMAL” FUNCTIONING. The habitual user must externally supply some of the drug just to feel like a typical, non-drug-using person would.
**Metabolite**

One final concept is important for an understanding of how drugs work.

A metabolite is a product of metabolism which is the chemical changes that take place when the drug reacts with enzymes and other substances in the body.

The body uses chemical reactions to break down the drug, and ultimately to eliminate it.

Example: when we drink alcohol, we initiate a series of chemical reactions that ultimately transform the alcohol into harmless carbon dioxide and water.

Sometimes, metabolites of the original drug are themselves drugs, and cause impairment.

For example, the body quickly metabolizes heroin into morphine, and it is the morphine that actually produces the effects the heroin user experiences.
F. Medical Conditions Which Sometimes Mimic Drug Impairment

Certain medical conditions or injuries may cause signs and symptoms similar to those of drug impairment.

There are times when a DRE may encounter situations where a subject arrested for drugged driving may be suffering from a medical condition that has affected the subject’s ability to operate a vehicle safely. Once the DRE makes the determination the evaluation the signs and symptoms identified are consistent with a possible medical issue, the DRE should consider taking appropriate steps to ensure the subject is referred to the proper medical personnel.

In such cases, the DRE should prepare the DRE drug evaluation report documenting his or her findings and indicating the opinion that they support medical impairment as the possible source of the impairment that has affected the subject’s ability to operate a vehicle safely. Appropriate discretion should be applied by the arresting officer whether or not an impaired driving charge is relevant, but the person should receive prompt, formal medical evaluation, if considered appropriate.

The older term, “medical rule out,” was not consistent with the law enforcement mission of the DRE evaluation, implied a formal medical evaluation which the DRE is not trained or licensed to provide, and used wording that is no longer used in the medical field due to its presumption of finality.
Medical Conditions

- Bipolar Disorder
- Conjunctivitis
- Diabetes
- Head Trauma
- Multiple Sclerosis / similar conditions

- Bipolar Disorder (Manic Depression) – a condition characterized by the alteration of manic and depressive states.
- Conjunctivitis – inflammation of the conjunctiva.

Conjunctivitis is a condition caused by infection, allergy, or irritation of the mucous membrane lining of the eyes, resulting in a “pink eye” appearance. A casual observer might mistake this for the bloodshot conditions associated with Cannabis or alcohol.

- Diabetes – a condition that can result in insulin shock (taking too much insulin) which may produce tremors, increased blood pressure, rapid respiration, lack of coordination, headache, confusion, and seizures.

The most common problem with diabetics arises when they take too much insulin, so that their blood sugar levels become extremely low. They may be very confused, sweat profusely, and exhibit increased pulse rate and increased blood pressure.

- Head Trauma – normally due to a severe blow or bump to the head.

Head trauma may injure the brain and create disorientation, confusion, lack of coordination, slowed responses and speech impairment.

- Multiple Sclerosis (MS) – a degenerative muscular disorder.

MS is a progressive disease in which the nerve fibers of the brain and spinal cord lose their myelin cover. Some signs and symptoms are abnormal sensations in the face or extremities, weakness, double vision, etc.
• Shock – a sudden or violent disturbance in the mental or emotional faculties. A shock victim may be dazed, uncoordinated, non-responsive. Other indicators include: extremely low blood pressure, fast but weak pulse, dizziness, moist clammy skin, profuse sweating, rapid shallow breathing, blue lips and fingernails.

• Stroke – a medical condition caused by a rupture or obstruction (as if by clot) of an artery of the brain.

Others – Carbon Monoxide poisoning, Seizures, Endocrine disorders, Neurological conditions, Psychiatric conditions and infections.

Normal conditions can affect vital signs: Exercise, Excitement, Fear, Anxiety, Depression, Other
Other Medical Conditions

How many different medical conditions are there?
Depending on source, from about 2,500 to 12,000 diseases and conditions!!

• Medical Conditions and Driving: A Review of the Literature (1960-2000).
• Get as much detail when you interview the subject about their medical conditions! The stage of their condition(s), whether it is treated or untreated, if it is in later stages, remission, or under control with medications.
• Almost all medical conditions present signs suggesting it is poly drug use.
• The location of the injury or disease will determine the signs and symptoms—for this reason, we CANNOT generalize a set of specific signs and symptoms for a condition as we do with the Drug Categories.
• In many injuries or diseases, the effects will be seen primarily on ONE SIDE of the body. This is the ONE SIDED (/Lateralized) SIGN. Impairment due to drugs will be seen on BOTH sides.
• If this is a medical condition, it will usually not go away in 24 hours as with a drug. It will be present well after the initial stop and arrest.
• INCOMPATIBLE or conflicting signs in the DRE evaluation (“mismatched” signs)—particularly the BACKGROUND (eating, work, hobbies, etc.), following directions, compliance, time prediction.
• COMBINED medical conditions and drug abuse: people with medical conditions also do use drugs, both legally and illegally. BOTH situations can have impairing effects and can be present at the time of the DRE evaluation.
• NHTSA published a literature review on this topic for your reference: Medical Conditions and Driving; A Review of the Literature (1960-2000).

NHSTA has produced this excellent guide reviewing numerous articles and studies on medical conditions and their effects on driving. Although this reference will not allow you to make a determination of which medical condition may be affecting a person, it will give you a good reference for understanding how many medical conditions adversely affect driving.
Preliminary Examination for a Possible Medical Impairment

- Questions
- Observations of face, breath and speech
- Initial checks of the eyes
- First check of the pulse

The Preliminary Examination Overview

The preliminary examination consists of:

- Questions.
- Observations of face, breath, and speech.
- Initial checks of the eyes.
- The initial check of the subject’s pulse.
Preliminary Examination Questions

The questions deal with injuries or medical problems the subject may have. They include:

- Are you sick or injured?
- Do you have any physical defects?
- Are you diabetic or epileptic?
- Do you take insulin?
- Are you under a doctor’s or dentist’s care?
- Are you taking any medications or drugs?

It is not only allowable, but recommended that the DRE ask more questions related to these areas. This is especially true if the subject answers any of these questions in the affirmative.
DRE Medical Impairment Definition

There are times when a DRE may encounter situations where a subject arrested for drugged driving may be suffering from a medical condition that has affected the subject’s ability to operate a vehicle safely. In other words, the DRE through his or her evaluation has eliminated impairing substances as the probable cause of impairment, and while doing so, identified signs and symptoms that are consistent with a medical issue. Once the DRE makes the determination, the DRE should consider taking appropriate steps to ensure the subject is referred to the proper medical personnel.

In such cases, the DRE should prepare the DRE drug evaluation report documenting his or her findings that support an opinion of a DRE medical impairment.

For purposes of DRE and the DEC Program, medical impairment is defined as, “An opinion made by a DRE based on the evaluation that the state of a suspected impaired driver is more likely related to medical impairment that has affected the subject’s ability to operate a vehicle safely.”

The suggested way to document this type of opinion in Step 11 of the DRE report would be: “It is my opinion that (Subject’s name) is unable to operate a vehicle safely due to medical impairment.”

DREs and other police officers will at times encounter individuals with mental illness or intellectual/developmental disabilities. These individuals may exhibit signs and symptoms very similar to those of an individual impaired by drugs and/or alcohol. These individuals may also be experiencing coexisting conditions of mental illness with drug impairment. It is important for DREs to make every effort to prevent violent interactions using an array of tools and resources necessary for positive, successful outcomes. Using a strategic approach to interactions with individuals with suspected mental health problems or intellectual/developmental disabilities can ensure officer safety through the DRE interaction.

G. Summary

Basic understanding of how the body works is necessary to:

- Understand why the drug evaluation is conducted in a systematic manner.
- Understand why the results, when viewed in their totality, provide reliable indicators of impairment within broad categories of drugs.

This limited overview will not qualify participants as medical specialists.

The knowledge gained during this session must be supplemented by additional reading and/or instruction.

The body of knowledge in this area is being constantly expanded.

The body maintains homeostasis (equilibrium) by constantly adjusting to changes in the external and internal environment:

When drugs are introduced into the body this process comes into play. When drugs interact in the body they tend to:

- speed things up, or slow things down, or confuse signals, or block signals, or
- some combination of the above.

The effects of drugs can be detected and / or observed in the drug influence evaluation.
TOPICS FOR STUDY

1. What is a neurotransmitter? What is a hormone?
2. What is a dendrite? What is an axon? What is a synapse?
3. Do arteries carry blood toward the heart or away from the heart?
4. What is unique about the Pulmonary Artery?
5. What are the two types of nerves that make up the Autonomic Nervous Sub-System?
6. Is Cocaine sympathomimetic or parasympathomimetic? What about Heroin?
7. Explain the concept of the “downside effect.” Explain the concept of “Negative Feedback.”
8. What do we call the nerves that carry messages away from the brain? What do we call the nerves that carry messages toward the brain?
QUESTIONS FOR PHYSIOLOGICAL PURSUIT

1. Name the major body systems.
   Muscular, Urinary, Respiratory, Digestive, Endocrine, Reproductive, Skeletal, Integumentary, Nervous, and Circulatory.

2. What vein carries oxygenated blood?
   Pulmonary vein. The pulmonary vein returns oxygenated blood from the lungs to the left side of the heart. The left side of the heart then pumps the oxygenated blood via arteries throughout the body. The pulmonary artery carries de-oxygenated blood from the right side of the heart to the lungs.

3. What is the function of the endocrine system?
   The endocrine system is composed of ductless glands that release chemical messengers, called hormones, into the bloodstream. The function is the regulation of various bodily processes by the production and release of hormones.

4. Explain the “downside” effect of a drug.
   The “downside” effect of a drug refers to the post euphoric stage of a drug’s effects. As the effects of a drug wear off, the individual may display effects that are essentially the opposite of the “high” state that was brought about by the drug. This effect is in part due to the body’s attempt to counteract the effects of a drug.

5. Define homeostasis.
   Homeostasis is basically a physiological equilibrium or dynamic balance. Homeostasis refers to the body’s mechanisms that keep the levels of fluids, salts, chemicals and other internal substances in a safe balance. The regulation of temperature is an example of homeostasis at work.

6. Hair and nails are part of what system?
   The Integumentary system. This system also includes the skin.

7. Name the two circulatory systems.
   The systemic circulatory system, which is driven by the left side of the heart, and pulmonary circulatory system, driven by the heart’s right side.

8. The functions of the organs of the body are controlled by what two systems?
   The endocrine and nervous system.

9. Define synapse, axon, and dendrite.
   These structures are all part of the nerve cell, or neuron. The axon is the part of the neuron that releases neurotransmitter from a terminal into the synapse. An electrical impulse causes the axon to release the neurotransmitter. The synapse is the gap between nerve cells and is also called the synaptic gap. The dendrite refers to a structure that receives the chemical message from the neurotransmitter. There are often many dendrites on each neuron. The neurotransmitter fits into receptor sites on the dendrite and causes an electrical message to be sent to the neuron’s body.
10. Define neurotransmitter and hormone.
   Both are chemical messengers. Neurotransmitters are chemicals that send messages within the nervous system. Hormones are released by glands in the endocrine system into the bloodstream.

11. _______ nerves carry messages AWAY from the brain to the body’s muscles and organs.
    Efferent, or Motor nerves. These nerves cause a motor response. Afferent nerves send sensory messages to the brain. The central nervous system interprets these messages and if appropriate, calls for a response through the efferent nerves.

12. The ______ nervous system commands the body to react to stress, fear, and excitement.
    The Sympathetic nervous system, a division of the Autonomic Nervous System, produces the body’s “fight or flight” response to real or perceived danger. Drugs that mimic the activation of the sympathetic nervous system are “sympathomimetic”. CNS Stimulants have effects closest to the effects of sympathetic nervous system activation.

13. Explain “negative feedback.”
    Refers to the body’s response to taking a drug that has effects similar to natural internal chemicals. After repeated exposure to the drug, the body responds by slowing, or even stopping the production of the internal chemical. In time, the body begins to rely on the drug. An example of negative feedback involving legitimate substances is insulin dependent diabetics. Once an individual begins to take insulin, the person’s body will eventually stop making its own insulin. The person must obtain insulin by administering it.

14. What two types of nerves make up the autonomic nervous subsystem?
    The Sympathetic and Parasympathetic nerves. The sympathetic nervous system initiates the body’s “fight or flight” response to real or perceived danger. The parasympathetic nervous system parallels or balances the sympathetic nervous system. This system initiates calming and digestive processes.

15. Define metabolite.
    A metabolite is the by-product of the body’s chemical breakdown of various substances for elimination. Metabolites may or may not be psychoactive by themselves. Often times a toxicological analysis will disclose various metabolites of a drug, rather than the parent drug.
Participant Manual

Drug Recognition Expert Course

Session 7
Examination of Vital Signs
Upon successfully completing this session the participant will be able to:

- Explain the purposes of the various vital signs examinations in the drug influence evaluation procedure.
- Explain the administrative procedures for these examinations.
- Explain the clues obtained from these examinations.
- Document the examinations of vital signs accurately and completely.
- Correctly answer the “topics for study” at the end of this session.

CONTENT SEGMENTS ................................................................. LEARNING ACTIVITIES

A. Purpose of the Examinations .............................................. Instructor-Led Presentations
B. Procedures and Clues ......................................................... Instructor-Led Demonstrations
C. Demonstrations ................................................................. Audio Tape Presentation
D. Documentation Procedures ................................................. Participant-Led Demonstrations
E. Practice ................................................................................ Participants’ Hands-On Practice

Reading Assignments
A. Purposes of the Examinations

The vital signs that are relevant to the drug influence evaluation include:

- Pulse Rate
- Blood Pressure
- Temperature

Different types of drugs affect these vital signs in different ways.

Certain drugs tend to “speed up” the body and elevate these vital signs.

Clarification:

- Pulse may quicken
- Blood pressure may rise
- Temperature may rise

Other drugs tend to “slow down” the body and lower these vital signs.

Clarification:

- Pulse may slow
- Blood pressure may drop
- Temperature may drop

Systematic examination of the vital signs gives us much useful information concerning the possible presence or absence of various categories of drugs.
B. Procedures and Clues

Measurement of Pulse Rate

Pulse is the expansion and contraction of an artery generated by the pumping action of the heart. Pulse Rate is the number of pulsations in an artery per minute.

- An artery is a strong, elastic blood vessel that carries blood from the heart to the body tissues.
- A vein is a blood vessel that carries blood back to the heart from the body tissues.
- When the heart contracts, it squeezes blood out of its chambers into the arteries.
- The surging blood causes the arteries to expand.
- By placing your fingers on the skin next to an artery and pressing down, you can feel the artery expand as the blood surges through.

By keeping your fingers on the artery and counting the number of pulses that occur in one minute, you will measure the pulse rate.

Pulse is easy to measure, once you locate an artery close to the surface of the skin.
Radial Artery Pulse Point

One convenient pulse point involves the radial artery.

The radial artery can be located in or near the natural crease of the wrist, on the side of the wrist next to the thumb.

Place the tips of your right hand’s index finger and middle finger into the crease of your wrist, and exert a slight pressure.

You should be able to feel the pulse in your radial artery.

Brachial Artery Pulse Point

Another pulse point involves the brachial artery.

The brachial artery can be located in the crook of the arm, halfway between the center of the arm and the side of the arm closest to the body.

Place the tips of your right hand’s index and middle fingers into the crook of your left arm, close to the body, and exert a slight pressure.

You should be able to feel the pulse in your brachial artery.
Carotid Artery Pulse Point

Another pulse point involves the carotid artery.
The carotid artery can be located in the neck, on either side of the center of the throat.
  • You should be able to feel the pulse in your carotid artery.
Basic Do’s and Don’ts of Measuring Pulse

- Don’t use your thumb to apply pressure while measuring a subject’s pulse
- When measuring the pulse rate, use time intervals of 30 seconds

- If you use the carotid artery pulse point, don’t apply pressure to both sides of the center of the throat: this can cut off the supply of blood to the brain

- When measuring the pulse rate, use time intervals of 30 seconds
Technical Terms Associated With Pulse Rate

- Tachycardia: Abnormally rapid heart rate
- Bradycardia: Unusually slow heart rate
- Arrhythmia: Abnormal heart rate rhythm

Some Technical Terms Associated with Pulse Rate

- Tachycardia: abnormally rapid heart rate
- Bradycardia: unusually slow heart rate
- Arrhythmia: abnormal heart rhythm

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<td>50 or less</td>
<td>76-78</td>
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<tr>
<td>52-54</td>
<td>80-82</td>
</tr>
<tr>
<td>56-58</td>
<td>84-86</td>
</tr>
<tr>
<td>60-62</td>
<td>88-90</td>
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<td>64-66</td>
<td>92-94</td>
</tr>
<tr>
<td>68-70</td>
<td>96-98</td>
</tr>
<tr>
<td>72-74</td>
<td>100 or more</td>
</tr>
</tbody>
</table>
Blood Pressure

Millimeters of Mercury = mmHg

Example: a blood pressure of 120 means that the blood is pressing on the walls of the artery with enough force to push liquid mercury 120 millimeters up a glass tube.

We commonly abbreviate “millimeters of mercury” as mmHg.
Definitions Concerning Blood Pressure

- **Blood Pressure**
  The force that the circulating blood exerts on the walls of the arteries

- **Systolic Pressure**
  The maximum blood pressure, reached as the heart contracts

- **Diastolic Pressure**
  The minimum pressure, reached when the heart is fully expanded

**Measurement of Blood Pressure**

- Blood Pressure is the force that the circulating blood exerts on the walls of the arteries.
- Blood pressure is measured in millimeters of mercury.
- Blood Pressure changes constantly as the heart contracts and relaxes.
- Blood Pressure reaches its maximum as the heart contracts and sends the blood surging through the arteries. This is called the systolic pressure.
- Blood Pressure reaches its minimum when the heart is fully expanded. This is called the diastolic pressure.
- It is always necessary to measure and record both the systolic and diastolic blood pressure.
Sphygmomanometer

The device used for measuring blood pressure is called a sphygmomanometer.

The sphygmomanometer has a special cuff that can be wrapped around the subject’s arm and inflated with air pressure.

As the pressure in the cuff increases, the cuff squeezes tightly on the arm.

Wrap the cuff around the participant volunteer’s arm and inflate it.

When the pressure gets high enough, it will squeeze the artery completely shut.

Blood will cease flowing through the brachial artery. And, since the brachial artery “feeds” the radial artery, blood will also cease flowing through the radial artery.
If we slowly release the air in the cuff, the pressure on the arm and on the artery will start to drop.

Release the pressure in the cuff on the participant volunteer’s arm.

Eventually, the pressure will drop enough so that blood will once again start to flow through the artery.

Blood will start flowing in the artery once the pressure inside the artery equals the pressure outside the artery.

The two pressures will become equal when the air pressure in the cuff drops down to the systolic pressure.

When that happens, blood will spurt through the artery each time the heart contracts.

Once the air pressure in the cuff drops down to the diastolic level, the blood will flow continuously through the artery.
Overview of Procedures for Measuring Blood Pressure

Apply enough air pressure to the cuff to cut off the flow of blood through the artery.
Slowly release the pressure in the cuff.
Slowly release the air pressure until the blood just begins to spurt through the artery: that level will be the systolic pressure.
Continue to release the air pressure until the blood flows continuously: that will be the diastolic pressure.
Apply the stethoscope to the skin directly above the artery.
Apply pressure to the cuff, enough to cut off the flow of blood.
When no blood is flowing through the artery, we hear nothing through the stethoscope.
Inflate the cuff on the participant volunteer’s arm.
Slowly release the air from the cuff, letting the pressure start to drop.
Release the air in the cuff.
When we drop to the systolic pressure, we start to hear a spurting sound.
As we continue to allow the air pressure to drop, the surges of blood become steadily longer.
When we drop to the diastolic pressure, the blood flows steadily and all sounds cease.
Korotkoff Sounds

The sounds that we listen to are called Korotkoff Sounds. They are divided into 5 phases:

- Phase 1 – the first appearance of clear, tapping sounds that gradually increase in intensity.
- Phase 2 – the sounds change to a murmur and take on a swishing quality.
- Phase 3 – the sounds develop a loud, knocking quality (not quite as clear as the Phase 1 sounds).
- Phase 4 – the sounds become muffled and again have a faint swishing quality.
- Phase 5 – the sounds cease.
Familiarization with the Sphygmanometer

The compression cuff contains an inflatable rubber bladder.

A tube connects the bladder to the manometer, or pressure gauge.

Clarification: the manometer displays the air pressure inside the bladder. In the DEC program, we use an aneroid (without fluid) pressure gauge.

Another tube connects the bladder to the pressure bulb, which can be squeezed to inflate the bladder.

The pressure control valve permits inflation of the bladder and regulates the rate at which the bladder is deflated.

To inflate the bladder, the pressure control valve must be twisted all the way to the right.

When the valve is twisted all the way to the right, air can be pumped into the bladder, but no air can escape from the bladder.

To deflate the bladder, twist the valve to the left.

The more the valve is twisted to the left, the faster the bladder will deflate.
Details of Blood Pressure Measurement

If it proves difficult to hear the Korotkoff sounds, simply have the subject elevate the arm and squeeze the fist several times, to drain the arm: the Korotkoff sounds louder.

The manometer (pressure gauge) may be clipped on the subject’s sleeve, so that it is readily viewable.

Twist the pressure control valve all the way to the right.
Details of Blood Pressure Measurement

- Place stethoscope over brachial artery
- Rapidly inflate bladder to 180 mmHg
- Twist the valve slightly to the left
- Keep your eyes on the gauge and listen for the Korotkoff sounds

Do’s and Don’ts of Blood Pressure Measurement

- Do wait 3 minutes to repeat the measurement, if needed
- Don’t re-inflate cuff once you start releasing the pressure

Put the stethoscope earpieces in your ears.

Make sure the earpieces are turned forward, i.e. toward the nose.

Place the diaphragm or bell of the stethoscope over the brachial artery.

Rapidly inflate the bladder to a pressure of at least 180.

Twist the pressure control valve slightly to the left to release the pressure slowly.

The pressure should be released at a speed that takes one full second for the needle to move a single gradation (i.e. 2 millimeters of mercury) on the gauge.

Keep your eyes on the gauge and listen for the Korotkoff sounds.

Do’s and Don’ts of Blood Pressure Measurement

If you inflate the bladder and then need to repeat the measurement, wait at least three minutes to allow the subject’s artery’s to return to normal.

- Do wait 3 minutes to repeat the measurement if a second measurement is needed.
- Don’t re-inflate cuff once you start releasing the pressure.
Technical Terms Associated With Blood Pressure

- Hypertension: Abnormally high blood pressure
- Hypotension: Abnormally low blood pressure

Some Technical Terms Associated with Blood Pressure

- Hypertension: abnormally high blood pressure.
- Hypotension: abnormally low blood pressure.
Measurement of Temperature

Body temperature is measured using an oral digital thermometer.

C. Demonstrations

Pulse Rate Measurement

- Radial artery pulse point:
- Carotid artery pulse point:

Blood Pressure Measurement

D. Documentation Procedures

E. Practice
TOPICS FOR STUDY / ANSWERS

1. Where is the Radial Artery pulse point?

2. Why should you never attempt to feel a subject’s pulse with your thumb?

3. Does an artery carry blood to the heart or from the heart?

4. What does the symbol “Hg” represent?

5. What is Diastolic pressure?

6. When do the Korotkoff Sounds begin?

7. Name and describe the major components of a Sphygmomanometer.

8. Which of the seven categories of drugs generally will cause blood pressure to be elevated?
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Upon successfully completing this session the student will be able to:

- Describe the sequence in which examinations and other activities are performed during the drug influence evaluation procedure.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES
A. Live Demonstrations ................................................................. Instructor-Led Presentations
B. Video Demonstrations ........................................................... Instructor-Led Demonstrations
......................................................................................................................... Video Presentations
......................................................................................................................... Reading Assignments
A. Live Demonstrations

For these live demonstrations, participants must be grouped into teams of not more than 12 members. Each team must be taken to a separate classroom. At least two instructors must work with each team. This is to ensure that all participants have the opportunity for a close and detailed observation of the demonstrations.

Preliminary eye checks:

- equal tracking
- equal pupil size
- resting nystagmus
- blindness
- eyelids

Vital Signs Examinations

- Blood Pressure
- Temperature
- Second Check of Pulse
Live Demonstrations

- Pupil Size Estimations
  - Room Light
  - Near Total Darkness
  - Direct Light

Dark Room Examinations

Pupil Size Estimations:

- Room light
- Near Total Darkness
- Direct light

Reaction to Light
Check of Nasal Area
Check of Oral Cavity

Statements made by subject

Behavior during entire evaluation
QUESTIONS?
Learning Objectives

- Explain a brief history of the CNS Depressant category of drugs
- Identify common drug names and terms associated with this category
- Identify common methods of administration for this category
- Describe the symptoms, observable signs and other effects associated with this category
- Explain the typical time parameters, i.e. onset and duration of effects associated with this category
- List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of this category of drugs
- Correctly answer the “topics for study” questions at the end of this session

Upon successfully completing this session the participant will be able to:

- Explain a brief history of the CNS Depressant category of drugs.
- Identify common drug names and terms associated with this category.
- Identify common methods of administration for this category.
- Describe the symptoms, observable signs and other effects associated with this category.
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CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES
A. Overview of the Category ............................................................. Instructor-Led Presentations
B. Possible Effects ................................................................. Instructor-Led Demonstrations
C. Onset and Duration of Effects ........................................ Reading Assignments
D. Overdose Signs and Symptoms .............................................. Video Presentations
E. Expected Results of the Evaluation........................................ Slide Presentations
F. Classification Exemplar
A. Overview of the Category

*CNS Depressants*

Central Nervous System Depressants slow down the operations of the brain.

- Depressants first affect those areas of the brain that control a person’s conscious, voluntary actions.
- Such as judgment, inhibitions and reaction time.
- As the dose is increased, depressants begin to affect the parts of the brain that control the body’s automatic processes, heartbeat, respiration, etc.

The CNS Depressant category includes the single most commonly abused drug in America.

- Alcohol has been used and abused since prehistoric times.
- Alcohol and its effects are familiar to most people.
- Alcohol is a model for the CNS Depressant category: with some exceptions, all depressants produce effects that are quite similar to the effects of alcohol.
Chloral Hydrate

Non-alcohol CNS Depressants have been around for more than 150 years. The first non-alcohol CNS Depressant was Chloral Hydrate. It was developed in 1832 and utilized clinically in 1869. Chloral Hydrate was derived from alcohol. Chloral Hydrate is still produced and prescribed today. It is a sedative used in the short-term treatment of insomnia and to relieve anxiety and induce sleep before surgery. “Noctec” is a registered brand name of Chloral Hydrate.
Sub Categories of CNS Depressants

There are six major subcategories of CNS Depressants other than alcohol.

Barbiturates

More than 250 different barbiturates have been produced; of these, about 50 have been accepted for medical use.

- Derivatives of Barbituric Acid
- First produced in 1864
- Very common in use and abuse today

Non-Barbiturates

Chloral Hydrate belongs to the non-barbiturate subcategory.

- Synthetic compounds with a variety of chemical structures
- Prescribed to help with some of the unintended side effects of barbiturates including sleepiness or drowsiness
- Still produce physical and psychological dependence

Anti-Anxiety Tranquilizers

The Anti-Anxiety Tranquilizers are also known as the “minor tranquilizers.” They include the group of drugs known as the “Benzodiazepines” examples of which are Valium, Xanax, and Librium.

- First produced in 1950
- In very wide spread use
- Frequently abused
Anti-Depressants
Sometimes called the “mood elevators.”

Anti-Psychotic Tranquilizers
Sometimes called the “major tranquilizers.”
Anti-psychotic tranquilizers were first introduced in the early 1950’s. They provide a way to manage schizophrenia and other mental disorders, and allow psychiatric patients to be released from hospitals and to lead fairly normal lives.
The most familiar Anti-Psychotic Tranquilizer is “Thorazine.”

Combinations
This subcategory includes a small class of depressants involving various combinations of the other five subcategories.
Specific Barbiturates Examples

<table>
<thead>
<tr>
<th>Drug</th>
<th>Brand Name</th>
<th>Street Names</th>
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<tbody>
<tr>
<td>Amobarbital</td>
<td>Amytal</td>
<td>Blues, Blue Heavens</td>
</tr>
<tr>
<td>Amosecobarbital</td>
<td>Tuinal</td>
<td>Rainbows, Christmas Trees</td>
</tr>
<tr>
<td>Pentobarbital</td>
<td>Nembutal</td>
<td>Yellows, Yellow Jackets</td>
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<tr>
<td>Phenobarbital</td>
<td>Luminal</td>
<td>Pink Ladies</td>
</tr>
<tr>
<td>Secobarbital</td>
<td>Seconal</td>
<td>Reds, Red Devils, RDs, Fender Benders, F-40's</td>
</tr>
</tbody>
</table>

The Barbiturates

- Amobarbital (Trade name “Amytal”) Street names “blues”; “blue heavens”
- Amosecobarbital (Trade name “Tuinal”) Street names “rainbows”; “Christmas Trees”

This is a combination of Amobarbital and Secobarbital.

- Pentobarbital (Trade name “Nembutal”) Street names “yellows”; “yellow jackets”
- Phenobarbital (Includes Luminal and other trade names) Street name “pink ladies”.
- Secobarbital (Trade name “Seconal”) Street names “reds”; “red devils”; “RDs”; “fender benders”; F-40s”
### Specific Non-Barbiturates

#### Examples

<table>
<thead>
<tr>
<th>Drug</th>
<th>Brand Name</th>
<th>Street Names</th>
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<tbody>
<tr>
<td>Carisoprodol</td>
<td>Soma</td>
<td></td>
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<tr>
<td>Chloral hydrate</td>
<td>Felsule, Noctec</td>
<td>Knock Out Drops, Mickey Finn</td>
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<tr>
<td>Diphenhydramine Hydrochloride</td>
<td>Benadryl, Sominex</td>
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<tr>
<td>Diphenylhydantoin Sodium</td>
<td>Dilantin</td>
<td></td>
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<tr>
<td>Eszopiclone</td>
<td>Lunesta</td>
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#### Specific Non-Barbiturates

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<thead>
<tr>
<th>Drug</th>
<th>Brand Name</th>
<th>Street Names</th>
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<tr>
<td>Ethchlorvynol</td>
<td>Placidyl</td>
<td>GHB, Liquid X</td>
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<td>Gamma Hydroxybutyrate</td>
<td>Noludar</td>
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<tr>
<td>Methyprylon</td>
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<td>Methaqualone</td>
<td>Parest, Quaalude, Sopor, Optimil, Mandrax</td>
<td>Ludes</td>
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<td>Paraldehyde</td>
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<tr>
<td>Zolpidem</td>
<td>Ambien, Edluar, Stilnoct</td>
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</table>

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**The Non-Barbiturates**

The absence of street names implies only that illicitly manufactured versions of these drugs are not common. The legally manufactured versions are abused, however.

- Carisoprodol (Trade name “Soma”)
- Chloral Hydrate (Trade names “Noctec”, “Sominos”) (Street names “Knockout drops”; “Mickey Finn”)
- Diphenhydramine Hydrochloride (Trade names “Benadryl”; “Sominex”; and “Nytol”)
- Diphenylhydantoin Sodium (Trade name “Dilantin”)
- Eszopiclone (Trade names “eszopiclone”, “Estorra” and “Lunesta”)
- Ethchlorvynol (Trade name “Placidyl”)
- Gamma Hydroxybutyrate (Street name “GHB”; “Liquid X”; “1,4-butanediol”)
- Methaqualone (Trade names “Parest”; “Quaalude”; “Sopor”; “Optimil”; “Mandrax”) (Street name “ludes”)
- Paraldehyde (Trade name “Paral”)
- Zolpidem (Trade names “Ambien”, “Edluar” and “Stilnoct”)
The Anti-Anxiety Tranquilizers

- Alprazolam (Trade names “Xanax”, “Niravam”) (Street name “Bars”; “Zannys”; “Blues”)
- Chlordiazepoxide (Trade name “Librium”)
- Clonazepam (Trade name “Klonopin”)
- Diazepam (Trade name “Valium”)
- Estazolam (Trade name “ProSom”)
- Flunitrazepam (Trade name “Rohypnol”) (Street name “Roofies”; “Roches”)
- Flurazepam (Trade names Dalmadorm”, “Dalmane”)
- Lorazepam (Trade names “Ativan” and “Temesta”)
- Meprobamate (Trade names “Equanil”, “Miltown”)
- Oxazepam (Trade name “Serax”)
- Temazepam (Trade name “Restoril”)
- Triazolam (Trade name “Halcion”)

---

**Specific Anti-Anxiety Tranquilizers Examples**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Brand Name</th>
<th>Street Names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alprazolam</td>
<td>Xanax</td>
<td>Bars, Zanny Bars</td>
</tr>
<tr>
<td>Chlordiazepoxide</td>
<td>Librium</td>
<td></td>
</tr>
<tr>
<td>Clonazepam</td>
<td>Klonopin</td>
<td></td>
</tr>
<tr>
<td>Diazepam</td>
<td>Valium</td>
<td></td>
</tr>
<tr>
<td>Estazolam</td>
<td>ProSom</td>
<td></td>
</tr>
</tbody>
</table>

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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Flunitrazepam</td>
<td>Rohypnol</td>
<td></td>
</tr>
<tr>
<td>Flurazepam</td>
<td>Dalmadorm, Dalmane</td>
<td></td>
</tr>
<tr>
<td>Lorazepam</td>
<td>Ativan, Temesta</td>
<td></td>
</tr>
<tr>
<td>Meprobamate</td>
<td>Equanil, Miltown</td>
<td></td>
</tr>
<tr>
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<td>Serax</td>
<td></td>
</tr>
<tr>
<td>Temazepam</td>
<td>Restoril</td>
<td></td>
</tr>
<tr>
<td>Triazolam</td>
<td>Halcion</td>
<td></td>
</tr>
</tbody>
</table>
The Anti-Depressants

- Amitriptyline Hydrochloride (Trade names “Elavil”; “Endep”)
- Bupropion (Trade name “Wellbutrin”)
- Citalopram (Trade name “Celexa”)
- Desipramine Hydrochloride (Trade names “Norpramin”; “Pertofrane”)
- Doxepin Hydrochloride (Trade names “Adapin”; “Sinequan”)
- Duloxetine (Trade name “Cymbalta”)
- Escitalopram (Trade name “Lexapro”)
- Fluoxetine (Trade names “Prozac”; “Sarafem”)
- Fluvoxamine (Trade name “Luvox”)
- Imipramine (Trade name “Tofranil”)
- Paroxetine (Trade name “Paxil”)

<table>
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</thead>
<tbody>
<tr>
<td>Amitriptyline</td>
<td>Elavil, Endep</td>
<td></td>
</tr>
<tr>
<td>Bupropion</td>
<td>Wellbutrin, Zyban</td>
<td></td>
</tr>
<tr>
<td>Citalopram</td>
<td>Celexa</td>
<td></td>
</tr>
<tr>
<td>Desipramine</td>
<td>Norpramin, Pertofrane</td>
<td></td>
</tr>
<tr>
<td>Doxepin Hydrochloride</td>
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</tbody>
</table>
Specific Anti-Depressants

<table>
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<tr>
<th>Drug</th>
<th>Brand Name</th>
<th>Street Names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenelzine Sulfate</td>
<td>Nardil</td>
<td></td>
</tr>
<tr>
<td>Sertraline</td>
<td>Zoloft</td>
<td></td>
</tr>
<tr>
<td>Trazodone</td>
<td>Desyrel</td>
<td></td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>Effexor</td>
<td></td>
</tr>
</tbody>
</table>

Specific Anti-Psychotic Tranquilizers Examples

<table>
<thead>
<tr>
<th>Drug</th>
<th>Brand Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpromazine</td>
<td>Thorazine</td>
</tr>
<tr>
<td>Droperidol</td>
<td>Inapsine, Innovar</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>Haldol</td>
</tr>
<tr>
<td>Lithium Carbonate</td>
<td>Lithane</td>
</tr>
</tbody>
</table>

- Phenelzine Sulfate (Trade name “Nardil”)
- Sertraline (Trade name “Zoloft”)
- Trazodone (Trade name “Desyrel”)
- Venlafaxine (Trade name “Effexor”)

Anti-Depressants Exceptions
Anti-Depressants may cause dry mouth, sore throat, blurred vision, urinary retention, muscle twitching, restlessness, and increased anxiety.

The Anti-Psychotic Tranquilizers

- Chlorpromazine (Trade name “Thorazine”)
- Droperidol (Trade name “Inapsine”)
- Haloperidol (Trade name “Haldol”)
- Lithium Carbonate (Trade name “Lithane”)

Revised: Drug Recognition Expert Course Session 9
10/2015 Central Nervous System Depressants Page 10 of 26
Some Combinations of Depressants

• Chlordiazepoxide in combination with Amitriptyline  
  Trade name: “Limbitrol”
• Chlordiazepoxide Hydrochloride in combination with Clidinium Bromide  
  Trade name: “Librax”
• Perphenazine in combination with Amitriptyline Hydrochloride  
  Trade name: “Triavil” and “Etrafon”

The Combinations

• Chlordiazepoxide in combination with Amitriptyline (trade name “Limbitrol”)
• Chlordiazepoxide Hydrochloride in combination with Clidinium Bromide (Trade name “Librax”)
• Perphenazine in combination with Amitriptyline Hydrochloride (Trade name “Triavil” and “Etrafon”)
Methods of Ingestion of CNS Depressants

- Most common and easiest method is orally
- There are reports of subjects crushing Xanax and Soma tablets, snorting the powder and getting an effect. This method results in a slow but long absorption process producing depressant symptoms for some time.
- Some abusers prefer to use intravenous injection for Barbiturates
- Some abusers experience a “flash” or “rush” from intravenous injection of Barbiturates, that they do not experience from oral ingestion

The injection paraphernalia used for Barbiturates are very similar to those used for Heroin. Examples:

- Spoon, for heating and dissolving the barbiturate
- Cotton, for filtering the solution when drawing it into the needle
- Hypodermic syringe
- Tourniquet

However, the Barbiturate abuser will use a larger hypodermic needle because the barbiturate solution is thicker than the heroin solution.

The injection sites on the skin of a Barbiturate abuser appear quite different from those of a Heroin addict. A large swelling, about the size of a quarter or fifty cent piece frequently will appear at the Barbiturate injection site.

Necrosis may occur: i.e. a decaying of the body’s tissue at the injection site.

The dead tissue may begin to separate from the living tissue, producing ulcerations.

The Barbiturate user who injects the drug usually will not display the same type of track marks as the heroin addict who uses repeated injections along the same vein. Barbiturate abusers often will inject in parts of the body other than the forearm, and will commonly exhibit the characteristic swellings at random locations on the extremities.
Possible Effects of CNS Depressants

- Reduced inhibitions
- Divided attention impairment
- Slowed reflexes
- Impaired judgment and concentration
- Impaired vision
- Lack of coordination
- Slurred, mumbled or incoherent speech
- Emotional instability

B. Possible Effects

CNS Depressants produce impairments of the human mind and body that essentially mirror alcohol impairment.

- Reduced social inhibitions
- Divided attention impairment
  - Clarification: impede the person’s ability to concentrate on more than one thing at a time.
- Slowed reflexes
- Impaired judgment and concentration
- Impaired vision
  - Elaboration: ability to focus eyes may be impaired; “double vision” may develop.
- Lack of coordination
- Slurred, mumbled, or incoherent speech
- Produce a variety of emotional effects, such as euphoria, depression, suicidal tendencies, laughing or crying without provocation, etc.

Generally speaking, a person under the influence of CNS Depressants will look and act drunk.
C. Onset and Duration Effects

Depressant drugs can be grouped loosely into four classes based on how quickly they take effect and how long their effects last.

Ultrashort:
- Very fast acting, very brief effects
- Take effect in a matter of seconds
- Effects last only a few minutes
- Very rarely are the “drugs of choice” for drug abusers

Ultrashort depressants are sometimes used at the beginning of a surgical operation, in conjunction with an inhaled anesthetic.

Clarification: to provide a momentary sedation to ease the patient’s anxiety and allow for the proper administration of the anesthetic.

 Psychiatrists sometimes use ultrashort depressants at the beginning of a session, to reduce the client’s inhibitions and foster a free and open communication.

An example of an ultra short depressant is Brevital Sodium which is a rapid, injectable barbiturate anesthetic mainly used in hospital settings.
**Short Acting CNS Depressants**

- They produce effects reasonably quickly
- Effects last long enough to “enjoy” the effects
- Most commonly abused class of CNS Depressants

---

**Short Acting**

Short: fairly fast acting, effects last for approximately 4 hours.

- They produce effects reasonably quickly
- The effects last long enough to “enjoy” the effects
- Generally takes effect in 10 to 15 minutes
- This is the most commonly abused class of CNS Depressants

Short Acting Depressants frequently are prescribed as a treatment for insomnia. They also may be used as a pre-anesthetic medication to calm a patient prior to surgery.

A common example of a short acting Depressant, Secobarbital, Brand name “Seconal”
Intermediate Acting CNS Depressants

- Relatively slow acting, but prolonged effects
- Generally take effect in about 30 minutes
- Effects typically last about 6-8 hours

Intermediate Acting

Intermediate: relatively slow acting, but prolonged effects.

- Generally take effect in about 30 minutes
- Effects typically last about 6 – 8 hours
- Fairly often abused, especially by users who desire a longer lasting state of intoxication. Medical use of this class of drugs is similar to that of short acting Depressants (i.e. treat insomnia, etc.) Common example of an intermediate Depressant: Amobarbital, brand name “Amytal”.

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Revised: Drug Recognition Expert Course Session 9
10/2015 Central Nervous System Depressants
Long Acting CNS Depressants

- Generally take effect about one hour after ingestion
- Effects typically last 8-14 hours
- Phenobarbital (Luminal), and Flurazepam (Dalmane) are examples

Long Acting: delayed but long lasting effects.

- Generally take effect about one hour after ingestion
- Effects typically last 8 – 14 hours.
- Generally not the “drugs of choice” for abusers, however, some people will abuse the long acting Depressants if the more popular short and intermediate types are not readily available.

Long acting Depressants are used medically in the control of epilepsy and of other conditions that can cause convulsions.

They can also be used to provide continuing sedation to patients suffering from extreme anxiety.

Two examples of a long acting depressant are Phenobarbital (Luminal) and Flurazepam (Dalmane), both used primarily as a daytime sedative and anticonvulsant.
How would you classify Alcohol in terms of the onset and duration of its effects?

Alcohol as a Specific Example
Non-Barbiturates

- “Mickey Finn” (Noctec or Felsule)
- Placidyl (Ethchlorvynol)
- Soma (Carisoprodol)
- GHB (Gamma Hydroxybutyrate)
- Ambien (Zolpidem)

Anti-Anxiety Tranquilizers

- Valium (Diazepam)
- Librium (Chlordiazepoxide)
- Xanax (Alprazolam)
- Serax (Oxazepam)
- Klonopin (Clonazepam)
- Ativan (Lorazepam)
- Rohypnol (Flunitrazepam)
D. Overdose Signs and Symptoms

Overdoses of the Central Nervous System Depressants produce symptoms essentially identical to those of alcohol overdoses.

- Subject will become extremely drowsy and may pass out
- The heartbeat (pulse) will be rapid and weak
- Respiration will become shallow
- Skin may feel cold and clammy
- One major danger with CNS Depressant overdoses is death from respiratory failure
- A sufficiently high dose of CNS Depressant will suppress the portions of the brain that control respiration

This situation only rarely occurs from alcohol intoxication: usually, a drinker will pass out before he or she consumes enough alcohol to suppress respiration completely. With other depressants, it is relatively easy to take a fatal overdose.
Another major danger with CNS Depressants occurs when they are combined with alcohol.

Clarification: the combination of alcohol and certain other CNS Depressants may produce an effect greater than the sum of the effects of the two drugs independently. There is at least an additive effect when alcohol and another depressant are taken together.

With many CNS Depressants, there may be more than an additive effect. Coroners have reported a number of cases in which neither the alcohol level nor the depressant level independently would have been close to a fatal dose.

It is not possible to predict how great of an effect will occur when alcohol is mixed with another depressant.

However, it is clear that the combination is always risky.
E. Expected Results of the Evaluation

Observable Evidence of Impairment

Horizontal Gaze Nystagmus will be present with subjects under the influence of CNS Depressants.

Vertical Gaze Nystagmus may be present, with high doses, of depressants for that individual.

Performance on Modified Romberg Balance, Walk and Turn, One Leg Stand, and Finger to Nose tests will be similar to that of subjects impaired by alcohol.

Vital Signs

• Pulse will be Down $^{(2)}$
• Blood pressure will be Down.
• $^{(2)}$Quaaludes, ETOH and possibly some anti-depressants may elevate.
• Body temperature generally will be in the Normal Range (98.6 plus or minus one degree)

Muscle Tone

• Muscle tone will be Flaccid
Dark Room Examinations

- Pupil sizes will generally be Normal
- \(^{(1)}\) Soma, Quaaludes and possibly some anti-depressants usually dilate pupils.
- Pupillary reaction to light will be Slowed.

**General Indicators**

- Disoriented
- Droopy eyelids (ptosis)
- Drowsiness
- Drunk-like behavior
- Gait ataxia (unsteady, staggering)
- Slow, sluggish reactions
- Thick, slurred speech
- Uncoordinated

**NOTE:**

- With Methaqualone, pulse will be elevated and body tremors will be evident.
- Alcohol, Quaaludes and possibly some anti-depressants elevate the pulse
- Soma, Quaaludes and possibly some anti-depressants usually dilate pupils

**Anti-Depressant Exceptions:**

- As a reminder, some Anti-Depressants may cause elevated pulse rate and pupil dilation.
- Anti-Depressants may cause dry, sore throat, dry mouth, blurred vision, urinary retention, muscle twitching, restlessness, and increased anxiety.
### CNS Depressant Symptomatology Chart

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGN</td>
<td>Present</td>
</tr>
<tr>
<td>VGN</td>
<td>Present (High dose for that individual)</td>
</tr>
<tr>
<td>Lack of Convergence</td>
<td>Present</td>
</tr>
<tr>
<td>Pupil Size</td>
<td>Normal (1)</td>
</tr>
<tr>
<td>Reaction to Light</td>
<td>Slow</td>
</tr>
<tr>
<td>Pulse Rate</td>
<td>Down (2)</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Down</td>
</tr>
<tr>
<td>Temperature</td>
<td>Normal</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Flaccid</td>
</tr>
</tbody>
</table>

(1) Soma, Quaaludes and some anti-depressants usually dilate pupils
(2) Quaaludes, ETOH and some anti-depressants may elevate
F. Classification Exemplar
TOPICS FOR STUDY

1. Name the six major subcategories of CNS Depressants.

2. Name the four groups of Depressants based on onset and duration time factors.

3. To which subcategory of Depressants does Thorazine belong? To which subcategory does Chloral Hydrate belong? To which subcategory does Xanax belong?

4. Name a CNS Depressant that usually causes the pupils to dilate.

5. What is the generic name for the drug that has the trade name “Prozac”?

6. What is a trade name for the generic drug "Alprazolam"?

7. What is the name of the subcategory of CNS Depressants that is also known as the "Minor Tranquilizers"?
**DRUG INFLUENCE EVALUATION**

**Evaluator:**
Officer Bradley Johnson, Manheim PD
Trooper Craig Johnson, PA State Police

**Recorded by:**
Trooper Craig Johnson, PA State Police
Officer Bradley Johnson, Manheim PD

**Date Examined:**
08/06/14 00:15 Harrisburg SP Barracks

**Arrestee Name:** Ludes, Lucy

**Officer's Signature:**

---

**Modified Romberg Balance**

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Angle of Onset</th>
<th>Steps taken</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 60 / 0202</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>2. 58 / 0218</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. 58 / 0240</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Walk and Turn Test**

- Took two extra steps on second nine steps.
- Couldn't keep balance.
- Starts too soon.
- Stops walking.
- Misses heel-toe.
- Steps off line.
- Raises arms.
- Actual steps taken: 9
- 1st Nine: 8
- 2nd Nine: 11

**Internal clock:**

- Estimated as 30 seconds

**Finger to Nose**

- Staggered.
- Took five steps.
- No test done (explain)

**Blood pressure:**
110 / 70

**Temperature:**
98.2 °

**What drugs or medications have you been using?**
- Some pills from my brother.
- A couple of pills

**Breath odor:**
- Alcohol
- Clear
- Nasal area: Clear
- Oral cavity: Clear
- Reaction to light: Slow
- Pupillary unrest: Yes
- Rebound dilation: No

**Type of footwear:**
- Loafers

**DRE #:**
22607

**Rolling Log #:**
14-08-155

**Case #:**
14-445788

**Session IX - #1**

**Officer's signature:**

---

**Opinion of Evaluator:**
- Not Impaired
- Alcohol
- CNS Stimulant
- Dissociative Anesthetic
- Dissociative Anesthetic
- Inhaled
- Medical
- CNS Depressant
- Hallucinogen
- Narcotic Analgesic
- Cannabis
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Ludes, Lucy

1. LOCATION: The evaluation was conducted at the Harrisburg State Police Barracks.

2. WITNESSES: Trooper Johnson of the PA State Police recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was notified that Trooper Cichra had arrested a driver for DUI and was requesting a drug evaluation. I contacted Trooper Cichra at the Harrisburg SP Barracks where it was determined that the suspect had been observed driving at 40 MPH on I-283. When contacted, the suspect was disoriented. She appeared to be drunk, but no alcoholic beverage was detected on her breath. She had six clues of HGN, and was unable to perform SFST’s as directed, and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the Interview Room. She was quiet, withdrawn, and slow to respond to questions. When she tried to walk, she was unstable, and several times used the wall to steady herself.

6. MEDICAL PROBLEMS AND TREATMENT: None observed or reported.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect exhibited a 2" front to back and side to side sway. She estimated 30 seconds in 46 seconds. Walk and Turn: The suspect lost her balance during the instructions, started too soon, stepped off the line twice, missed heel to toe, and raised her arms for balance. On the turn, she staggered, and took five steps to return back down the line. One Leg Stand: The suspect swayed, raised her arms for balance, hopped, and put her foot down. Finger to Nose: Suspect missed the tip of her nose on five of the six attempts.

8. CLINICAL INDICATORS: The suspect exhibited six clues of HGN and had a Lack of Convergence. Two of her pulse rates were below the DRE average range. Her Systolic blood pressure was also below the DRE average range.

9. SIGNS OF INGESTION: None were evident.

10. SUSPECT’S STATEMENTS: Suspect admitted taking some medicine to help her sleep that her brother gave her a couple of hours before driving. She did not know the name of the medicine and stated that it made her very sleepy.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a CNS Depressant and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:
**DRUG INFLUENCE EVALUATION**

**Evaluator:** Officer Russ Kenney  
**Milford PD**

**Reviewer/Witness:** Sgt Wesley Slought  
**Ohio State Patrol**

**Arresting Name (Last, First, Middle):** Downers, Dudley R.

**Date of Birth:** 04/02/86

**DRE #:** 9296

**Rolling Log #:** 14-03-55

**Case #:** 14-2235

**Session IX - #2**

**Crash:** None

**Fatality:** None

**Property:** None

**Arresting Officer (Name, ID):** Trooper Christopher Ellison #22367

**Agency:** Ohio State Patrol

**Date Examined:** 03/16/14

**Time / Location:** 2130 Milford PD

**Results:**
- **Breath Odor:** Normal
- **Eye Reflexes:** Normal
- **Corrective Lenses:** None
- **Coordination:** Cooperative, Carefree
- **Speech:** Slurred
- **Pupillary Reaction:** Normal
- **Vertical Nystagmus:** Able to follow stimulus
- **Pupil Size:** Equal
- **Reaction to Light:** Normal
- **Corrective Lenses:** None
- **Eye Refraction:** Normal
- **Face:** Normal
- **Attitude:** Cooperative
- **Facial Expression:** Cooperative
- **Pupil Size:** Equal
- **Reaction to Light:** Normal

**Results:**
- **Blood Test:** Refused
- **Chemical Test:** Refused
- **Urine Test:** Refused

**Date:** 04/02/86

**Time:** 03:16

**Location:** Milford PD

**Chemical Test:** Refused

**Urine Test:** Refused

**Blood Test:** Refused

**Date of Arrest:** 03/16/14

**Time of Arrest:** 20:25

**Time DRE was notified:** 20:55

**Evaluation Start Time:** 21:30

**Evaluation Completion Time:** 22:30

**Precinct/Station:** Milford

**Opinion of Evaluator:** Not Impaired

**Drug Impairment:**
- **Alcohol:** No
- **CNS Stimulant:** No
- **Dissociative Anesthetic:** No
- **Ihalant:** No
- **Medicinal CNS Depressant:** No
- **Heroin:** No
- **Hallucinogen:** No
- **Narcotic Analgesic:** No
- **Cannabis:** No

**What has been eaten today?**
- Ham Sandwich, Chips

**When?** 7 pm

**What has been drinking?**
- Water

**How much?** N/A

**When did you last sleep?**
- About 7 hours

**How long?**
- N/A

**Are you under the care of a doctor or dentist?**
- Yes

**What drugs or medications have you been using?**
- Medicine to help me sleep

**How much?**
- 1 pill

**At work?**
- About 10 pm

**Where were the drugs used?**
- At work

**Date / Time of Arrest:** 03/10/14 20:20

**Time DRE was notified:** 20:55

**Evaluation Start Time:** 21:30

**Evaluation Completion Time:** 22:30

**Precinct/Station:** Milford

**Officer's Signature:**

**Opinion of Evaluator:**
- Not Impaired
- Alcohol
- CNS Stimulant
- Dissociative Anesthetic
- Inhalant
- Medical
- CNS Depressant
- Hallucinogen
- Narcotic Analgesic
- Cannabis

**Reviewed/approved by / date:** Jan 26, 2015
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Downers, Dudley

1. LOCATION: The evaluation took place at the Milford PD Interview Room.

2. WITNESSES: Sgt. Wesley Stought of the OH SP witnessed and recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was a 0.00%

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to conduct a drug evaluation for Trooper Ellison of the Ohio SP. When I contacted Trooper Ellison, he advised that he had observed the suspect driving under the speed limit on I-50, and was also unable to maintain a single lane of travel. According to Trooper Ellison, the suspect appeared to intoxicated, but no alcoholic beverage was detected on his breath. The suspect exhibited six clues of HGN, had difficulty performing the SFST’s, and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the MPD Interview Room. He was swaying noticeably, had thick, slurred speech, and was slow to respond to questions. When he walked, he was unstable, and used furniture to steady himself.

6. MEDICAL PROBLEMS AND TREATMENT: The suspect stated he was not under the care of a doctor and had no medical conditions that he was aware of. He said he was “just tired.”

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 3” front to back, and approximately 2” side to side. He estimated 30 seconds in 38 seconds. Walk and Turn: The suspect lost his balance twice during the instructions stage, missed touching heel to toe four times, stepped off the line three times, raised his arms for balance, and lost his balance while turning. One Leg Stand: Suspect swayed, used his arms for balance, and put his foot down once while standing on the left foot and twice while standing on the right foot. Finger to Nose: The suspect missed the tip of his nose on each of the six attempts. He had slow hand and arm movements as he attempted to touch his nose.

8. CLINICAL INDICATORS: The suspect exhibited six clues of HGN with an angle of onset at approximately 35 degrees. He also had a Lack of Convergence. One of his pulse rates was below the DRE average range. His blood pressure was also below the DRE average ranges.

9. SIGNS OF INGESTION: None observed.

10. SUSPECT’S STATEMENTS: The suspect admitted taking a “little blue pill” when he left work to help him sleep when he got home.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a CNS Depressant and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:
Modified Romberg Balance:
3" 3" 0" 0"

Walk and Turn Test:
M M M

Swayed forward on each attempt.

Blood pressure: 106 / 68
Temperature: 98.0 ○

Muscle tone: □ Normal □ Flaccid □ Rigid

Comments: Nothing observed.

What drugs or medications have you been using?
"Just some Xanax" "One or two. Can't remember."

How much?
6 pm

Time of use?
McDonald's

Where were the drugs used? (Location)

Date of arrest: 09/06/14 1820
Time DRE was notified: 1845
Evaluation start time: 1910
Evaluation completion time: 2020

Officer's signature:

Opinion of Evaluator:
□ Not Impaired □ Alcohol □ CNS Stimulant
□ Medical □ CNS Depressant □ Dissociative Anesthetic
□ None □ CNS Depressant □ Inhaled
□ Yes □ Yes □ NO
□ No □ Alcohol □ Narcotic Analgesic
□ Yes □ Cannabis

Rev 07/22
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Flynn, Mickey

1. **LOCATION:** The evaluation took place at the West Sacramento CHP Office.

2. **WITNESSES:** Officer Gary Martens of the CHP DRE Unit recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was a 0.00%

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was requested to conduct a drug evaluation for Officer Morgan of the CHP. I contacted Officer Morgan at the West Sacramento CHP Office where it was determined that she had located the suspect slumped over the steering wheel of a vehicle stopped partially in the SB lane of SR 49. She determined that the suspect was the driver of the vehicle and that he was possibly impaired. Officer Morgan administered SFSTs, which the suspect was unable to perform as directed, and was arrested for DUI. The suspect had six clues of HGN.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in a slumped over in a chair in the interview room. The suspect was mumbling, and had thick, slurred speech. He was slow to respond to questions, and had a drunk-like appearance.

6. **MEDICAL PROBLEMS AND TREATMENT:** The suspect stated he was under the care of a doctor for stress, and he was taking medication that made him very tired at times.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: The suspect swayed approximately 3" front to back, and he estimated 30 seconds in 50 seconds. Walk and Turn: The suspect lost his balance twice during the instructions stage, missed heel to toe three times, stepped off the line five times, raised his arms for balance, and staggered to the right on the turn. One Leg Stand: Suspect swayed, used his arms for balance, and put his foot down once while standing on the left foot and once while standing on the right foot. Finger to Nose: The suspect missed the tip of his nose on five of the six attempts, and swayed forward on each attempt.

8. **CLINICAL INDICATORS:** The suspect exhibited six clues of HGN with an angle of onset at approximately 40 degrees. A Lack of Convergence was present. His pulse rates and blood pressure were below the DRE average ranges.

9. **SIGNS OF INGESTION:** None observed.

10. **SUSPECT’S STATEMENTS:** The suspect admitted taking Xanax several times during the day for stress, but could not remember how many he took.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of a CNS Depressant and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A blood sample was collected from the suspect.

13. **MISCELLANEOUS:**
Participant Manual

Drug Recognition Expert Course

Session 10
Central Nervous System Stimulants
Learning Objectives

• Explain a brief history of the CNS Stimulant category of drugs
• Identify common drug names and terms associated with this category
• Identify common methods of administration for this category
• Describe the symptoms, observable signs and other effects associated with this category

Upon successfully completing this session the participant will be able to:

• Explain a brief history of the CNS Stimulant category of drugs.
• Identify common drug names and terms associated with this category.
• Identify common methods of administration for this category.
• Describe the symptoms, observable signs and other effects associated with this category.
• Describe typical time parameters, i.e. onset and duration of effects, associated with this category.
• List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of this category of drugs.
• Correctly answer the “topics for study” questions at the end of this session.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES
A. Overview of the Category ..............................................................Instructor Led Presentations
B. Possible Effects ................................................................. Review of the Drug Evaluation and Classification Exemplars
C. Onset and Duration Effects.................................and Classification Exemplars
D. Overdose Signs and Symptoms........................ Reading Assignments
E. Expected Results of the Evaluation..............................Video Presentations
F. Classification Exemplar .................................................. Slide Presentations

Revised: Drug Recognition Expert Course Session 10
07/2015 Central Nervous System Stimulants Page 1 of 22
A. Overview of the Category

CNS Stimulants speed up the operation of the Central Nervous System.

- “Speed Up” does not mean “improve.”
- The “speeding up” results in increased heartbeat, pulse, respiration, blood pressure, and temperature.

All of these effects can lead to physical harm to the stimulant user.

- However, Robert Louis Stevenson wrote “The Strange Case of Dr. Jekyll and Mr. Hyde” while under the influence of Cocaine. He wrote sixty thousand words in six days.

The “speeding up” also produces nervousness, irritability, and an inability to concentrate or think clearly.

These psychological effects can lead to unpredictable and bizarre behavior by the stimulant user.
Subcategories of CNS Stimulants

There are three major subcategories of Central Nervous System Stimulants.

Cocaine

The Amphetamines

Examples:

- Methamphetamine
- Amphetamine Sulfate
- Desoxyn
  - Also includes (d-methamphetamine) (d-desoxyephedrine) and Methedrine.
  - Desoxyn was first developed in 1919 and has been used clinically since 1930. Mainly used for the treatment of obesity, narcolepsy and attention disorder.
Others

There are many “other” CNS Stimulants (i.e., non-Cocaine and non-Amphetamines); the ones listed on the visual are only a few of those.

- **Ritalin** (methylphenidate hydrochloride)
  - Also brand names of Concerta, Daytrana. Used in the treatment of depression, narcolepsy and ADD (Attention Deficit Disorder)

- **Ephedrine** – (Primatene, Quadrinal)
  - Can be found in some naturally-occurring plants such as the Chinese herb ma huang. Used as a nasal decongestant and bronchodilator. Contained in numerous OTC supplements and energy products

- **Caffeine**
  - Contained in coffee and numerous energy drinks. Some “Monster drinks” contain as much as 240 milligrams of caffeine. Can be fatal at about 10 grams.
Cocaine

Coca plant: Scientific name “Erythroxylon Coca.”

Cocaine derives from the coca plant.

- The plant is native to South America.
- Cocaine is made from the leaves of the coca plant.
- Archaeological evidence indicates that natives of Peru chewed coca leaves 5,000 years ago.
- Sigmund Freud personally experimented with Cocaine for approximately 3 years.
- Small quantities of Cocaine originally were included in the formula of Coca Cola.
- Use of Cocaine in products as Coca Cola was outlawed by the Pure Food and Drug Law of 1906.
**Amphetamines**

Amphetamines were first synthesized near the end of the 19th Century. The first use of Amphetamines for medical purposes began in the 1920’s. Initial medical application was to treat colds.

- Amphetamines cause the nasal membranes to shrink.
- This gives temporary relief from stuffy nasal passages.

Amphetamines were prescribed for the treatment of narcolepsy and ADHD (attention deficit hyperactivity disorder).

Amphetamine use grew rapidly when amphetamines were distributed to soldiers during World War II.
Present day medical purposes for amphetamines include:

- **Control appetite.** Many over the counter appetite control products contain CNS Stimulants as their active ingredient.
- **Control symptoms of narcolepsy.** Narcolepsy is an extremely rare disorder that causes the individual to fall asleep compulsively, often several hundred times per day.
- **Control certain hyperactive behavioral disorders.** Example: Ritalin is commonly prescribed for children diagnosed with ADD or similar disorders.
- **Relieve or prevent fatigue to allow persons to perform essential tasks of long duration.** The U.S. Air Force previously gave pilots amphetamines to keep them alert on long flights. Amphetamines have also had other short term military applications.
- **Treat mild depression.**
- **Antagonize the effects of depressant drugs.**
- **Prevent and treat surgical shock.**
- **Maintain blood pressure during surgery.**
- **Treat Parkinson’s Disease.**
- **Enhance the action of certain analgesic (pain killer) drugs.**

Numerous pharmaceutical companies manufacture Amphetamines for these purposes.
Commonly Prescribed Pharmaceutical Amphetamines

- **Dexedrine**
  Dextroamphetamine Sulfate
- **Adderall**
  Dextroamphetamine and Amphetamine
- **Benzedrine**
  Amphetamine Sulfate
- **Desoxyn**
  Methamphetamine Hydrochloride

Examples of common pharmaceutical Amphetamines:

- Dexedrine (dextroamphetamine sulfate) used to treat narcolepsy and hyperkinetic behavior, and for weight control. (Street names “Dexies”; “Hearts”)
- Adderall (Combination of Dextroamphetamine and Amphetamine Sulfate) It is used for the treatment of attention deficit hyperactivity disorder (ADHD) and narcolepsy.
- Benzedrine (Amphetamine Sulfate) used to treat narcolepsy, hyperkinetic behavior and weight problems. (Street names “Bennies”; “Whites”; “Cartwheels”)
- Desoxyn (Methamphetamine Hydrochloride, also known as Desoxyephedrine) used in weight reduction.
Large quantities of Amphetamines are also illegally manufactured in this country. The most commonly abused illicit Amphetamine is Methamphetamine. Methamphetamine Hydrochloride is a white to light brown crystalline powder, or clear chunky crystals resembling ice. Methamphetamine base is a liquid. The majority of street Methamphetamine is produced in Clandestine laboratories. Medicinally, forms of Methamphetamine can be used in the treatment of:

- Narcolepsy
- Attention Deficit Disorder (ADD)
- Attention Deficit Hyperactivity Disorder (ADHD)

Methamphetamine is also known as Methedrine or Methamphetamine Hydrochloride. Its’ more common street names are “speed”; “crank”; “ice”; “crystal”; “meth”; and “water.”
### Other CNS Stimulants

(Besides Cocaine or Amphetamines)

- **Ritalin**
  - Methylphenidate Hydrochloride
- **Ephedrine**
- **Cathine and Cathinone**
- **Methcathinone**

---

**Other CNS Stimulants**

There are some other CNS Stimulants, apart from Cocaine or the Amphetamines.

**Ritalin**

Ritalin is a manufactured, non-Amphetamine CNS Stimulant:

- Generic name Methylphenidate Hydrochloride
- Used to treat mild depression, hyperkinetic behavior, narcolepsy and drug induced lethargy produced by CNS Depressants.
- Has many of the basic clinical effects of Amphetamine.

**Ephedrine** is a licitly manufactured stimulant primarily used as a nasal decongestant and bronchodilator. It can also be found in herbal preparations and numerous over-the-counter (OTC) substances.

**Cathine and Cathinone** are the two psychoactive chemicals derived from the Khat plant. It originates from the sub-Sahara regions of Africa. Also known as “cat.”

**Methcathinone** is illicitly manufactured from common household chemicals. Effects are very similar to Methamphetamine.
Methods of Ingestion of CNS Stimulants

There are a variety of ways in which the different CNS Stimulants may be ingested. Cocaine is commonly insufflated (snorted), smoked, injected and taken orally.

In order to be smoked, a pure form of Cocaine is required.

- Much of the Cocaine sold in this country is mixed with other materials, or chemically bonded to other elements.
- Various chemical processes can be used to “free” the Cocaine from other elements and impurities.
- One such process produces pure Cocaine in the form of small chunks.
- These chunks are known as “Crack” or “Rock Cocaine.”
- Legally-manufactured Amphetamines are taken orally, in the form of tablets, capsules and liquid elixirs.
### Methods of Ingesting Stimulants

- **Methamphetamine**
  - Injection
  - Orally
  - Snorting
  - Smoking

- **Other Amphetamines**
  - Orally
    - (tablets, capsules, etc.)

- Illicitly manufactured Methamphetamine most commonly is injected or smoked but sometimes may be snorted or taken orally.

- The smokable forms of Methamphetamine are known as “Crystal Meth” or “Ice.” They contain the same active chemical compound as powdered Methamphetamine, but undergo a re-crystallization process in which some impurities are removed.

- Amphetamine Sulfate usually is produced in tablet form (called “mini bennies”) and is taken orally.
B. Possible Effects

Cocaine, Amphetamines and most stimulants produce euphoria, a feeling or state of intense excitement and happiness.

- A feeling of super strength and absolute self-confidence may also be present.
- With Cocaine, but not with Amphetamines, there is an anesthetic effect, and the dulling of pain may contribute to the euphoria.

CNS Stimulant users tend to become hyperactive, indicated by nervousness, extreme talkativeness, an inability to sit still, and users may grind their teeth (which is called Bruxism).

CNS Stimulants tend to release inhibitions, allowing users to commit acts that they normally would avoid.

CNS Stimulant users misperceive time and distance.

Example: to the subject, time seems to be speeded up, so that 2 hours may seem like two minutes.

Persons under the influence of CNS Stimulants become easily confused, and lose the ability to concentrate or to think clearly for any length of time.
C. Onset and Duration of Effects

The onset and duration of effects are quite different for Cocaine as compared to Amphetamines.

- Generally speaking, Cocaine’s effects are much briefer than are Amphetamine’s.
- The time parameters of Cocaine vary with the method of ingestion.

**Cocaine: Smoked**

When Cocaine is smoked, or “freebased,” the drug goes immediately to the lungs, and is absorbed into the bloodstream very rapidly.

- The smoker begins to feel the effects of the Cocaine virtually immediately.
- The “rush” or euphoria is reported to be very intense.
- However, the euphoric effect only last 5 – 10 minutes after the Cocaine is smoked.

**Cocaine: Injected**

When Cocaine is injected, the drug is passed directly to the bloodstream, where it is carried swiftly to the brain.

- The effects are felt within seconds.
- The onset of effects is very intense.
- **Injection sites will be discussed in Narcotic Analgesics**
- The effects generally last 5 - 15 minutes.

*Source: “Disposition of Toxic Drugs and Chemicals in Man”, 9th Edition, R. Baselt*
Cocaine: Snorted

When Cocaine is snorted (insufflated), the onset of effects is not quite as rapid as with smoking or injecting.

- The user typically feels the onset of effects within 30 seconds after snorting the drug.
- Although the “rush” occurs, it is not quite as intense as it is when the Cocaine is smoked or injected.
- The effects from snorting usually last from 30 – 90 minutes.

Cocaine: Oral Ingestion

- Oral ingestion of Cocaine usually is the least preferred method.
- The effects of Cocaine taken orally may last from 45 – 120 minutes.
- The user generally does not begin to feel the effects for 3 – 5 minutes.
- The effects are not as intense as they are with other methods of ingestion.
- However, the effects may last 15 – 30 minutes longer than with other methods.

With all methods of ingestion, the duration of Cocaine’s effects tend to be briefer than the effects of most other drugs.

- As the effects wear off, it becomes very difficult to observe evidence of impairment.
- If the subject is not evaluated by a DRE fairly soon after the subject has been apprehended, the DRE may not uncover evidence of the CNS Stimulant.
Methamphetamine Time Factors

- Effects are felt within seconds
- “Rush” is very intense for 5-30 seconds
- Effects can last up to 12 hours

Methamphetamine: Injected

When Methamphetamine is injected, the initial effects are very similar to the injection of Cocaine.

- The user begins to feel the effects within a few seconds.
- The “rush” is very intense, and lasts at a high level of intensity for 5 – 30 seconds.
- Unlike Cocaine, Methamphetamine’s effects are longer and may last up to 12 hours after injection.

Methamphetamine: Smoked

When Methamphetamine is smoked, the rush is very intense.
The user stays “high” for 4 – 8 hours with residual effects lasting up to 12 hours.

Methamphetamine: Snorted and Orally

When taken orally the onset of effects is delayed, the rush is much less intense and the effects last longer.
D. Overdose Signs and Symptoms

Overdose of Cocaine or Amphetamines can cause the pleasurable effects to turn into panic and often violent behavior. If the overdose is caused by Cocaine, it is commonly referred to as Cocaine Psychosis or Cocaine Delirium.

- Subject may suffer convulsions and faint or pass into a coma.
- Heartbeat (pulse) will increase, possibly dramatically.
- Hallucinations may occur.

Example: The feeling that bugs are crawling under the skin is also known as “Coke Bugs.” The medical term for this condition is formication.

- Death can occur from sudden respiratory failure, or from heart arrhythmia, leading to cardiac arrest.
- Another danger is that subjects may attempt to treat CNS Stimulant overdoses with Barbiturates, possibly leading to overdose of CNS Depressants.
Evaluation of Subjects Under the Influence of CNS Stimulants

• HGN or VGN - None
• Lack of Convergence - None
• Impaired performance should be evident on Modified Romberg Balance, Walk and Turn, One Leg Stand and Finger to Nose

E. Expected Results of the Evaluation

Observable Evidence of Impairment

• Horizontal Gaze Nystagmus will not be present with subjects under the influence of CNS Stimulants.
• Vertical Gaze Nystagmus will not be present.
• Lack of Convergence will not be evident.
• Performance on Modified Romberg Balance should be impaired.
• Performance on Walk and Turn may be impaired due to the subject’s hyperactivity and inability to concentrate. Example: subject may start too soon on the Walk and Turn, and may tend to walk fast, thus losing balance or missing heel-to-toe.
• Performance on the One Leg Stand may be impaired due to the subject’s hyperactivity. Example: subject may also count very rapidly on the One Leg Stand test.
• Performance on the Finger to Nose test should be impaired. His or her finger movements may be abrupt, jerky and inaccurate.
Evaluation of Subjects Under the Influence of CNS Stimulants

Vital Signs:
- Pulse - Up
- Blood pressure - Up
- Body temperature - Up

Muscle Tone - Rigid

Dark Room Examinations:
- Pupils - Dilated (Mydriasis)
- Pupillary reaction to light - Slow

Vital Signs

- Pulse generally will be increased.
- Blood pressure will generally be elevated.
- Body temperature generally will be elevated.

Muscle Tone

- Muscle tone will be Rigid.

Dark Room Examinations

- Pupils generally will be dilated.
- The technical term for “dilated pupils” is Mydriasis.
- Pupil reaction to light generally will be slow.
- Rebound Dilation may be observed.
General Indicators

- Anxiety
- Body tremors
- Bruxism (grinding teeth)
- Dry mouth
- Euphoria
- Excited
- Exaggerated reflexes
- Eyelid and leg tremors
- Increased alertness
- Insomnia
- Irritability
- Restlessness
- Rigid muscle tone
- Talkative
- Redness to nasal area
- Runny nose

CNS Stimulant Symptomatology Chart

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<tr>
<th></th>
<th>HGN</th>
<th>VGN</th>
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<tr>
<td>Lack of Convergence</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Pupil Size</td>
<td>Dilated</td>
<td></td>
</tr>
<tr>
<td>Reaction to Light</td>
<td>Slow</td>
<td></td>
</tr>
<tr>
<td>Pulse Rate</td>
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<tr>
<td>Blood Pressure</td>
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<tr>
<td>Temperature</td>
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<td></td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Rigid</td>
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</tr>
</tbody>
</table>

Revised: Drug Recognition Expert Course
07/2015
Central Nervous System Stimulants
Page 20 of 22
F. Drug Evaluation and Classification Exemplar Demonstrations

...
TOPICS FOR STUDY

1. Why is it sometimes difficult for a DRE to obtain evidence of CNS Stimulant influence when examining a cocaine user?

2. What kinds of illicitly manufactured Amphetamines are most commonly abused?

3. Name two CNS Stimulants other than Cocaine or the Amphetamine compounds.

4. How do CNS Stimulants usually affect the blood pressure and pulse rate?

5. True or False: A person under the influence of a CNS Stimulant alone usually will not exhibit Horizontal Gaze Nystagmus?

6. What is “bruxism”?
### DRUG INFLUENCE EVALUATION

**Evaluator:** Officer Larry Curtis  
**Recorder/Witness:** Cpl. Mark Morton  
**Date Examined:** 02/08/14  
**Recorder/Officer:** Pulaski Co. Jail  
**Given by:** TFC Jeff Hust  
**Witness:** Arkansas State Police  
**Date of Birth:** 07/10/87  
**Race:** W  
**Officer's Signature:**  
**Arresting Officer (Name, ID#):**  
**Arresting Agency:** Arkansas State Police  
**Date of Arrest:** 02/08/14  
**Time of arrest:** 2140  
**Location:** 2235  
**Number of Tests:**  
**Evaluator Opinion:** Not Impaired  
**Alcohol:**  
**CNS Stimulant:**  
**Dissociative Anesthetic:**  
**Hallucinogen:**  
**Narcotic Analgesic:**  
**Inhalant:**  
**Time of DRE was notified:** 2215  
**Evaluation start time:** 2230  
**Evaluation completion time:** 2330  
**Witness:**  
**Precinct/Station:**  
**Date/Time of arrest:** 02/08/14  
**Time DRE was notified:** 2215  
**Evaluation start time:** 2230  
**Evaluation completion time:** 2330  
**Officer's Signature:**  
**Test or tests refused:**  
**Chemical Test:** Yes  
**Test or tests refused:**  
**Breath Odor:**  
**Time of last drink:** N/A  
**Test Results:** 0.00  
**Test Results:** 45752  
**Test or tests refused:**  
**Urine:**  
**Blood:**  

#### Miranda Warning Given
- Date: 02/08/14  
- Time: 2235  
- Location: Pulaski Co. Jail  
- Officer: Larry Curtis AR HP  
- Cpl. Mark Morton AR State Police  

#### Bruxism
- Opinions: Excited, Talkative
- Breath Odor: Rancid
- Quick, Slurred at times
- Quick movements.

#### Breath Odor
- Room Light: 2.5 - 5.0
- Darkness: 5.0 - 8.5
- Direct: 2.0 - 4.5

#### Muscles
- Normal
- Flaccid
- Rigid

#### Pulse and Time
<table>
<thead>
<tr>
<th>Time</th>
<th>Value</th>
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</thead>
<tbody>
<tr>
<td>1.</td>
<td>102 / 2250</td>
</tr>
<tr>
<td>2.</td>
<td>104 / 2302</td>
</tr>
<tr>
<td>3.</td>
<td>102 / 2315</td>
</tr>
</tbody>
</table>

#### Modified Romberg Balance
- Walked quickly.
- Sways while balancing
- Uses arms to balance
- Hops
- Puts foot down

#### Internal Clock
- 3" 3" 0" 0"
- Estimated as 30 seconds

#### Describe Turn
- Quick, spinning turn
- Cannot do test (explain)

#### Type of Footwear
- Slip-on boots

#### Unit Test
- Right Count: 40
- Left Count: 42

#### Finger to Nose
- Room Light: 2.5 - 5.0
- Darkness: 5.0 - 8.5
- Direct: 2.0 - 4.5

#### Pupil Size
- Left: Normal
- Right: Normal

#### Coordination
- Poor, Quick, Unstable
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Rocke, Crystal

1. LOCATION: The evaluation was conducted at the Pulaski County Jail.

2. WITNESSES: Corporal Mark Morton of the Arkansas SP witnessed the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to conduct a drug evaluation by Trooper Hust. I contacted him at the County Jail where it was determined he had stopped the suspect for driving 100 mph and for driving without headlights on I-30. According to Tpr. Hust, the suspect was excited, animated, very talkative, and restless. She performed poorly on the SFST’s, and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room with Trooper Hust. She was moving back in forth in her chair and could not remain still. Her speech was fast and slurred. Her reflexes were quick and exaggerated.

6. MEDICAL PROBLEMS AND TREATMENT: None observed and none stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 3” front to back, and estimated 30 seconds in 12 seconds. Walk and Turn: Suspect lost her balance twice during the instructions, started too soon twice, stopped walking once to regain her balance, raised her arms for balance four times, made an abrupt spinning turn, and missed heel to toe twice on the second nine steps. One Leg Stand: Suspect swayed, used her arms to balance, and hopped. She put her foot down twice when standing on the left foot, and once while standing on the right foot. Finger to Nose: Suspect missed the tip of her nose on four of the six attempts, and had very quick hand movements.

8. CLINICAL INDICATORS: Suspect’s pulse, blood pressure, and body temperature were elevated, and all were above the DRE average ranges. Her pupils were dilated in all three lighting levels, and they reacted slowly to light. HGN, VGN and LOC were not present.

9. SIGNS OF INGESTION: White powder residue was located in the suspect’s left nostril.

10. SUSPECT’S STATEMENTS: The suspect denied using any drugs.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a CNS Stimulant and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS:
DRUG INFLUENCE EVALUATION
12269

Officer Kirk McDowell, Oklahoma City PD

Recorder I Witness

Crash: IB] None
D Fatal Fi lniury
Date of B irth

Officer C. Vinson, Norman PD

!Arrestees Name (Last, t-1rst, 1v11uu1e)

Tweaker, Ira

2315

I

08/24/78

Breath Results:
Results:
0.00

Date Examined I Time I Location

10/02/14

I

DRE#

Evaluator

Oklahoma Co. Jail

Komng Log#

t;ase#

14-775220

14-08-022

D Pro pertv
�ex

M

I

Arresting Officer (Name, ID#)

Officer J. Murphy

#18122

Arres_tmg vrncer Agenc
o

Race

w

Oklahoma City P

Test Refused
Instrument #

I

Session X - #2

u

IChemical Test

I

Test or tests refused

14330

Blood

Urine LJ

D

0

Time of last drink?
Miranda Warning Given
What have you been drinking? How much
IBJ Yes IW hathaveyoueaten today? When?
N/A
Given by:
Water and juice
Couple bottles
Officer Murphy
D No
Cold cereal 11 am
Are you sick or injured?
Are you diabetic or epileptic?
\Nhen did you last sleep? How long?
Time now I Actual
I
O Yes @No
Two days aqo I 5-6 hours
D Yes IBJ No
"About 1 am" / 2318
Are you under the care of a doctor or dentist?
Do you have any physical defects?
Do you take insulin?
D Yes 0 No
D Yes 0 No
D Yes 0 N o
Coordination:
Are you taking any medication or drugs?
Attitude:
f
Staggering, Poor
O Yes � No
Cooperative, Restless
race:
Breath Odor:
Speech:
I

I

I

Talkative, Rapid

Corrective Lenses:

D Uneaual (exolain)
Pulse and time
HGN

J

1. __!Q§_

2322
2334
2345

2. _.1_Q§_ I
3. --108 I

None

Maximum Deviation

None
None

None
None

Angle of Onset

:r ¥

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• ',,,

I

" I � "' '

estimated as 30 seconds

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PUPIL SIZE

A

J .J1Q__

Officer's Signature:

2220

Opinion of Evaluator:

,/

Raises arms

I

9.0
9.0

Temperature

�

99.8 0

==

I NIA

Time DRE was notified:

2245

O Not Impaired
OMedical

Nothing observed.

I

How much?

I
I

Evaluation start time:

2315

v,'"_

0Alcohol
OCNS Depressant

I

Time of use?

N/A

Reviewed/approved by I date:
.CNS Stimulant
OHallucinogen

'1\'l el

42

R

Nasal area:

Redness, Sores, Bloody

Oral cavity:

Clear

!Reaction to Light

==c

Slow
LEFT ARM

�

�

====
I

N/A

Evaluation completion time:

2355

Right Count

Type of footwear:
Runninq shoes

6.0
6.0

�

One Leg Stand

Quick movements. Fast count.

Pupillary Unrest
D Yes �No

RIGHT ARM

D Droopy

D D Hops
D [] Puts foot down

Direct
2.0-4.5

Darkness
5.0-8.5

� Nom1al

[] Sways while balancing
[] [] Uses arms to balance

9

Cannot do test (explain)
N/A

Room light
2.5-5.0

L

[]

,/,/,/

9

�

D

"I don't use druas anvmore"
Date I Time of arrest

,I
,/,!

Actual steps taken

D

3

2nd Nine

Steps off line

Rebound Dilation:
Yes
[BJ No

1

1s t Nine

Eyelids

38

Left eye

,/

Misses heel-toe

Right Eye

Musde tone:
�Rigid
OFtaccid
Normal
Comments:
What drugs or medications have you been using?

10/02/14

Stops walking

6.5
6.5

Quick hand and am1 movements.

148

Right eye

[] Equal D Unequal

Left Count

(3€)

Starts too soon

Left Eye

:_/�

Blood pressure

Convergence

Cannot keep balance

I

uescnoe Turn
Spinninq tum

Finger to Nose
{Draw lines to spots touched)

2

"'

I

Took quick, jerky steps throughout test.

1nterna1 c1olif\.

20

.....

[ 0) I«)

Able to follow stimulus
fBl Yes 0No

Left Eye

None

2" 2"

Bruxism

Right Eye

Vertical Nystagmus
O Yes [BJ No

Lack of Smooth Pursuit

Modified Romberg Balance Walk and Tum Test

3" 3"

I

Tracking:

IBl None D Left D Right

IBl Nonnal D Bloodshot D Watery

[BJ Equal

Pupil Size:

Blindness:

Eyes: LJ Reddened Conjunctiva

D Contacts if so D Hard D Soft

D Glasses

Flushed

Rancid

�None

I

�

VI/here were the drugs used? (Location)
Precinct/Station:

0Dissociative Anesthetic
ONarcotic Analgesic

Olnhalant
ocannabis

RevOl/15


DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Tweeker, Ira

1. LOCATION: The evaluation was conducted at the Oklahoma County Jail.

2. WITNESSES: The evaluation was recorded by Officer Vinson of the OK City PD.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was contacted by Officer Murphy requesting a drug evaluation. After arriving at the County Jail, Officer Murphy reported that he had stopped the suspect for driving 65 mph in a 30 mph zone and for failing to stop at a stop sign. The suspect was very talkative and restless at roadside. He was unable to perform the SFST’s as directed, and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room standing next to Officer Murphy. He was very fidgety and could not stand still. When told to sit down, the suspect would sit for a few seconds, and then quickly get back up.

6. MEDICAL PROBLEMS AND TREATMENT: None observed and none stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 3” front to back and 2” side to side. He estimated the passage of 30 seconds in 20 seconds. Walk & Turn: The suspect started too soon, stepped off the line twice, raised his arms for balance five times, and turned using an abrupt spinning movement. One Leg Stand: Suspect swayed while balancing, used his arms for balance, hopped once when standing on his left foot, and put his foot down once while standing on his right foot. Finger to Nose: Suspect missed the tip of his nose on each attempt. He made quick arm movements, and was grinding his teeth during the test.

8. CLINICAL INDICATORS: The suspect’s pulse, blood pressure and body temperature were above the DRE average ranges. His pupils were dilated in all three lighting conditions.

9. SIGNS OF INGESTION: The suspect’s nostrils were red and bloody.

10. SUSPECT’S STATEMENTS: The suspect denied using drugs. When asked about drug use he would only state, “I used to do Meth, but I don’t use anymore.”

11. DRE'S OPINION: In my opinion, the suspect is under the influence of a CNS Stimulant and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:

Rev. 10/15
# Drug Influence Evaluation

**Evaluator:** Officer Jeramey Peters, Auburn Hills PD

**Arrestee's Name:** Crank, Christy 10/09/85 F

**Recorder:** Officer Wes Evans

**Arresting Officer:** Trooper Troy Meder #18754

**Date Examined:** 09/29/14 0130 Auburn Hills PD

**Chemical Test:** None

---

**Physical Examination:**

- **PUPIL SIZE:**
  - Right Eye: None
  - Left Eye: None

- **Convergence:** Right eye Left eye

- **Corrective Lenses:** None

- **Glasses:** None

- **Contacts:** None

- **Coordination:** Poor, Quick

- **Speech:** Rapid

- **Attitude:** Indifferent

- **Face:** Flushed, Red sores

- **Corrective:** None

- **Rapid:** None

- **Flashed:** None

- **Rigidity:** None

- **Score:** None

- **Unequal (explain):** None

---

**Pulse and Time:**

1. 102 / 0150
2. 98 / 0205
3. 98 / 0218

**Time Now / Actual:** 09/29/14 0130 Auburn Hills PD

**Breath Odor:** None

**Breath Results:** 0.00

**Test Refused:** None

**Chemical Test:** None

**Test or tests refused:** None

---

**Results:**

- **Blood Pressure:** 158 / 96
- **Temperature:** 99.8 °

**Bloodshot:** None

**Drugs or Medications:**

- **Do you take insulin:** Yes

---

**Muscle Tone:**

- **Normal:** None

- **Rigidity:** None

- **Flaccid:** None

**Comments:** None

---

**Vision:**

- **Left Eye:**
  - Right Eye:
  - Left Eye:
  - Convergence:

**Vital Signs:**

- **Blood Pressure:** 158 / 96
- **Temperature:** 99.8 °

**Breath Odor:** None

**Breath Results:** 0.00

**Test Refused:** None

**Chemical Test:** None

**Test or tests refused:** None

---

**Time Now / Actual:** 09/29/14 0130 Auburn Hills PD

**Breath Results:** 0.00

**Test Refused:** None

**Chemical Test:** None

**Test or tests refused:** None

---

**Comment:**

- **Quick hand and arm movements.**

- **Blurred vision:** None

- **Muscle tone:** None

- **Flaccid:** None

- **Rigidity:** None

---

**Reaction to Light:**

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<tr>
<th>Room Light</th>
<th>Darkness</th>
<th>Direct</th>
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<td>2.5 - 5.0</td>
<td>5.0 - 8.5</td>
<td>2.0 - 4.5</td>
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</table>

**Pupillary Response:**

- **Pupillary Uneven:** None

**Reaction to Light:**

- **Normal:** None

- **Redness:** None

**Nasal Area:**

- **Clear:** None

- **Red:** None

**Oral Cavity:**

- **Clear:** None

- **Red:** None

**Red mark on inside of left forearm.**

---

**Officer's Signature:** Jeramey Peters, Auburn Hills PD

---

**Witness:**

- **Witness Crash:** None
- **Neither:** None

**Officer:** Wes Evans

** Arresting Officer:** Trooper Troy Meder #18754

**Date of Birth:** 10/09/85 F

**Sex:** F

**Race:** W

**Property:** None

**At Time of Arrest:**

- **What drugs or medications have you been using:** None
- **How much:** None
- **Where were the drugs used:** None

**Time of Last Drink:**

- **Date:** None
- **Time:** None

**Time of Last Drink:**

- **Date:** None
- **Time:** None

**Time of Arrest:**

- **Date:** None
- **Time:** None

---

**Officer's Signature:** Jeramey Peters, Auburn Hills PD

---

**Opinion of Evaluator:**

- **Not Impaired:** None
- **Alcohol:** None
- **CNS Stimulant:** None
- **Dissociative Anesthetic:** None
- **Inhalant:** None
- **Medical:** None
- **CNS Depressant:** None
- **Hallucinogen:** None
- **Narcotic Analgesic:** None
- **Cannabis:** None

---

**Officer's Signature:** Jeramey Peters, Auburn Hills PD

---

**Date of Incident:** 09/29/14 2415

**Time DRE was notified:** 2450

**Evaluation start time:** 0130

**Evaluation completion time:** 0225

**Precinct/Station:** None

---

**Officer's Signature:** Jeramey Peters, Auburn Hills PD

---

**Opinion of Evaluator:**

- **Not Impaired:** None
- **Alcohol:** None
- **CNS Stimulant:** None
- **Dissociative Anesthetic:** None
- **Inhalant:** None
- **Medical:** None
- **CNS Depressant:** None
- **Hallucinogen:** None
- **Narcotic Analgesic:** None
- **Cannabis:** None

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**Officer's Signature:** Jeramey Peters, Auburn Hills PD

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- **Alcohol:** None
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- **Alcohol:** None
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**Evaluation start time:** 0130

**Evaluation completion time:** 0225

**Precinct/Station:** None

---

**Officer's Signature:** Jeramey Peters, Auburn Hills PD
Suspect: Crank, Christy

1. **LOCATION:** The evaluation was conducted at the Auburn Hills PD Interview Room.

2. **WITNESSES:** Officer Evans of the Grand Blanc Township PD recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was contacted by Trooper Meder requesting a drug evaluation. After contacting Trooper Meder it was determined that he had stopped the suspect for excessive speed and for following other vehicles too closely. He advised that the suspect was very talkative and restless at roadside. She had difficulties performing the SFST’s and was arrested for DUI.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect standing in the interview room with Trooper Meder and Officer Evans. She was moving about and could not stand still. Her speech was quick, and she was very talkative. Her hand and arm movements were exaggerated and quick. She appeared to be grinding her teeth at times.

6. **MEDICAL PROBLEMS AND TREATMENT:** None observed and none stated.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: The suspect swayed approximately 3” front to back and side to side. She estimated the passage of 30 seconds in 18 seconds, and was grinding her teeth during the test. Walk & Turn: The suspect could not keep her balance during the instructions, started too soon twice, missed heel to toe five times, stepped off the line twice, and raised her arms for balance three times. She stopped and then spun around on the turn, nearly falling. One Leg Stand: Suspect swayed while balancing, and used her arms for balance. She put her foot down twice while standing on her left foot, and once while standing on her right foot. Finger to Nose: The suspect missed the tip of her nose on five of the six attempts, and had quick hand and arm movements.

8. **CLINICAL INDICATORS:** The suspect’s pulse, blood pressure, and body temperature were above the DRE average ranges. Her pupils were dilated in all three lighting conditions.

9. **SIGNS OF INGESTION:** A red mark was located on the inside of the suspect’s left arm.

10. **SUSPECT’S STATEMENTS:** She denied using drugs each time she was asked.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of a CNS Stimulant and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A blood sample was collected from the suspect.

13. **MISCELLANEOUS:** Rev. 10/15
Drug Recognition Expert Course

Session 11
Practice: Eye Examinations
Upon successfully completing this session the participant will be able to:

- Conduct examinations of pupil size and reaction to light under both lighted and darkened room conditions.
- Describe the eye examination procedures.
- Document the results of the eye examinations.

CONTENT SEGMENTS .................................................................................. LEARNING ACTIVITIES

A. Procedures for this Session ........................................................... Instructor-Led Presentations
B. Room Light Examinations ........................................................... Participants’ Hands-On Practice
C. Dark Room Examinations ............................................................. Instructor-Led Coaching
D. Session Wrap-Up ............................................................................. Participant-Led Coaching
A. Procedures for this Session

Team Assignments

- Participants will work in three or four member teams.
- Make team assignments.
- At any given time, one member of the team will be engaged in conducting and recording eye examinations of another member.
- The remaining member(s) will help coach and critique the participant who is conducting the examinations.

Team Practice

Participants will take turns serving as test administrator, test subject and coach.

Teams initially will practice under lighted room conditions.

- Check pupil size under normal room light.
- Check reaction to light and pupil size using a penlight in a lighted room.

Teams subsequently will practice under darkened room conditions.

- Check pupil size in near total darkness.
- Check reaction to light and pupil size under direct light.
- Participants will record their estimations using Eye Examinations Data Sheet. There are copies of the Eye Examination Data Sheet in the Participant’s Manual.
B. Room Light Examinations

Pupil Size Estimation

• Pupil size estimation, under room light.
• Pupil reaction and size estimation, under direct light.

Sequence of roles should be as follows:

• Test Administrator
• Test Subject
• Coach
• Test Administrator (continue cycle)
C. Dark Room Examinations

Pupil Size Estimation

- Pupil size estimation, under near total darkness.
- Pupil reaction and size estimation, under direct light.

Allow participants approximately 90 seconds for the eyes to adapt to the darkened conditions.

Sequence of roles should be as follows:

- Test Administrator
- Test Subject
- Coach
- Test Administrator (continue cycle)
EYE EXAMINATIONS DATA SHEET

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Reaction: 

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Reaction:
D. Session Wrap-Up
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Learning Objectives

- Correctly administer the preliminary examinations and psychophysical tests used in the drug influence evaluation procedure.
- Observe and record the subject’s performance on the preliminary examinations and psychophysical tests.
- Determine the level of impairment based on the results of the subject’s preliminary examinations and psychophysical tests.

Upon successfully completing this session the participant will be able to:

- Correctly administer the preliminary examinations and psychophysical tests used in the drug influence evaluation procedure.
- Observe and record the subject’s performance on the preliminary examinations and psychophysical tests.
- Determine the level of impairment based on the results of the subject’s preliminary examinations and psychophysical tests.

CONTENT SEGMENTS .................................................................................. LEARNING ACTIVITIES
A. Procedures ............................................................................................. Instructor-Led Presentations
B. Hands-On Practice .................................................................................. Participant-Led Practice
C. Session Wrap-Up ..................................................................................... Instructor Discussion
A. Procedures

The preliminary examinations and psychophysical tests include:

- Pupil Size Estimation (Room Light)
- Horizontal Gaze Nystagmus
- Vertical Gaze Nystagmus
- Lack of Convergence
- Modified Romberg Balance
- Walk and Turn
- One Leg Stand (Both Legs)
- Finger to Nose
- Pulse Rate
Team Member Duties

- One team member will administer the tests to the volunteer
- One team member will record the results on the report form
- The other team member(s) will assist the test administrator in observing the volunteer’s performance on the tests

Some volunteers will have BACs above 0.10, others will have lower BACs.

The following safety precautions will be strictly enforced:

- No weapons will be present.
- Volunteers will not be left unattended at any time.

B. Hands-On Practice

*Test Administration*
C. Session Wrap-Up

Feedback of teams’ assessments:
Ask each team briefly to describe the evidence that led the members to their conclusions about a particular volunteer’s BAC.

Feedback of volunteer’s BACs:

Discussion
Participant Manual

Drug Recognition Expert Course

Session 13

Physician's Desk Reference (PDR) and Other Reference Sources
Upon successfully completing the session, the participant will be able to:

- Explain how the various sections of the PDR can provide information that will:
  - a) Aid in the drug influence evaluation
  - b) Aid in courtroom testimony.
- Use the PDR in a practical exercise.
- Learn about other resources available to assist DREs.

**CONTENT SEGMENTS**

**LEARNING ACTIVITIES**

A. Procedures .................................................. Instructor-led Presentation
B. Practical Exercises
C. Other Resources Available
A. Procedures

*PDR: Physician's Desk Reference*

PDR is published annually.

Many versions are published:
- Prescription
- Non-prescription
- Ophthalmology

PDR supplements are published periodically as new products are introduced during the year.

Function of the publisher is compilation, organization and distribution of information.

Product descriptions are prepared by the manufacturer, and edited and approved by their respective medical directors.

Additional information on the various drugs can be obtained from the manufacturer.
Sections of a PDR

• Section 1
  • Manufacturers Index – List of manufacturers (with phone numbers) who have provided prescribing information.

• Section 2
  • Product Name Index and Discontinued Products – Alphabetical listing of products available and a listing of discontinued products. Newer editions of the PDR will have a merging of Sections 2 and 4.

• Section 3
  • Product Category Index – Products listed according to appropriate category.

• Section 4
  • Generic and Chemical Name Index – Products listed under generic and chemical name headings according to the principal ingredient(s).

• Section 5
  • Product Identification Section

• Section 6
  • Product Information Section – It also includes common names, generic compositions, or chemical names.
Sections of a Physician’s Desk Reference

Section 7:
• Diagnostic product information

Section 8:
• Poison control centers

Section 9:
• Guide to management of drug overdose

Use of the PDR in DEC Program

To identify prescription drugs.
This information is contained in the product identification section.

To identify the effects of prescription drugs for comparison with observed effects.
This information is contained in the product information section.

How to use the PDR

Identification of an unknown product.
Identification of drug pharmacology.
Example: MS Contin tablets (Morphine Sulfate).

Location and acquisition of agency’s PDR(s)

B. Practical Exercise
Suggested Criteria for Identifying a Non-PDR Source

- Be less than five years old (by copyright date)
- Be readily available in print or online
- Be periodically updated
- Be utilized by practitioners in the scientific and healthcare fields
- At a minimum, contain information on a particular drug’s: name, forms, actions and side effects

C. Other Resources

Suggested criteria to identify a non-PDR drug reference

When selecting an acceptable drug reference DRE’s should consult references that meet the below criteria:

- Be less than five years old (by copyright date).
- Be readily available in print or online.
- Be periodically updated.
- Be utilized by practitioners in the scientific and healthcare fields.
- At a minimum, contain information on a particular drug’s:
  - Trade (brand), generic, and alternate common names.
  - Available forms (liquid, pill, injectable, etc.).
  - Pharmacologic / therapeutic actions (as used clinically, both “on” and “off” label).
  - Adverse reactions and side effects.

The reason for this is to keep from consulting references that have become outdated and inaccurate.
Acceptable resources may be in-print, electronic, or a combination.

Acceptable written examples include:

- The Complete Guide to Prescription and Non-prescription Drugs
- The Pill Book
- Nursing Drug Handbook
- Nurse Pocket Drug Guide
- Drug Identification Bible (available at: http://www.drugidbible.com)
- Davis’s Drug Guide for Nurses
- Tarascon Pocket Pharmacopoeia (for those with some pharmacology education)
- The Monthly Prescriber’s Reference (MPR)
- Disposition of Toxic Drugs and Chemicals in Man, *(Source: Randall C. Baselt. Biomedical Publications)*
Acceptable electronic examples include:

- Drugs.com
- RxList.com
- WebMD.com/Drugs/Index-drugs.aspx
- Eprocrates.com
- iMeds – Medical Reference for Android
- Monthly Prescriber’s Reference (MPR)
- PDR.net

Other Information Sources

- National Highway Traffic Safety Administration, Enforcement and Justice Services Division
- Office of National Drug Control Policy
- State DEC Program Coordinator
- Governor’s Office of Highway Safety (GOHS)
Other Information Sources

- The National Traffic Law Center (NTLC)  
  http://www.ndaa.org/ntlc_home.html
- Local poison control center
- Medical dictionary

- Drugs and Human Performance Fact Sheets  
- Newspaper and magazine articles on drugs and drug impaired driving, including counter-culture magazines such as “High Times.”
- Software programs such as Pharmacists, Body Works, Mosby's Medical Dictionary and other programs are available on disks and CDs. Various resources are available through online services and the Internet.

The IACP Drug Evaluation and Classification Program website is http://www.decp.org
QUESTIONS?
Upon successfully completing this session the participant will be able to:

- Explain a brief history of the Hallucinogen category of drugs
- Identify common drug names and terms associated with this category
- Identify common methods of administration for this category
- Describe the symptoms, observable signs and other effects associated with this category
- Describe typical time parameters, i.e. onset and duration of effects, associated with this category
- List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of this category of drugs
- Correctly answer the “topics for study” questions at the end of this session

**CONTENT SEGMENTS** .......................................................................................... LEARNING ACTIVITIES

A. Overview of the Category ............................................................. Instructor-Led Presentations
B. Possible Effects .............................................................................. Review of Drug Evaluation and Classification Exemplars
C. Onset and Duration Effects ............................................................. Reading Assignments
D. Overdose Signs and Symptoms ...................................................... Video Presentations
E. Expected Results of the Evaluation ................................................ Slide Presentations
F. Classification Exemplars
A. Overview of the Category

Hallucinogens are drugs that affect a person’s perceptions, sensations, thinking, self-awareness and emotions.

The word “Hallucinogen” means something that causes hallucinations.

Definition from The Random House College Dictionary (Revised Edition, 1980)

A hallucination is a sensory experience of something that does not exist outside the mind.

Seeing, hearing, smelling, tasting or feeling something that isn’t really there.

Having distorted sensory perceptions, so that things look, sound, smell, etc. differently than they really are.

Hallucinogenic drugs usually produce what are called pseudo-hallucinations: i.e. the user typically is aware that what he or she is seeing, hearing, smelling, etc. isn’t real, but is a product of the drug.

But emphasize that the fact that the user knows the hallucinations aren’t real doesn’t make those hallucinations any less dangerous if they occur while driving.
Synesthesia

One common type of hallucination produced by these drugs is called Synesthesia, which is a sensory perception disorder, in which an input via one sense is perceived by the brain as an input via another sense. In its simplest terms, it is a transposition of senses.

Examples: The user may “see a flash of color, or some other sight, when the telephone rings.”

- Sounds for example, may be transposed into sights.
- Sights may be transposed into odors.
- The user may “smell” a particular fragrance when he or she looks at something painted yellow.
- The illusions and distorted perceptions produced by hallucinogenic drugs may be very alarming, even terrifying.
- They may produce panic and uncontrolled excitement.

The user may be unable to cope with the terror, and may attempt to flee wildly.

A user who is emotionally or mentally unstable may become psychotic in response to this frightening experience.
Flashback
A terrifying “bad trip” sometimes may be re-experienced as a flashback.

In simple terms, a flashback is a vivid recollection of a portion of a hallucinogenic experience. A flashback does not occur because of a residual quantity of drug in the user’s body. Instead, a flashback essentially is a very intense daydream. But point out that subsequent use of the drug may precipitate a flashback, by causing the user to re-experience the frightening illusions of the previous “bad trip.”
Types of Flashbacks

There are **three types** of flashback:

- **Emotional**: most dangerous - feelings of panic, fear, etc.; the sensations of a “bad trip.”
- **Somatic**: Altered body sensations, tremors, weakness, dizziness, crawly, tingly feelings on the skin.
- **Perceptual**: Distortions of vision, hearing, smell, taste and touch (associated with original “trip” least harmful, unless driving a motor vehicle)

**Delusion and Illusion**

Remember that hallucinogens produce delusions, illusions, or both.

- A delusion is a false belief.
  
  Example of a delusion: “I am an Elephant.”

- An illusion is a false perception, i.e. a misrepresentation of what the senses are receiving.
  
  Example of an illusion: “I see an Elephant.”
“Psychotomimetic” means “something that mimics psychosis.” A psychosis is a major mental disorder. It implies a loss of touch with reality.

Because they often make the user appear to be psychotic, Hallucinogens are sometimes called psychotomimetic drugs.

“Psychotomimetic” means “something that mimics psychosis.” A psychosis is a major mental disorder. It implies a loss of touch with reality.

Some Hallucinogens come from natural sources, while others are synthetically manufactured. Peyote, Psilocybin and Salvia Divinorum are examples of naturally occurring Hallucinogens.
LSD, TMA, DMT, MDMA, MDA, and 2CB are examples of synthetically manufactured Hallucinogens.

- LSD: Lysergic Acid Diethylamide.
- 25I-NBOMe: 2-(4-iodo-2,5-dimethoxyphenyl)-N-(2-methoxybenzyl)ethanamine. This synthetic drug and analogs exhibit effects similar to LSD. Referred to as “N-Bomb” or “Smiles”.
- TMA: Trimethoxyamphetamine
- DMT: Dimethyltryptamine
- MDMA is an abbreviation for 3,4-Methylenedioxymethamphetamine and is commonly referred to as “Ecstasy.” It is a hallucinogen that also acts as a stimulant. It produces an energizing effect, as well as distortions in time and perception and enhances enjoyment from tactile experiences.
- MDA is an abbreviation for 3,4-Methylenedioxyamphetamine. It is normally produced as a clear liquid, or as a white powder in capsule or tablet form.
- 2CB (4-Bromo-2, 5-Dimethoxyphenethylamine) is a white powder usually found in pressed tablets or gel caps. It is considered a synthetic psychedelic amphetamine. (DEA, Feb. 2011)
- STP is also known as DOM (2, 5-dimethoxy-4-methylamphetamine). STP is an abbreviation for “Serenity, Tranquility and Peace.”
Peyote is a small, spineless cactus.
The active, hallucinogenic ingredient in peyote is Mescaline.
Mescaline is a chemical relative of adrenaline. Effects may be similar to those that would result from a massive rush of adrenalin.
Mescaline was first isolated from Peyote in 1856. It was named after the Mescalero Apaches.
Peyote is used legally in religious ceremonies of the Native American Church.
Psilocybin is a drug found in a number of different species of mushrooms of the genus Psilocybe.
There are over 185 known species of mushrooms that contain psilocybin and psilocin.
These mushrooms also have been used in Native American religious ceremonies for thousands of years.
An unstable derivative of Psilocybin, called Psilocin, is also found in these mushrooms and also has hallucinogenic properties.
Psilocybin is chemically very similar to serotonin, a neurotransmitter that is found in the brain.
The effects of psilocybin may be similar to what would happen if the brain were suddenly flooded with Serotonin.
Salvia Divinorum, also known as S. divinorum or Salvia, is a naturally occurring Hallucinogen.

Salvia divinorum is a perennial herb in the mint family native to certain areas of Mexico. The plant, which can grow to over three feet in height, has large green leaves, hollow square stems and white flowers with purple calyces, can also be grown successfully outside of this region.

Salvia divinorum has been used by the Mazatec Indians for its ritual divination and healing. The active constituent of Salvia divinorum has been identified as Salvinorin A.

According to a National Survey on Drug Use and Health Report published by SAMHSA in February 2008, it is estimated that 1.8 million persons aged 12 or older used Salvia divinorum in their lifetime.

There are several methods of ingesting Salvia with varying durations of hallucinogenic effects:

- Dried leaves of Salvia can be smoked like marijuana, in a bong, pipe or as a joint, with the effects lasting up to 15-30 minutes.
- Fresh leaves can be chewed as a quid. The leaves of Salvia produce extractions of Salvinorin A before the leaves are removed from the mouth. Effects from chewing Salvia can last up to one hour.
- Salvinorin A can also be vaporized and inhaled by heating the leaves in a pipe of tin foil and the vapors inhaled through a glass pipe.

Effects of Salvia Divinorum include: intense hallucinations; feelings of floating through space or flying; twisting and spinning. Physical effects include dizziness; nausea; lack of coordination; slurred speech, confused sentence patterns; and chills.

Some common street names for Salvia Divinorum include: Salvia, Sally D, Magic Mint, Maria Pastora, and Diviner’s Sage.

Salvia is not listed under the Controlled Substance Act (CSA) or approved for medical use.

LSD is perhaps the most famous of the synthetically manufactured Hallucinogens.

• “LSD” is an abbreviation of Lysergic Acid Diethylamide.

It was first produced in 1938, although its hallucinogenic properties were not discovered until 1943.

• LSD was used in psychotherapy during the 1940’s and early 1950’s.

  Example: it was occasionally used in the treatment of alcoholism.

Although LSD is a synthetic drug, it was first derived from Ergot, a fungus that grows on rye and other grains.

In the Middle Ages, when people accidentally ate this fungus, their resulting bizarre behavior was thought to stem from possession by the Devil.

• Ergot is still used medically to treat migraine headaches. Sandoz Laboratories markets a combination of caffeine and Ergot called Cafergot.

• 2CB (4-Bromo-2, 5-Dimethoxyphenethylamine) is a popular drug first synthesized in 1974.

• 2CB is considered both a psychedelic and an entactogen.

• “Entactogen” is a term used by psychiatrists to classify Ecstasy (MDMA). It literally means “touching within.”

• 2CB is a white powder usually found in pressed tablets or gel caps.

• 2CB is sometimes referred to as “Venus”; “Nexus”; and “Bromo-Mescaline.”
MDA, MDMA, STP, and TMA are synthetically manufactured hallucinogens that sometimes are called “Psychedelic Amphetamines.”

- MDA is an abbreviation for 3, 4-Methylenedioxyamphetamine.
- MDMA is an abbreviation for 3,4-Methylenedioxymethamphetamine
- STP is an abbreviation for 2,5-Dimethoxy-4-methylamphetamine
- TMA is an abbreviation for 3, 4, 5-Trimethoxyamphetamine.
- Chemically related to Amphetamines and produce many effects similar to those of CNS Stimulants.
- Chemically related to Mescaline.

Among users, MDA sometimes is referred to as the “Mellow Drug of America.”

An important fact about Hallucinogens is that they are not addictive, in the sense that cessation of use does not produce withdrawal signs or symptoms; however, regular users do develop tolerance to these drugs.
Methods of Ingestion of Hallucinogens

The most common method of ingesting Hallucinogens is orally. Some Hallucinogens can also be smoked. However, LSD cannot be ingested by smoking. LSD is usually ingested orally, which produces rapid effects. It can also be absorbed by placing drops in the eye.

Some Hallucinogens can be ingested and absorbed through the skin. MDA can also be insufflated, or “snorted.”
B. Possible Effects

The effects of Hallucinogens vary widely, and are affected by the user’s personality, mood and expectations, and by the surroundings in which the drug is taken.

The most common effect of the Hallucinogen is hallucination: the distorted perception of reality, often with a mixing of senses that makes it virtually impossible for the drug influenced user to function in the real world.

Generally, Hallucinogens intensify whatever mood the user is in at the time the drug is taken.

- If the user is depressed, the drug will deepen the depression.
- If the user is feeling pleasant, the drug will heighten that feeling.

If the user expects that the drug will help him or her achieve new insights or an expanded consciousness, the “trip” will seem to have that effect.

However, Hallucinogens also often uncover mental or emotional flaws that the user was unaware of possessing.

Therefore, many users who expect a positive experience with the drug will encounter instead the panic of a “bad trip.”
C. Onset and Duration Effects

*Time Factors of Peyote*

The time parameters associated with Hallucinogens vary from drug to drug.

The effects of Peyote (Mescaleine) begin to be felt within approximately one-half hour after eating the cactus “buttons.”

30 minutes: nausea, possibly leading to vomiting; mild rise in blood pressure, pulse, temperature and heart rate; pupils dilate.

One hour: sensory changes begin; visual distortions accompanied by rich colors; objects take on new forms and begin to move; shapes “come alive.”

3 – 4 hours: sensory changes reach their peak; synesthesia (transposition of senses) commonly occurs.

10 hours: gradual decline in effects.

12 hours: nearly total recovery from effects.

24 hours: the majority of the Mescaleine has been excreted from the body.
Time Factors of Psilocybin

Psilocybin also begins to exert its effects within one-half hour.

First 30 minutes: dizziness, light headed feeling, giddiness; the extremities (hands, feet, etc.) may feel very light or very heavy.

30 – 60 minutes: vision blurs; colors become brighter, leave longer lasting after images; objects take on sharp visual definition; hearing becomes more acute.

60 – 90 minutes: color patterns and shapes start to develop; the surfaces of objects appear to develop waves and wave-like patterns; distance perception becomes impaired; feelings of euphoria develop.

90 – 120 minutes: body sensations increase, along with mental perceptions; user commonly becomes introspective, with increased bodily sensations and mental perceptions.

120 – 180 minutes: effects start to diminish.

180 – 300 minutes: Nearly complete resolution of drug-induced effects.

Source: Drug Identification Bible, 2014
LSD’s effects begin to be felt within 30 – 45 minutes.

30 – 45 minutes: blood pressure, pulse and temperature rise; pupils dilate; hair starts to stand on end (Piloerection); nausea, dizziness and headache development.

4 – 6 hours: effects reach their peak.

7 – 9 hours: effects diminish.

10 – 12 hours: user feels normal.

MDMA’s effects usually begin within several minutes to a half hour if taken orally. Psychological effects include confusion, depression, anxiety and paranoia. The duration effects can last from 1 – 12 hours depending on dosage.

2CB’s effects are dose related.

Lower doses (5-15mg) produce enhanced sensual sensations and feelings of being “in one’s body.”

At higher doses (15-30mg) it produces intense visual effects that includes moving objects with “trails” behind them and colors appearing from nowhere.

Onset and duration of effects of other Hallucinogens vary widely from about two hours to about 24 hours.
D. Overdose Signs and Symptoms

The most common danger of an overdose of Hallucinogen is an intense “bad trip,” which can result in severe and sometimes permanent damage.

It is unlikely that other Hallucinogens would directly result in death from overdoses.

However, an overdose can be extremely dangerous and indirectly result in death.

The extreme panic and agitation of a “bad trip” have been known to result in suicide or in accidental death as the user attempts to flee the hallucinations.

Sometimes Hallucinogens induce a perception of invulnerability in the user, leading to bizarre and very dangerous behavior, and death.

Example: at least one LSD user was killed when he attempted to stop a train. Others have died from jumping off buildings believing they can fly.

Some evidence suggests that prolonged use of LSD may produce organic brain damage, leading to impaired memory, reduced attention span, mental confusion and impaired ability to deal with abstract concepts.
E. Expected Results of the Evaluation

Observable Evidence of Impairment

Eye Exams:

- Neither Horizontal Gaze nor Vertical Gaze Nystagmus will be present.
- Lack of Convergence will not be evident.

Psychophysical Tests:

- Performance on the Modified Romberg Balance test will be impaired, particularly in the subject’s estimation of the passage of 30 seconds.
- Performance on the Walk and Turn, One Leg Stand, and Finger to Nose tests will be markedly impaired due to the subject’s severe visual distortion, impaired perception of distance and decreased muscle coordination.

Vital Signs

Pulse will generally be elevated
Blood pressure generally will be elevated
Body temperature generally will be elevated
**Dark Room**

Pupils generally will be dilated

Reaction to light will usually be normal. Certain Psychedelic Amphetamines may cause slowing of the pupil’s reaction to light.
**General Indicators**

- Body tremors
- Dazed appearance
- Difficulty with speech
- Disoriented
- Flashbacks
- Hallucinations
- Memory loss
- Nausea
- Paranoia
- Perspiring
- Piloerection
- Poor perception of time and distance
- Synesthesia
- Uncoordinated
### Hallucinogen Symptomatology Chart

<table>
<thead>
<tr>
<th></th>
<th>None</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGN</td>
<td>None</td>
</tr>
<tr>
<td>VGN</td>
<td>None</td>
</tr>
<tr>
<td>Lack of Convergence</td>
<td>None</td>
</tr>
<tr>
<td>Pupil Size</td>
<td>Dilated</td>
</tr>
<tr>
<td>Reaction to Light</td>
<td>Normal (3)</td>
</tr>
<tr>
<td>Pulse Rate</td>
<td>Up</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Up</td>
</tr>
<tr>
<td>Temperature</td>
<td>Up</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Rigid</td>
</tr>
</tbody>
</table>

(3) Certain psychedelic amphetamines may cause slowing.

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**F. Classification Exemplar**

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TOPICS FOR STUDY

1. What does “synesthesia” mean?

2. What is a “flashback”? What are the three types of “flashback”?

3. Name two naturally occurring Hallucinogens.

4. What is a “bad trip”?

5. What does “psychotomimetic” mean?

6. What is an “illusion”? What is a “delusion”?

7. What is the difference between “hallucinations” and “pseudo-hallucinations”?

8. What is “piloerection”?
**DRUG INFLUENCE EVALUATION**

**Evaluator:** Officer Chris Thurman, Louisville Metro PD
**DRE #** 16444  
**Rolling Log #** 14-07-12  
**Case #** 14-07-6755  
**Session XIV - #1**

**Record/Witness:** Officer Dean Kisling, Louisville Metro PD  
**Crash:** None  
**Fatal Injury:** No  
**Property:** 

**Arrestee's Name:** Trumpet, Angel  
**Date of Birth:** 01/23/92  
**Sex:** F  
**Race:** White  
**Arresting Officer Agency:** Kentucky Vehicle Enforcement

**Date Examined / Time / Location:** 07/29/14 1830 Jefferson Co. Jail  
**Recorder:** Wrtness Crash: None  
**Arresting Officer:** Officer Chris Thurman, Louisville Metro PD  
**Name, ID:** 16444 14-07-12  
**Session:** XIV - #1  
**Instrument #** 36642  
**Test Results:** 0.00  
**Chemical Test:** None  
**Blood Test:** Yes

**Breath Results:** Test Refused: D  
**Chemical Test:** Test or tests refused: 

**Officer:** Officer Dean Kisling, Louisville Metro PD  
**Name, ID:** D  
**Fatal Injury:** No  
**Property:** 

**Arrestee's Name:** Trumpet, Angel  
**Last, First, Middle:** 01/23/92  
**Sex:** F  
**Race:** White  
**Arresting Officer Agency:** Kentucky Vehicle Enforcement

**Date Examineo** 07/29/14  
**Time** 1830  
**Location** Jefferson Co. Jail  
**Breath Results:** 0.00  
**Instrument #** 36642

**Officer's Signature:** Officer Chris Thurman, Louisville Metro PD  
**Date DRE was notified:** 1745  
**Officer's Signature:** Officer Dean Kisling, Louisville Metro PD

**Opinion of Evaluator:** Not Impaired  
**Medical:** 

**PUPIL SIZE**  
**Room Light:** 2.5 - 5.0  
**Darkness:** 5.0 - 8.5  
**Direct:** 2.0 - 4.5

**Finger to Nose**  
(Prepared to speak touched)  
**N/A**

**Reaction to Light**  
Normal  
**N/A**

**Nasal area:** Clear  
**Oral cavity:** Brown coating on tongue

**Type of footwear:** Lace-up work boots

**Finger to Nose**  
**Room Light:** 2.5 - 5.0  
**Darkness:** 5.0 - 8.5  
**Direct:** 2.0 - 4.5

**HGN**  
Lack of Smooth Pursuit  
Maximum Deviation  
Angle of Onset  
None

**Difficulty to stand:** Test stopped

**Describe Turn**  
N/A  
**Cannot do test:** (explain)

**Type of Trunk:** N/A

**N/A**

**Muscle tone:** 
Normal  
Flaccid  
Rigid

**Comments:**  
Nothing observed.

**Blood pressure:** 148 / 96  
**Temperature:** 99.8 o

**Duress:**  
**N/A**

**Time of use:** N/A  
**Where were the drugs used? (Location):**

**Date / Time of arrest:** 07/29/14 1705  
**Time DRE was notified:** 1745  
**Evaluation start time:** 1830  
**Evaluation completion time:** 1940  
**Precinct/Station:**

**Opinion of Evaluator:** Not Impaired  
**Medical:** Alcohol  
**CNS Stimulant:** 
**CNS Depressant:** 
**Hallucinogen:** 
**Dissociative Anesthetic:** 
**Inhalant:** 
**Narcotic Analgesic:** 
**Inhaling:**
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Trumpet, Angel

1. LOCATION: Evaluation was conducted in the Interview Room of the Jefferson Co. Jail.

2. WITNESSES: Officer Dean Kisling of the Louisville Metro PD recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was a 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was contacted by Officer Belcher and requested to conduct a drug evaluation. I contacted him at the jail where he advised he had located the suspect stopped partially in the travel portion of I-64. The suspect appeared dazed and very disoriented. Several times she pointed to some lights near the Interstate and told Officer Belcher that she stopped because the lights were so bright. Officer Belcher administered roadside SFST’s, which she was unable to perform as directed, and she was subsequently arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: The suspect was seated in the Interview Room and was staring straight ahead. When I entered the room she quickly turned and asked “Are you God?” I responded by giving her my name and asking for consent to conduct a drug evaluation. She replied, “They sent you, it must be okay.” Her speech was rapid, and she stuttered at times. She was perspiring heavily, and at times acted very paranoid.

6. MEDICAL PROBLEMS AND TREATMENT: The suspect indicated that she had an upset stomach from something she ate, but did not require medical assistance.

7. PSYCHOPHYSICAL TESTS: At times the suspect was unable to stand without assistance. Due to her poor balance, and it was necessary to terminate the Modified Romberg Balance, Walk and Turn, and One Leg Stand tests for her safety. The Finger to Nose test was conducted while she was seated. She missed the tip of her nose on all six attempts, and got visibly upset when she could not touch her nose.

8. CLINICAL INDICATORS: The suspect’s pupils were dilated in two of the lighting levels. Her pulse, blood pressure, and temperature were elevated, and above the DRE average ranges.

9. SIGNS OF INGESTION: The suspect’s breath was rancid smelling.

10. SUSPECT’S STATEMENTS: The suspect stated she was fasting for religious reasons, and is not allowed to use alcohol or drugs. The suspect stated she got hungry so she purchased some “organic mushrooms” from a guy at a truck stop near Lexington.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a Hallucinogen and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS: Rev. 10/15
**DRUG INFLUENCE EVALUATION**

**Evaluator**
Sergeant Allan Kolak  Cape Coral PD

**Arrestee’s Name (Last, First, Middle)**
Tripp, Brad

**Recorder**
Kyle Clark  IPTM

**Date Examined**
07/18/88

**Given by:**
Sergeant Allan Kolak  Cape Coral PD

**Miranda Warning Given**
Yes

**Recorded by:**
Kyle Clark  IPTM

**Date of Birth**
07/18/88

**Deputy Darrel Kehne #9077**

**Test Results:**
None

**Chemical Test**
Test or tests refused

**Location**
2210 Collier Co. Jail

**Date Examined / Time / Location**
05/17/14  2210 Collier Co. Jail

**Period of use?**
N/A

**Deputy Darrel Kehne #9077**

**Test Results:**
None

**Chemical Test**
Test or tests refused

**Date of Birth**
07/18/88

**Are you diabetic or epileptic?**
Yes [X] No

**Blood Odor:**
None

**Breath Results:**
Results: 0.00

**Chemical Test**
Urine [X] Blood 

**Blood Odor:**
None

**Pupil Size:**
Right Eye 6.0  Left Eye 9.0

**Room Light**
0.0 2.0 - 4.5

**Darkness**
5.0 - 8.5

**Direct**
6.0

**Pupil Size**
Left Eye 6.0  Right Eye 9.0

**Room Light**
0.0 2.0 - 4.5

**Darkness**
5.0 - 8.5

**Direct**
6.0

**Pupil Size**
Left Eye 6.0

**Room Light**
0.0

**Darkness**

**Direct**

**Blood pressure**
160 / 96

**Temperature**
99.8°F

**Muscle tone:**
Normal

**Comments:**
Nothing detected.

**Date / Time of arrest:**
05/17/14  2105

**Time DRE was notified:**
2125

**Evaluation start time:**
2210

**Evaluation completion time:**
2305

**Precinct/Station:**

**Opinion of Evaluator:**

**Name:**

**ID#**

**Rev-01/13**
Suspect: Tripp, Brad

1. LOCATION: The evaluation was conducted in the Collier County Jail Interview Room.

2. WITNESSES: DRE State Coordinator, Kyle Clark witnessed and recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to conduct a drug evaluation for Deputy Kehne and contacted him at the County Jail. He advised that he had arrested the suspect after observing him driving along the gravel shoulder of Beach Road trying to pass some slower moving vehicles. According to Deputy Kehne, the suspect was acting very strange and at times began talking to imaginary people. The suspect also claimed that the overhead lights on Deputy Kehne’s patrol car were burning his eyes and skin. He did poorly on the SFSTs’ and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect sitting in the interview room. He appeared to be extremely disoriented. At times, he was talking to himself, and once he pointed to the clock on the wall and began talking to it.

6. MEDICAL PROBLEMS AND TREATMENT: None observed and none stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 3” front to back and side to side. He estimated 30 seconds in 16 seconds. Walk & Turn: He lost his balance twice during the instructions, started too soon, stopped while walking three times, missed heel to toe numerous times, and used his arms for balance throughout the test. On the turn, he lost his balance and nearly fell. One Leg Stand: Suspect swayed while balancing and used his arms. He put his foot down once while standing on his left foot and twice while standing on the right foot. Finger to Nose: Suspect missed the tip of his nose on each attempt and used the pads of his fingers three times.

8. CLINICAL INDICATORS: The suspect’s pulse, blood pressure, and temperature were all elevated and above the DRE average ranges. The suspect’s pupils were dilated and above the DRE average ranges. HGN, VGN and LOC were not present.

9. SIGNS OF INGESTION: None observed.

10. SUSPECT’S STATEMENTS: The suspect stated that he felt hot and denied drug use.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a Hallucinogen and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS:
<table>
<thead>
<tr>
<th>Officer's Signature:</th>
<th>Officer Tim McCarson Albuquerque PD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name, ID#:</td>
<td>14-10-070</td>
</tr>
<tr>
<td>DRE #:</td>
<td>14-72135</td>
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<tr>
<td>Case #:</td>
<td>Session XIV - #3</td>
</tr>
<tr>
<td>Arrester's Name:</td>
<td>Intervening Alcohol</td>
</tr>
<tr>
<td>ellido, First, Middle</td>
<td></td>
</tr>
<tr>
<td>Record #:</td>
<td>6994</td>
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<tr>
<td>Location:</td>
<td>Albuquerque PD</td>
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<tr>
<td>Date Examined:</td>
<td>10/21/14</td>
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<tr>
<td>Time / Location:</td>
<td>1410 Albuquerque PD</td>
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<tr>
<td>Breath Results:</td>
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<tr>
<td>Test Refused:</td>
<td>Yes</td>
</tr>
<tr>
<td>Chemical Test:</td>
<td>Blood</td>
</tr>
<tr>
<td>Test or tests refused:</td>
<td>Yes</td>
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<tr>
<td>Time now / Actual</td>
<td>4 pm / 1415</td>
</tr>
<tr>
<td>When did you last sleep?</td>
<td>3-4 hours</td>
</tr>
<tr>
<td>How long?</td>
<td></td>
</tr>
<tr>
<td>Are you sick or injured?</td>
<td>Yes</td>
</tr>
<tr>
<td>Are you diabetic or epileptic?</td>
<td>No</td>
</tr>
<tr>
<td>Do you take insulin?</td>
<td>Yes</td>
</tr>
<tr>
<td>Do you have any physical defects?</td>
<td>No</td>
</tr>
<tr>
<td>Are you under the care of a doctor or dentist?</td>
<td>No</td>
</tr>
<tr>
<td>Attitude:</td>
<td>Poor</td>
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<tr>
<td>Coordination:</td>
<td>Staggering</td>
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<td>None</td>
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<tr>
<td>Glasses:</td>
<td>Hard</td>
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<td>Soft</td>
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<td>Bloodshot:</td>
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<tr>
<td>Watery:</td>
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<td>Pupil Size:</td>
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<tr>
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</tr>
<tr>
<td>Pulse and time:</td>
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</tr>
<tr>
<td>1. 100 / 1425</td>
<td></td>
</tr>
<tr>
<td>2. 102 / 1433</td>
<td></td>
</tr>
<tr>
<td>3. 102 / 1433</td>
<td></td>
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<tr>
<td>Modified Romberg Balance:</td>
<td>Walk and Turn Test</td>
</tr>
<tr>
<td>Vertical Nystagmus:</td>
<td>Able to follow stimulus</td>
</tr>
<tr>
<td>Reaction to Light:</td>
<td>D Sways while balancing</td>
</tr>
<tr>
<td>Right Leg:</td>
<td></td>
</tr>
<tr>
<td>Right Count:</td>
<td>D D Hops</td>
</tr>
<tr>
<td>Right Foot:</td>
<td>D D Puts foot down</td>
</tr>
<tr>
<td>Right Count:</td>
<td>D D Puts foot down</td>
</tr>
<tr>
<td>Room Light:</td>
<td>2.5 - 5.0</td>
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<tr>
<td>Darkness:</td>
<td>5.0 - 8.5</td>
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<tr>
<td>Direct:</td>
<td>2.0 - 4.5</td>
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<td>Pupillary Unrest:</td>
<td>D No</td>
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<tr>
<td>Reaction to Light:</td>
<td>D Normal</td>
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<tr>
<td>Left Eye:</td>
<td>7.5</td>
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<tr>
<td>Right Eye:</td>
<td>7.5</td>
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<tr>
<td>Swayed badly:</td>
<td>Rigid</td>
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<tr>
<td>Rigid movements:</td>
<td>Used pads</td>
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<tr>
<td>Blood pressure:</td>
<td>146 / 98</td>
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<tr>
<td>Temperature:</td>
<td>99.8 °</td>
</tr>
<tr>
<td>Muscle tone:</td>
<td>Rigid</td>
</tr>
<tr>
<td>What drugs or medications have you been using?</td>
<td>Just a couple Molly's</td>
</tr>
<tr>
<td>How much?:</td>
<td>Nothing detected</td>
</tr>
<tr>
<td>Time of use?:</td>
<td></td>
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<tr>
<td>Where were the drugs used? (Location):</td>
<td>In the Park at the concert</td>
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<td>10/21/14 1320</td>
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<tr>
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<td>Opinion of Evaluator:</td>
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<td>Hallucinogen</td>
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<td>Inhalant:</td>
<td></td>
</tr>
<tr>
<td>Review / Approve by date:</td>
<td>Dec 01 15</td>
</tr>
</tbody>
</table>
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Flipping, Candi

1. LOCATION: The evaluation was conducted at the Albuquerque PD.

2. WITNESSES: The evaluation was recorded by Sgt. Joel Holt of the Rio Rancho PD.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to conduct a drug evaluation for Officer Golson of the APD. When contacted, he advised that he had observed the suspect driving 20 miles under the posted speed limit and weaving over the lane divider line on Lomas Blvd. When contacted, the suspect was extremely disoriented and had difficulty speaking. She was unable to do SFST’s due to her poor balance and coordination. No alcohol was detected, and she was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the booking area of the jail. She was perspiring heavily, and appeared dazed and disoriented. She responded slowly to my greeting, but was cooperative, and was responsive to my questions. She mumbled to herself and had rambling and slurred speech.

6. MEDICAL PROBLEMS AND TREATMENT: Suspect stated she felt nauseous.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Suspect swayed approximately 3” front to back and side to side. She estimated 30 seconds in 46 seconds. Walk & Turn and One Leg Stand: The suspect was unable to perform the tests and both had to be stopped for safety reasons. Finger to Nose: Suspect swayed noticeably and she missed the tip of her nose on all six attempts. She also used the pads of her fingers on each attempt.

8. CLINICAL INDICATORS: The suspect’s pupils were dilated and above the DRE average ranges in all three lighting levels. The suspect’s pulse, blood pressure, and body temperature were elevated, and were also above the DRE average ranges.

9. SIGNS OF INGESTION: Nothing was observed.

10. SUSPECT’S STATEMENTS: The suspect admitted taking “a couple Molly’s” at a rave earlier in the evening. She said they made her happy and helped her enjoy the music.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a Hallucinogen and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:
Participant Manual

Drug Recognition Expert Course

Session 15
Practice: Test Interpretation
Learning Objectives

• Analyze the results of a complete drug influence evaluation and identify the category or categories of drugs affecting the individual examined
• Articulate the basis for the drug category identification

Upon successfully completing this session the participant will be able to:

• Analyze the results of a complete drug influence evaluation and identify the category or categories of drugs affecting the individual examined.
• Articulate the basis for the drug category identification.

CONTENT SEGMENTS..................................................................................... LEARNING ACTIVITIES
A. Interpretation Demonstration................................................................. Instructor-Led Demonstrations
B. Interpretation Practice................................................................................ Small-Group Practice
C. Session Wrap-Up..................................................................................Participant-Led Presentations
A. **Interpretation Demonstrations**

---

**Preliminary examination**

---

**Eye examinations**

---

**Psychophysical tests**

---
Vital Signs Examinations

- Pulse
- Blood pressure
- Temperature

Dark Room Examinations

- Room light
- Near-total darkness
- Direct light
- Check nasal area and oral cavity
Narrative report
Interpretation Demonstrations
Case 2: Subject Baker

- Preliminary Examination
- Eye Examinations
- Psychophysical Tests

Preliminary examination

Eye examination

Psychophysical tests
Vital Signs examinations


Dark Room examinations


Other evidence and additional observations


Narrative Report


Opinion of the evaluator


B. Interpretation Practice

Team Practice

Teams will present their conclusions to the entire class.

Allow teams approximately 15 minutes to review the three exemplars and reach their conclusions.

Subject Charles
Subject Dodge
Subject Edwards
C. Session Wrap-Up
### DRUG INFLUENCE EVALUATION

**Evaluator**
Officer Mark Ashby
Thornton PD

**Recorder/Witness**
Deputy Mark George
Boulder Co. S.O.

**Arrestee's Name (Last, First, M/1st, i-1rst, MlacleJ**
Adams, Frank B.

**Date Examined**
10/06/14

**Officer**
Officer Mark Ashby
Thornton PD

**Arresting Officer (Name, ID#)**
Officer Alan Ma
#15588

**Date of Birth**
01/12/62

**Date of Arrest**
10/06/14

**Location**
2118

**County Jail Intake Center**

**Date of Intake**
2215

**Chemical Test**

- **Blood**

**Test or tests refused**

- **Chemical Test**

**Rolling Log #**
14-10-125

**Case #**
14-97302

**Session**
XV - #1

---

**Breath Results**
0.00

**Test Refused Instrument #**
44352

**Chemical Test**

- **Blood**

---

**Date of Intake**
2215

**County Jail Intake Center**

**Officer's Signature**
Adams, Frank B.

---

**Glasses O Contacts, if so**

- **None**

**Corrective Lenses**

- **None**

**Eye**

- **Reddened**

---

**HGN**

- **Right Eye**

**Eye**

- **Reddened**

---

**Rebound Dilation**

- **Right Eye**

**Pupillary Unrest**

- **Right Eye**

---

**Type of Footwear**

- **Dress shoes**

---

**Weight**

- **Normal**

---

**What drugs or medications have you been using?**

- **“Something to help me sleep.” “1 or 2 pills”**

---

**Date Time of arrest**
10/06/14

**Time DRE was notified**
2118

**Evaluation start time**
2150

**Evaluation completion time**
2215

**Precinct/Station**

- **2310**

---

**Blood pressure**

- **104 / 64**

**Temperature**

- **97.4 °**

---

**Reaction to Light**

- **Normal**

---

**Medication**

- **Alcohol**

---

**Muscle tone**

- **Flaccid**

---

**Medical**

- **CNS Depressant**

---

**Drug Refusal**

- **None**

---

**Officer's Signatures**

- **Signed by**

---

**Witness**

- **Deputy Mark George**

---

**Blood Results**

- **0.00**

---

**Actual When did you last sleep? How long?**

- **6 pm**

**Test or tests refused**

- **Chemical Test**

---

**Blood pressure**

- **104 / 64**

**Temperature**

- **97.4 °**

---

**Pupil Size**

- **Equal**

---

**Address**

- **2118**

---

**Medical ONS Depressant OHallucinogen OAnalgesic Ocannabis**
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Adams, Frances A.

1. LOCATION: The evaluation was conducted at the Denver County Jail.

2. WITNESSES: Deputy Mark George of the Boulder County S.O. recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Officer Alan Ma at the County Jail for a drug evaluation. Officer Ma advised that he arrested the suspect for DUI after observing his vehicle drifting outside its traffic lane and then making an improper turn. When stopped, the suspect had six clues of HGN and VGN. The suspect performed poorly on the SFST’s and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the booking room at the jail. His head was tilted forward, his eyes were closed, and his breathing was deep and slow. He responded slowly to questions. His speech was slow, slurred and thick. Several times when he stood, he would stagger and use the wall to steady himself.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated by the suspect.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Suspect had an approximate 2” front to back and 3” side to side sway. He estimated 30 seconds in 36 seconds. Walk & Turn: The suspect lost his balance twice during the instructions stage, stopped while walking three times, missed heel to toe six times, stepped off the line twice, and raised his arms for balance five times. He turned by walking around using both feet. One Leg Stand: Suspect swayed while balancing, used his arms for balance, and hopped several times. Finger to Nose: The suspect missed the tip of his nose on three of the six attempts.

8. CLINICAL INDICATORS: Suspect had six clues of HGN with a 35 degree angle of onset. VGN and a Lack of Convergence were present. The suspect’s pulse rates, blood pressure, and temperature were below the DRE average ranges.

9. SIGNS OF INGESTION: Nothing observed.

10. SUSPECT’S STATEMENTS: The suspect said he was taking some medicine to help him sleep. When asked the name of the medicine, he could remember.

11. DRE'S OPINION: In my opinion, the suspect is under the influence of a ____________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: The suspect provided a blood sample for analysis.

13. MISCELLANEOUS:
## DRUG INFLUENCE EVALUATION

**Evaluator:** Trooper Joseph Germano NY State Police  
**DRE#:**olfingTog 'ff 10712 14-08-021  
**Case #** 14-99875 Session XV - #2  
**Recorder/Witness:** Trooper David Olney NY State Police  
**Date Examined:** 08/04/14  
**Time:** 2210 Cooperstown PD  
**Date of Birth:** 10/15/88  
**Sex:** M  
**Race:** B  
**Date of Arrest:** 08/04/14  
**Time:** 2110  
**Location:** Cooperstown PD  
**Chemical Test:**  
- **Urine:**  
- **Blood:**  
**Test Results:** 0.00  
**Test Refused:**  
**Instrument #:** 44321  
**Arresting Officer (Name, ID#):** Trooper Jim Guerriere #5525  
**An=unicer AgenFr New York State Police**

### Breath Results:
- **Test Results:** 0.00  
- **Test Refused:**

### Time of Last Drink:
- **Time:** 9:30 pm  
- **Date:** This morning

### Other Relevant Information:
- **What have you eaten today? When?** French Fries 2 hours ago  
- **What have you been drinking? How much?** Water 1 bottle

### Physical Examination:
- **Eye Area:**  
  - Reddened Conjunctiva
  - No Blindness
  - Normal Pupil Size
  - Droopy Eyelids
  - No Pupil Unequal

### DRE Test Results:

<table>
<thead>
<tr>
<th>Test</th>
<th>1st Nine</th>
<th>2nd Nine</th>
<th>Actual steps taken</th>
</tr>
</thead>
<tbody>
<tr>
<td>M +</td>
<td>10</td>
<td>10</td>
<td>9 9</td>
</tr>
</tbody>
</table>

### Comments:
- Nothing detected.

### Blood Pressure and Temperature:
- **Blood pressure:** 142 / 98  
- **Temperature:** 99.7 °

---

**Opinion of Evaluator:** Not Impaired  
**Medical:**  
**CNS Depressant:**

---

**Date of Arrest:** 08/04/14  
**Time DRE was notified:** 2210  
**Evaluation start time:** 2130  
**Precinct/Station:** 2315  
**Reviewed/approved by:**

---

**MIRANDA WARNING GIVEN**
- **Yes:**  
- **No:**

---

**Type of Footwear:** Lace-up shoes  
**Finger to Nose:**

---

**HGN:**
- **Lack of Smooth Pursuit:** None
- **Maximum Deviation:** None
- **Angle of Onset:** None

---

**Walking and Tying Test:**
- **Cannot keep balance:**
  - Starts too soon
  - Sways while balancing
  - Uses arms to balance
  - Hops
  - Puts foot down

---

**Corrective Lenses:** None  
**Contacts:** None

---

**Nystagmus:**
- **Right Eye:**
  - 1st Nine
  - 2nd Nine
- **Left Eye:**
  - 1st Nine
  - 2nd Nine

---

**Pupil Size:** Room Light 2.5 - 5.0  
**Darkness 5.0 - 8.5  
**Direct 2.0 - 4.5

**Rebound Dilation:**
- **Left Eye:** 8.0  
- **Right Eye:** 8.0

---

**Convergence:**
- **Right eye:** Left eye

---

**Reaction to Light:** Slow
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Baker, Samuel

1. LOCATION: The evaluation was conducted at the Cooperstown Police Department.

2. WITNESSES: The evaluation was witnessed and recorded by Tpr. Olney of the NY SP.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to meet Trooper Guerriere at the Cooperstown PD for a drug evaluation. It was determined that Trooper Guerriere had arrested the suspect for DUI after observing his vehicle cross the center line and nearly collide with another vehicle. Tpr. Guerriere reported that the suspect was fidgety acting. His speech was quick and difficult to understand at times. He was unable to complete the SFSTs as directed, and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect standing in the breath testing room. He was repeatedly shifting his weight from foot to foot, and appeared restless. He was frequently moving his hands and arms. His speech was fast and slurred. His pupils appeared to be dilated, and he was grinding his teeth.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Suspect had an approximate 3” front to back and 2” side to side sway. He estimated 30 seconds in 21 seconds. Walk & Turn: The suspect started too soon, missed heel to toe three times, and raised his arms for balance five times, and performed the test quickly. One Leg Stand: Suspect swayed while balancing, used his arms for balance, and put his foot down once. He also counted quickly, and slurred his numbers when counting. Finger to Nose: Suspect missed the tip of his nose on three of the six attempts, and had quick, jerky arm and hand movements.

8. CLINICAL INDICATORS: The suspect’s pulse, blood pressure, and temperature were all elevated and above the DRE average ranges. The suspect’s pupils were dilated and above the DRE average ranges in two of the lighting levels.

9. SIGNS OF INGESTION: The suspect had a reddened nasal area, and his nose was runny.

10. SUSPECT'S STATEMENTS: The suspect denied using drugs.

11. DRE'S OPINION: In my opinion, the suspect is under the influence of a ____________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS:

Rev. 10/15
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Charles, Mary

1. LOCATION: The evaluation was conducted at the WSP Office in Seattle.

2. WITNESSES: The evaluation was witnessed by Sgt. Courtney Stewart of the WSP.

3. BREATH ALCOHOL TEST: The suspect’s breath test was a 0.05%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to conduct a drug evaluation for Officer Jongma of the Seattle PD. Officer Jongma advised that the suspect had been reported as a possible DUI. The suspect was located traveling NB on I-5 near King Street and her vehicle was unable to maintain a single lane of travel. When contacted, the suspect had slow, sluggish reactions. Her speech was thick and slurred. She performed poorly on the SFST’s and was arrested for DUI. She admitted drinking a couple of glasses of wine earlier in the evening.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room. She was swaying as she stood, and was unstable on her feet. Her speech was slow, thick, and slurred. She was very emotional at times and began crying several times.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect had an approximate 2” circular sway. She estimated 30 seconds in 32 seconds. Walk & Turn: She lost her balance twice during the instructions, stopped walking once, missed heel to toe twice, and stepped off the line twice, and raised her arms for balance. She lost her balance, staggering on the turn. One Leg Stand: She swayed, and used her arms for balance, and put her foot down once while standing on her left foot and twice while standing on the right. Finger to Nose: Suspect missed the tip of her nose on three of the six attempts.

8. CLINICAL INDICATORS: Suspect had four clues of HGN, with a Lack of Convergence. Her pulse rates and blood pressure were at the low end of the DRE average ranges.

9. SIGNS OF INGESTION: The suspect had an odor of an alcoholic beverage on her breath.

10. SUSPECT’S STATEMENTS: Suspect admitted drinking a “couple glasses of wine” and admitted smoking some marijuana 2 or 3 days ago.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of ________________ and is unable to operate a vehiclesafely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS: 

Rev. 10/15
DRUG INFLUENCE EVALUATION

Evaluator: Sergeant Joseph Milos
Bellevue PD

DRE #: 4477
Rolling Log #: 14-02-008

Case #: 14-20258
Session XV - #4

Evaluator's Name (Last, First, Middle): Dodge, Fred A.

Date or Time: 10/13/75
Sex: M
Race: W

Arresting Officer (Name, ID#): Sergeant Dale Hilderbrand #6047

Arresting Agency: Grand Island Police Department

Date Exam/Air / Time / Location: 02/22/14 0210 Grand Island PD

Breath Results: Test Refused

Chemical Test: Instrument # 37755

Blood: X

Witness: Crash: [El None

Arresting Officer: Sergeant Martin Denton Nebraska SP

Date of Arrest: 02/15

Summary of Arrest:

Dodge, Fred A. 10113/75 M Grand Island Police Department

Date Examined / Time: 02/22/14 0210

Grand Island PD

Breath Results: Test Refused

Chemical Test: Instrument # 37755

Blood: X

Yes

What have you eaten today? When?

What have you been drinking? How much?

Time of last drink?

Coffee 2-3 cups

Time now:

What have you done in the last 24 hours?

When?

What have you been doing in the last 24 hours?

Where?

Yes

No

Do you have any physical defects?

Yes

No

Are you under the care of a doctor or dentist?

Yes

No

Attitude:

Rapid, Slurred

Speech:

Breath Odor:

Rapid, Slurred

Corrective Lenses:

Glasses D Contacts if so D Hard D Soft

Pupil Size:

Equal

Blindness:

None D Left D Right

Able to follow stimulus

0 Yes 0 No

Coordination:

Poor, Quick

Walk when tested:

Rapid, Slurred

Nasal area:

Reddened

Clear

Rebound Dilation:

Yes

No

Pupillary Unrest

0 Yes 0 No

Reaction to Light:

Slow

Type of footwear:

N/A

General:

N/A

Jaw:

Normal

Droopy

Right Eye

Left Eye

Vertical Nystagmus

0 Yes 0 No

Corrective Lenses:

Glasses D Contacts if so D Hard D Soft

Pupil Size:

Equal

Refused

Where were the drugs used? (Location)

Refused

Time of use?

Refused

Blood pressure: 162 / 96

Muscle tone:

Normal

Flaccid

Rigid

Comments:

"I'm not answering that."

What drugs or medications have you been using?

Refused

How much?

Refused

Time of use?

Refused

Where were the drugs used? (Location)

Refused

Date / Time of arrest:

02/22/14 0108

Time DRE was notified:

0130

Evaluation start time:

0210

Evaluation completion time:

0250

Precinct/Station:

Reviewed/approved by:

Date:

Observer's Signature:

DRE #:

Opinion of Evaluator:

Not Impaired

Alcohol

CNS Stimulant

Disassociative Anesthetic

Inhalant

Medical

CNS Depressant

Hallucinogen

Narcotic Analgesic

Cannabis
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Dodge, Fred

1. LOCATION: The evaluation was conducted at the Grand Island Police Department.

2. WITNESSES: The evaluation was recorded by the arresting officer, Sergeant Dale Hilderbrand of the Grand Island PD, and witnessed by Sgt. Martin Denton of the NE SP.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was contacted and requested to conduct a drug evaluation for Sgt. Hilderbrand. It was determined that the suspect had been involved in an attempt to elude and was apprehended after a short pursuit. The suspect was very restless, animated, and unable to stand still. He was very talkative, and his speech was rapid and slurred. He had difficulty performing the SFST’s, and was arrested for DUI and several other related charges.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at the PD. His speech was rapid, loud and slurred. He had quick movements and was unable to stand still. He was constantly moving around the room. He appeared to be sweating and his pupils appeared to be dilated.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Suspect had an approximate 2” side to side sway. He estimated 30 seconds in 22 seconds. Walk & Turn: Suspect lost his balance once during the instructions, twice started the test too soon, stopped while walking four times, missed heel to toe once, raised his arms for balance, and walked rapidly. One Leg Stand: Suspect swayed while balancing, and put his foot down once while standing on his right foot. Finger to Nose: Suspect missed the tip of his nose on all six attempts.

8. CLINICAL INDICATORS: The suspect’s pulse rates, blood pressure, and temperature were above the DRE average ranges. His pupils were dilated, with a slow reaction to light.

9. SIGNS OF INGESTION: The suspect had two red puncture marks on the inside of his left forearm. When asked about them, he laughed, and said, “I’m not answering that.”

10. SUSPECT’S STATEMENTS: Suspect denied any drug use.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a ____________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:
**DRUG INFLUENCE EVALUATION**

**Evaluator:** Sergeant Jim Roy  
**Colchester Police Dept.**  
**DRE #:** 12574  
**Mapping Log #:** 14-08-035  
**Case #:** 14-10075  
**Session XV - #5**

**Recorder / Witness:** Lt. John Flannigan  
**Vermont State Police**  
**Arrestee's Name (Last, First, Middle):** Edwards, Joan L.  
**Date of Birth:** 01/16/92  
**Sex:** F  
**Race:** W  
**Arresting Officer (Name, ID):** Officer Ron Hoague #13224  
**Arresting Officer Agency:** St. Albans Police Department

**Date Examined / Location:** 08/15/14  
**Police Dept.:** Colchester PD  
**Session #:** XV - #5  
**Instrument #:** 45401  
**Chemical Test:** No  
**Blood:** X

**Miranda Warning Given:** Yes  
**What have you eaten today?** Veggie burger  
**What have you been drinking?** “Just water”  
**Time of last drink?** N/A

**Officer's Signature:**

**Recorder:**

**Date Examined:** 08/15/14  
**Police Dept.:** Colchester PD  
**Session #:** XV - #5  
**Instrument #:** 45401  
**Chemical Test:** No  
**Blood:** X

**Miranda Warning Given:** Yes  
**What have you eaten today?** Veggie burger  
**What have you been drinking?** “Just water”  
**Time of last drink?** N/A

**Officer's Signature:**

**Officer Hoague:**  
**St. Albans Police Department**

---

**Time Now / Actual:** 9 pm / 2015  
**When did you last sleep?** I don’t remember”  
**Are you sick or injured?** No  
**Are you diabetic or epileptic?** No

**Are you under the care of a doctor or dentist?** No  
**“Just some herbal vitamins”**  
**Attitude:** Disoriented, Cooperative  
**Coordination:** Poor

**Speech:** Incoherent at times, Rambling  
**Breath Odor:** Normal  
**Flushed, Sweaty**

**Corrective Lenses:** None  
**Glasses:** No  
**Contacts, if so:** No  
**Hard:** No  
**Soft:** Yes

**Pupil Size:** Equal  
**Unusually (explain):**

**Pulse and Time:**

<table>
<thead>
<tr>
<th>Time</th>
<th>106 / 2025</th>
<th>102 / 2038</th>
<th>104 / 2055</th>
</tr>
</thead>
</table>

**Modified Romberg Balance:**

1. Walk and Turn Test
   - Lack of Smooth Pursuit
   - None
   - None
   - None
   - None
   - None
   - None
   - None

2.(internal clock estimated as 30 seconds)

- Finger to Nose
  - Made a walking turn.
  - Cannot do test (explain)

**Blood pressure:** 166 / 98  
**Temperature:** 101 °

**Muscle tone:** Rigid  
**Flaccid**  
**Nothing observed.**

**Rebound Dilation:**

- Pupillary Unrest
- Reaction to Light: Normal

**Type of footwear:** Flip-Flops

**Room Light:**

<table>
<thead>
<tr>
<th>PUPIL SIZE</th>
<th>2.5 - 5.0</th>
<th>5.0 - 8.5</th>
<th>2.0 - 4.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Eye</td>
<td>7.0</td>
<td>9.5</td>
<td>8.0</td>
</tr>
<tr>
<td>Right Eye</td>
<td>7.0</td>
<td>9.5</td>
<td>8.0</td>
</tr>
</tbody>
</table>

**Nasal area:** Clear  
**Oral cavity:** Clear

**Officer's Signature:**

---

**Officer Hoague:**  
**St. Albans Police Department**

---

**DRE #:** Reviewed/approved by / date:

---

**Opinion of Evaluator:**

- Not Impaired
- Alcohol
- Medical
- CNS Stimulant
- Dissociative Anesthetic
- Inhalant
- Narcotic Analgesic
- Cannabis

Rev 07/13
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Edwards, Joan

1. LOCATION: The evaluation was conducted at the Colchester Police Department.

2. WITNESSES: Lt. John Flannigan from the VT State Police recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to conduct a drug evaluation for Officer Hoague at the Colchester PD. Officer Hoague advised me that the suspect had been sitting on the hood of her vehicle near I-89 South waving her arms, and screaming at vehicles as they passed by. It was determined that she had driven her vehicle to that location after attending a concert in Canada earlier in the day. She was suspected of being under the influence of drugs, and was administered SFST’s which she had difficulty completing and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at CPD. She appeared dazed, disoriented and had difficulty standing.

6. MEDICAL PROBLEMS AND TREATMENT: Suspect stated she felt sick to her stomach and felt like “throwing-up,” but did not require medical assistance.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Suspect had an approximate 1” front to back and 3” side to side sway. She estimated 30 seconds in 62 seconds. Walk & Turn: Suspect lost her balance once during the instructions stage, twice started the test too soon, missed touching heel to toe on all her steps, and used her arms for balance six times. She made an improper turn by walking around using both feet. One Leg Stand: Suspect put her foot down three times on each foot. The test was stopped for safety reasons after she nearly fell. Finger to Nose: The suspect missed the tip of her nose on all six attempts.

8. CLINICAL INDICATORS: The suspect’s pulse, blood pressure and temperature were elevated and above the DRE average ranges. Her pupils were dilated in all lighting levels.

9. SIGNS OF INGESTION: None were evident.

10. SUSPECT’S STATEMENTS: Suspect admitted taking “something” while at the concert that made her feel weird. Some friends gave it to her and she didn’t know what it was.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a ____________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:

Rev. 10/15
Participant Manual

Drug Recognition Expert Course

Session 16
Dissociative Anesthetics

100 Minutes
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Learning Objectives

- Explain a brief history of Dissociative Anesthetics and specifically PCP and its analogs
- Identify common drug names and terms associated with this drug category
- Identify common methods of administration for this drug category
- Describe the symptoms, observable signs and other effects associated with this drug category
- Describe the typical time parameters associated with this drug category
- List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of this drug category
- Correctly answer the “topics for study” questions at the end of this session

Upon successfully completing this session the participant will be able to:

- Explain a brief history of Dissociative Anesthetics and specifically PCP and its analogs.
- Identify common drug names and terms associated with this drug category.
- Identify common methods of administration for this drug category.
- Describe the symptoms, observable signs and other effects associated with this drug category.
- Describe the typical time parameters associated with this drug category
- List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of this drug category
- Correctly answer the “topics for study” questions at the end of this session

CONTENT SEGMENTS ................................................................. LEARNING ACTIVITIES
A. Overview of Dissociative Anesthetics ........................................... Instructor-Led Presentations
B. Possible Effects of Dissociative Anesthetics ................................. Review of DEC Exemplars
C. Onset and Duration of Effects ......................................................... Reading Assignments
D. Signs and Symptoms of Dissociative .............................................. Slide Presentations
E. Anesthetics Overdose
F. Expected Results of the Evaluation
G. Classification Exemplars
A. Overview of Dissociative Anesthetics

Dissociative Anesthetics include drugs that inhibit pain by cutting off or disassociating the brain’s perception of pain. The drugs within this category normally will induce a state of sedation, immobility, amnesia and marked analgesia.
Phencyclidine (PCP)

Phencyclidine or PCP, is a drug that, along with its analogs, are examples of this distinct drug category.

The chemical for PCP is Phenyl Cyclohexyl Piperidine.

PCP shares some characteristics with each of the three categories of drugs.

It produces some effects that are similar to the effects of CNS Depressants.
  • Examples of effects PCP shares with Depressants: Nystagmus, slurred speech, slowed responses.

It produces some effects that are similar to those of CNS Stimulants.
  • Examples of effects PCP shares with CNS Stimulants: elevated vital signs and restlessness.

In some respects it acts like a Hallucinogen.
Phencyclidine was first developed in the late 1950’s. It was developed by Parke-Davis and Company, a leading pharmaceutical firm.

- The developers were searching for a drug that would serve as an efficient intravenous anesthetic.
- PCP proved to be a very effective anesthetic.
- An anesthetic is an agent that reduces or abolishes pain sensitivity.
- It was patented and marketed in 1963 under the trade name Sernyl.
- It was used in the treatment of mental and psychological disorders, including schizophrenia.
- Many adverse side effects were experienced by persons who had been treated with PCP.
- In 1967, use of Phencyclidine as an anesthetic for humans was discontinued.
- In 1968, Parke-Davis re-patented PCP under the trade name Sernylan, which was restricted to use as a veterinary anesthetic.
- Sernyl for animals = Sernylan.
- However, Sernylan was often illicitly diverted to “street” use, so most legitimate manufacturing of PCP was stopped in 1978.
PCP is relatively easy to manufacture.

- The chemicals required to produce it are readily available commercially.
- The formula for producing PCP has been widely publicized.
- The hardware needed to combine the chemicals is very basic.
Methods of Ingestion: PCP

- Many users ingest PCP by smoking.
- PCP can be applied in either powder or liquid form to a variety of vegetable or leafy substances, which can then be smoked in a pipe or homemade cigarette.
- Popular substances include mint leaves, parsley, oregano, tobacco, or marijuana.
- Commercially prepared cigarettes can also be dipped in liquid PCP, allowed to dry and then smoked.
- Some users prefer to dip a string in liquid PCP, and then insert the string into a tobacco cigarette.

White cigarette paper will be stained brown if adulterated with PCP. Brown cigarette paper will show white crystals, when adulterated.
PCP and Analogs
Methods of Ingestion
• Insufflation (inhaling; snorting)
• Orally
• Injection
• Eyedropper
• Transdermal absorption

PCP can also be insufflated or “snorted.”

It can also be taken orally, in capsule or tablet form.

Some users inject liquid PCP, either directly into a vein, under the skin or into a muscle.

Some users have administered PCP to themselves by dripping liquid PCP onto their eyes, using an eyedropper.

Transdermal absorption of PCP has also been reported (i.e. when applied to the skin, especially as a liquid, PCP can penetrate directly into the body and bloodstream).

**Liquid PCP is especially dangerous because it can be absorbed through the skin. Hence, it could be used as a weapon.**
Ketamine

Another drug in this category is called Ketamine. It continues to be manufactured and sold legitimately.

Ketamine is a white, crystalline powder or clear liquid.

Ketamine is used as a rapid surgical anesthetic, both for animals and humans, especially children.

- Some brand names of Ketamine: Ketalar (human use), Ketaset, Ketavet, Vetalar and Vetamine (veterinary use).
- Ketamine is being studied as a possible treatment of depression.
- Methoxetamine – a research chemical not currently approved for human or veterinary use. Methoxetamine has a similar abuse profile to Ketamine, and can cause pain suppression, tachycardia, hypertension, and altered perception and memory. Signs and symptoms include dissociated and catatonic state, nausea, vomiting, and visual hallucinations.


Ketamine street names include “K,” “Special K,” “Vitamin K,” “Jet” and “Super acid.”
Methods of Ingestion

Ketamine can be applied in either powder or liquid form to a variety of vegetable or leafy substances, which can then be smoked in a pipe or homemade cigarettes.

Popular substances include mint leaves, parsley, oregano, tobacco, or marijuana.

Commercially prepared cigarettes can also be dipped in liquid Ketamine, allowed to dry and then smoked.

Some users prefer to dip a string in liquid Ketamine, and then insert the string into a tobacco cigarette.
Dextromethorphan (DXM)

Another drug in this category is Dextromethorphan. It is sometimes referred to as “DXM” and is an ingredient found in numerous over-the-counter cough and cold remedies.

- Point out that DREs frequently encounter persons abusing DXM due to it’s availability in so many over-the-counter products.
- Point out in some respects, DXM’s effects can be similar to a CNS Depressant, CNS Stimulant, and Hallucinogen. It has been classified as a CNS Depressant in some medical texts and scientific/ research reports.
- Point out that DXM is often in other over-the-counter substances containing Acetaminophen, Chlorpheniramine, and Guaifenesin.
- DXM is a synthetically produced substance that is chemically related to Codeine, although it is not an opiate.
- When ingested in recommended dosage levels, DXM generally is a safe and highly effective cough suppressant; however, when ingested in large amounts, it produces negative physiological effects.
- DXM abusers normally ingest the drug orally, although some snort
- Some abusers ingest 250 to 1,500 milligrams in a single dosage.
Street names for Dextromethorphan include:

- Triple C
- Robo
- Robo-Tripping
- Skittles
- Robo-dosing
- Robo-fire
- Rojo
- Candy
- Velvet
- DM

Methods of ingesting Dextromethorphan include:

- Orally
- Injection
- Insufflation (snorting)
B. Possible Effects of Dissociative Anesthetics

Possible effects of PCP and other Dissociative Anesthetics may include the following adverse side effects (Source: Drug ID Bible, 2012):

- Delirium: confusion, incoherent speech, excitement, illusions, hallucinations, and disorientation.
- Agitation, anxiety
- Rigid muscle tone
- Elevated blood pressure
- Convulsions: involuntary contortion of the muscles, producing contortion of the body and limbs.
- Difficulty in speech
- Hallucinations
- Violent reactions

Some lingering and long term effects were also noted.

- Some patients complained of dizziness for several hours after their attention and consciousness appeared to be cleared of PCP’s effects.
- Some patients report memory disorders and other psychological disorders resembling schizophrenia for several months and even years afterwards.

PCP has sometimes been called a psychotomimetic drug; i.e. it produces effects that mimic psychosis, or “craziness.” When the psychosis remains long after the drug has dissipated, we say that its effects were psychotogenic, i.e. it didn’t simply mimic craziness, it caused craziness.

PCP is classified as a Dissociative Anesthetic, because it cuts off the brain’s perceptions of the senses.

- PCP users often feel that their heads are physically separated from their bodies.
- They sometimes report feeling they are dead, and that their heads are floating away.
Cases of terribly bizarre, self-destructive behavior have been reported with persons under the influence of PCP.

- One young man methodically pulled his own teeth out, using a pair of pliers.
- Point out that PCP can render the user impervious to pain. It anesthetizes the central nervous system to the extent that surgery could be performed on the user while he or she is wide awake.
- Another individual suffered hallucinations of unbelievably grotesque monsters, and gouged out his own eyes to avoid seeing the monsters.
- Another young man drank rat poison, attempting to kill rats that he imagined were inhabiting his body.
- A nude woman plunged a butcher knife into her own eye, chest, groin and abdomen. She then threatened a police officer with the knife and was shot to death.

C. Onset and Duration of Effects

PCP

• When PCP is smoked or injected, onset occurs within 1 – 5 minutes.
• When inhaled ("snorted") onset occurs in 2 – 3 minutes.
• Onset is considerably slower when PCP is taken orally: 30 – 60 minutes.
• The effects reach their peak in about 15 – 30 minutes, assuming the PCP was smoked, injected or snorted.
• The effects generally last 4 – 6 hours, but they can go somewhat longer.
• The user usually, but not always returns to normal within 24 – 48 hours.

Onset and Duration of Effects

Ketamine

• Within seconds if smoked; duration varies.
• 1 – 5 minutes if injected; lasting 30 – 45 minutes.
• 5 – 10 minutes if snorted; lasting 45 – 60 minutes.
• 15 – 20 minutes if orally; lasting 1 – 2 hours.
Onset and Duration of Effects for Dextromethorphan (DXM)

- Rapidly absorbed from the gastrointestinal tract
- Peak plasma concentration is reached in approximately 2.5 hours
- Expect antitussive effects in 15 – 30 minutes
- Duration of effects is approximately 3 – 6 hours

Dextromethorphan

- Rapidly absorbed from the gastrointestinal tract and peak plasma concentrations are reached in approximately 2.5 hours.
- DXM is widely distributed and is rapidly and extensively metabolized by the liver.
- DXM exerts its antitussive effects within 15 – 30 minutes of oral administration. The duration of action is approximately 3 – 6 hours with conventional dosage forms.
**DXM Plateau (or effect)**

Abusers will also ingest various amounts of DXM depending on their body weight and the effect or “plateau” that they are attempting to achieve. Plateau’s include:

1st Plateau: Mild inebriation.

2nd Plateau: An effect similar to alcohol intoxication with mild hallucinations.

3rd Plateau: An altered state of consciousness where the abuser’s senses, particularly vision, can become impaired.

4th Plateau: Mind and body dissociation or an “out of body” experience.

Other effects include: blurred vision, body itching, rash, sweating, fever, hypertension, shallow respiration, diarrhea, toxic psychosis, and an increased heart rate, blood pressure and body temperature.

Acute dose between 250 – 1500 mg.
D. **Signs and Symptoms of Dissociative Anesthetic Overdose**

In addition to the bizarre, violent and self-destructive behavior discussed previously, persons severely intoxicated by Dissociative Anesthetics may exhibit definite and extreme symptoms signifying a medically dangerous condition.

- A deep coma, lasting up to 12 hours.
- Seizures and convulsions.
- A danger associated with severe Dissociative Anesthetics intoxication is that the person may die due to respiratory depression.
- There is also some evidence that Dissociative Anesthetics may trigger a heart attack, if the user had some pre-existing condition disposing him or her to possible cardiac problems.
- Eyes generally open with a blank stare.

There is also some evidence that prolonged use of Dissociative Anesthetics can lead to psychosis, which can be permanent.
E. Expected Results of the Evaluation

- Horizontal Gaze Nystagmus generally will be present with a very early angle of onset.
- Vertical Gaze Nystagmus usually will be present.
- Lack of convergence will generally be present.
- Performance on Modified Romberg Balance will be impaired: internal clock may be slowed.
- Performance on Walk and Turn, One Leg Stand, and Finger to Nose will be impaired: muscle tone will usually be rigid.

With PCP, the subject may exhibit a “high gait ataxia” (unsteady, uncoordinated walk) or “moon walking,” i.e. taking abnormally high and slow steps, as though he or she were trying to step over obstacles in his or her path.
Vital Signs

• Pulse rate will generally be up
• Blood pressure will generally be elevated.
• Body temperature will generally be up.

Dark Room

• Pupil size will be within the DRE average ranges.
• Reaction to light will be normal.
General Indicators

Subjects Under the Influence of Dissociative Anesthetics

- Blank stare
- Confused
- Chemical odor (PCP)
- Cyclic behavior (PCP)

General Indicators

- Difficulty with speech
- Disoriented
- Early HGN angle of onset
- Hallucinations
- Incomplete verbal responses
- Non-Communicative
- Perspiring (PCP)
- Sensory distortions
- Possibly violent
- Slurred and repetitive speech
- Warm to touch (PCP)
- Loss of Memory

General Indicators

- Blank stare
- Confused
- Chemical odor (PCP)
- Cyclic behavior (PCP)
- Difficulty with speech
- Disoriented
- Early HGN angle of onset
- Hallucinations
- Incomplete verbal responses
- Non-Communicative
- Perspiring (PCP)
- Sensory distortions
- Possibly violent
- Slurred and repetitive speech
- Warm to touch (PCP)
- Loss of Memory
### Dissociative Anesthetic Symptomatology Chart

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGN</td>
<td>Present</td>
</tr>
<tr>
<td>VGN</td>
<td>Present</td>
</tr>
<tr>
<td>Lack of Convergence</td>
<td>Present</td>
</tr>
<tr>
<td>Pupil Size</td>
<td>Normal</td>
</tr>
<tr>
<td>Reaction to Light</td>
<td>Normal</td>
</tr>
<tr>
<td>Pulse Rate</td>
<td>Up</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Up</td>
</tr>
<tr>
<td>Temperature</td>
<td>Up</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Rigid</td>
</tr>
</tbody>
</table>

### Summary

- Expected Results of the Evaluation. **“Normal” for pupil sizes refers to within the DRE average ranges.**

- Point out that as with other drug categories, DREs should not specify the exact drug such as PCP, Ketamine or DXM.

- When a DRE concludes that a subject is impaired by a Dissociative Anesthetic, such as PCP or DXM, the report should state that “the subject is under the influence of a Dissociative Anesthetic.”
F. Classification Exemplar

Point out that as with other drug categories, DREs should not specify the exact drug such as PCP, Ketamine or DXM.
TOPICS FOR STUDY

1. What was the original purpose for which PCP was first patented and marketed?

2. Why do many PCP smokers prefer to adulterate mentholated cigarettes with PCP?

3. What is Ketamine?

4. What does the term “dissociative anesthetic” mean?

5. “Phencyclidine” is a contraction of what three words?
**DRUG INFLUENCE EVALUATION**

**Evaluator:** Sr. Cpl. Larry Allen  
**Dallas PD**  
**DRE #:** 6072  
**Rolling Log #:** 14-04-123  
**Case #:** 14-77654  
**Session XVI - #1**

**Recorded Witness:**  
**Sgt. Matthew Dusek**  
**Dallas DPS**

**Arresting Officer (Name, ID#):**  
**Officer Stephen Burress #18470**

**Date Examined:** 04/07/14  
**Time:** 1620  
**Location:** Irving PD

**Breath Results:** Test Refused  
**Chemical Test:** Urine  
**Results:** 89015  
**Instrument #:**

**Witness:**  
**Sgt. Matthew Dusek**  
**Dallas DPS**

**Date of Birth:** 11/02/50  
**Sex:** M  
**Race:** W

**Arresting Officer:**  
**Irving Police Department**

**Evaluator DRE #:** I  
**Ko1mg Log #:** Gase#  
**Sr. Cpl. Larrv Allen Dallas PD 6072 14-04-123 14-77654 Session XVI - #1**

**Recorder:**

**Witness:**  
**Sgt. Matthew Dusek**  
**Dallas DPS**

**Arresting Officer:**  
**Irving Police Department**

**Date Examined:** 04/07/14  
**Time:** 1620  
**Location:** Irving PD

**Breath Results:** Test Refused  
**Chemical Test:** Urine  
**Results:** 89015  
**Instrument #:**

**Witness:**  
**Sgt. Matthew Dusek**  
**Dallas DPS**

**Arresting Officer:**  
**Irving Police Department**

**Date Examined:** 04/07/14  
**Time:** 1620  
**Location:** Irving PD

**Breath Results:** Test Refused  
**Chemical Test:** Urine  
**Results:** 89015  
**Instrument #:**

**Witness:**  
**Sgt. Matthew Dusek**  
**Dallas DPS**

**Arresting Officer:**  
**Irving Police Department**

**Date Examined:** 04/07/14  
**Time:** 1620  
**Location:** Irving PD

**Breath Results:** Test Refused  
**Chemical Test:** Urine  
**Results:** 89015  
**Instrument #:**

**Witness:**  
**Sgt. Matthew Dusek**  
**Dallas DPS**

**Arresting Officer:**  
**Irving Police Department**

**Date Examined:** 04/07/14  
**Time:** 1620  
**Location:** Irving PD

**Breath Results:** Test Refused  
**Chemical Test:** Urine  
**Results:** 89015  
**Instrument #:**
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Dexing, Delbert R.

1. LOCATION: The evaluation was conducted at the Irving Police Department.

2. WITNESSES: Sergeant Matthew Dusek of the Texas DPS recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was on-duty and requested to contact Officer Burress of the Irving PD for a drug evaluation. Officer Burress advised that he had stopped the suspect for speeding and for following other vehicles too closely. Officer Burress noted that the suspect had bloodshot eyes, slurred speech, and appeared to be impaired. The suspect had six clues of HGN, but no odor of an alcoholic beverage was detected on his breath. He performed poorly on the SFST’s and was arrested for DUI. He admitted taking some cold medicine earlier in the evening.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at IPD. His face was flushed, and his speech was slurred. His movements were slow and deliberate. He seemed disoriented and confused, and had poor balance.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated by the suspect.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 3” front to back and side to side, and estimated 30 seconds in 28 seconds. Walk & Turn: Suspect lost his balance twice during the instructions, stopped while walking three times, missed touching heel to toe twice, raised his arms for balance six times, and turned by taking rigid steps with both feet. One Leg Stand: The suspect swayed while balancing, used his arms for balance, and was very rigid. Finger to Nose: Suspect missed the tip of his nose on four of the six attempts, and had slow, rigid hand and arm movements.

8. CLINICAL INDICATORS: HGN was present with an immediate onset. Vertical Gaze Nystagmus and Lack of Convergence were also present. The suspect’s pulse rates, blood pressure and body temperature were all elevated and above the DRE average ranges.

9. SIGNS OF INGESTION: None were evident.

10. SUSPECT’S STATEMENTS: Suspect admitted taking about a dozen “red cold pills.”

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a Dissociative Anesthetic and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:
**DRUG INFLUENCE EVALUATION**

Evaluator: Officer Michael Boyls  
LAPD 13542 14-116 14-335259  
Session XVI - #2

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<thead>
<tr>
<th>Evaluator DRE#</th>
<th>Recorder</th>
<th>DRE#</th>
<th>Sex</th>
<th>Race</th>
<th>Arresting Officer Name</th>
<th>Arresting Officer ID#</th>
<th>Arresting Officer Agency</th>
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<tr>
<td>Officer Kamaron Sardar LAPD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>#10175</td>
<td>LAPD</td>
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<th>Time</th>
<th>Location</th>
<th>Breath Results</th>
<th>Test Refused</th>
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<td>Metro Detention Center</td>
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<td>34310</td>
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<thead>
<tr>
<th>Miranda Warning Given</th>
<th>What have you eaten today?</th>
<th>When?</th>
<th>What have you been drinking?</th>
<th>How much</th>
<th>Time of last drink?</th>
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<tr>
<td>Yes</td>
<td>&quot;Sandwich&quot; &quot;Noon&quot;</td>
<td></td>
<td></td>
<td></td>
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<th>Arresting Officer Name</th>
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<tr>
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<td>14-116</td>
<td>F</td>
<td>W</td>
<td>Helen Pallares</td>
<td>#10175</td>
<td>LAPD</td>
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<thead>
<tr>
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<th>Comment</th>
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<tr>
<td>Michael Bays LAPD</td>
<td>Slow, Thick, Confused</td>
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<table>
<thead>
<tr>
<th>Officer's Signature</th>
<th>Near Vision</th>
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<tbody>
<tr>
<td>Michael Bays LAPD</td>
<td>Poor, Slow</td>
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<thead>
<tr>
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<th>Chemical-like</th>
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<tr>
<td>Michael Bays LAPD</td>
<td>Breath Odor:</td>
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<table>
<thead>
<tr>
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<th>Drugs or Medications</th>
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<tr>
<td>Michael Bays LAPD</td>
<td>Yes</td>
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<table>
<thead>
<tr>
<th>Officer's Signature</th>
<th>Precinct/Station</th>
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<td>LAPD</td>
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<th>Evaluation completion time</th>
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<tr>
<th>Officer's Signature</th>
<th>Report Approved by date</th>
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<td>05/02/14 2214</td>
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<th>Direct 2.0 - 4.5</th>
<th>Nasal area</th>
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<td>6.5</td>
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<th>Direct 2.0 - 4.5</th>
<th>Reaction to Light</th>
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<tr>
<td>Left Eye</td>
<td>4.0</td>
<td>6.5</td>
<td>3.5</td>
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<th>Darkness 5.0 - 8.5</th>
<th>Direct 2.0 - 4.5</th>
<th>Reaction to Light</th>
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<tbody>
<tr>
<td>Right Eye</td>
<td>4.0</td>
<td>6.5</td>
<td>3.5</td>
<td>Normal</td>
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<table>
<thead>
<tr>
<th>Rebound Dilation</th>
<th>Pupillary Unrest</th>
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</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
</tr>
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<table>
<thead>
<tr>
<th>Reaction to Light</th>
<th>Right Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reaction to Light</th>
<th>Left Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type of footwear</th>
<th>Lace-up boots</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>Temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>135 / 96</td>
<td>100 o</td>
</tr>
</tbody>
</table>

**Comments:**

- Slow, deliberate movements.
- Nothing detected or observed.

**What drugs or medications have you been using?**

- No response

**Where were the drugs used? (Location):**

- N/A

**How much?**

- N/A

**Time of use?**

- N/A

**Date/Time of arrest:**

- 05/02/14 2214

**Data/Time DRE was notified:**

- 05/02/14 2240

**evaluation start time:**

- 2310

**Evaluation completion time:**

- 2355

**Comments:**

- N/A

**Opinion of Evaluator:**

- Not Impaired

**Medical:**

- Alcohol

**Drug:**

- CNS Stimulant

**Hallucinogen:**

- Narcotic Analgesic

**Inhalant:**

- Cannabis

**Disassociative Anesthetic:**
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Sherms, Shelly

1. **LOCATION:** The evaluation was conducted at the LAPD Metro Detention Center.

2. **WITNESSES:** Officer Kamaron Sardar of the LAPD recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was requested to contact Officer Pallares for a drug evaluation. Officer Pallares advised that she had stopped the suspect after she nearly hit several parked vehicles along 4th Street. According to Officer Pallares, the suspect was slow to respond, and appeared dazed, and disoriented. Her speech was slow, thick, and slurred. She was very confused, and was not sure of her surroundings. She performed poorly on the SFSTs and was arrested for DUI.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the interview room at the Detention Center. She appeared dazed, disoriented and had a fixed stare. Her movements were very slow and rigid-like, and she was perspiring heavily.

6. **MEDICAL PROBLEMS AND TREATMENT:** None noted or stated.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: The suspect swayed approximately 3” in a front to back and side to side motion, and estimated the passage of 30 seconds in 42 seconds. Walk & Turn: Suspect lost her balance twice during the instructions stage, stopped walking five times, missed heel to toe five times, and raised her arms for balance eight times. She also took the wrong number of steps, and was stiff and rigid throughout the test. One Leg Stand: Suspect lost her balance and used the wall to steady herself. The test was stopped for safety reasons. Finger to Nose: Suspect missed the tip of her nose on five of the six attempts. Her arm movements were slow and rigid.

8. **CLINICAL INDICATORS:** The suspect had six clues of HGN with an immediate angle of onset, and had VGN. She was unable to converge her eyes and looked straight ahead. Her pulse, blood pressure, and temperature were all elevated and above the DRE average ranges.

9. **SIGNS OF INGESTION:** Suspect had a strong chemical-like odor on her breath.

10. **SUSPECT’S STATEMENTS:** The suspect denied using any drugs.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of a Dissociative Anesthetic and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A blood sample was collected from the suspect.

13. **MISCELLANEOUS:**

**Rev. 10/15**
**DRUG INFLUENCE EVALUATION**

Evaluator: Sgt. Gerry Britt  |  Yarmouth PD
---|---
Recorder: Sgt. Don Decker  |  Nahant PD
Arrestee's Name: Krystal, K. J.

Date Examined: 09/28/14  |  Time Location: 2145 Middleboro PD

Officer's Signature: Sgt. Gerry Britt  |  Yarmouth PD

**Modified Romberg Balance**

<table>
<thead>
<tr>
<th>Pulse and time</th>
<th>HGN</th>
<th>Right Eye</th>
<th>Left Eye</th>
<th>Convergence</th>
<th>Left Count</th>
<th>Right Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>98 / 2150</td>
<td>Lack of Smooth Pursuit</td>
<td>Present</td>
<td>Present</td>
<td>Cannot keep balance</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>98 / 2202</td>
<td>Maximum Deviation</td>
<td>Present</td>
<td>Present</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>100 / 2215</td>
<td>Angle of Onset</td>
<td>Immid</td>
<td>Immid</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Walk and Turn Test**

- 2" 2" 2"
- Walked stiff legged.
- Slow and rigid movements.
- Breath Odor: Normal
- Type of footwear: N/A

**Blood pressure**

<table>
<thead>
<tr>
<th>Room Light</th>
<th>Darkness</th>
<th>Direct</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5 - 5.0</td>
<td>5.0 - 8.5</td>
<td>2.0 - 4.5</td>
</tr>
<tr>
<td>Left Eye</td>
<td>Right Eye</td>
<td></td>
</tr>
<tr>
<td>4.5</td>
<td>4.5</td>
<td></td>
</tr>
<tr>
<td>7.0</td>
<td>7.0</td>
<td></td>
</tr>
<tr>
<td>4.0</td>
<td>4.0</td>
<td></td>
</tr>
</tbody>
</table>

**Comments:**

- Nothing detected.
- No drugs detected.
- N/A for location of use.

**Type of Footwear:** N/A

**Nasal area:** Clear

**Oral cavity:** Clear

**Reaction to Light:** Normal

**Ear:** Normal

**Eye:** Normal

**Muscle tone:** Normal

**Opinion of Evaluator:** Not Impaired

**Officer's Signature:** Reviewed/approved by / date.

**Case #:** 14-33598
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: **Krystal, K.J.**

1. **LOCATION:** The evaluation was conducted at the Middleboro PD Booking Room.

2. **WITNESSES:** Sgt. Don Decker of the Nahant PD recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was requested to contact Sgt. Batista for a drug evaluation. It was determined that Sgt. Batista had stopped the suspect after observing his vehicle fail to stop at a red light, nearly hitting another vehicle. According to Sgt. Batista, the suspect was disoriented, and at times non-responsive. His speech slow and thick. At times he would stop talking while in the middle of a sentence. The suspect had difficulty performing the SFSTs and was arrested for DUI.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the booking room at the PD. He appeared disoriented and had a fixed stare. His movements were very slow and deliberate. Several times he used a chair to steady himself when he stood.

6. **MEDICAL PROBLEMS AND TREATMENT:** None noted or stated.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: The suspect had an approximate 2” front to back and side to side sway. He estimated the passage of 30 seconds in 38 seconds. Walk & Turn: Suspect lost his balance once during the instruction stage, stopped walking six times, missed heel to toe five times, and raised his arms for balance during the entire test. The suspect stopped at the turn and had to be reminded what to do. One Leg Stand: Suspect swayed, and put his foot down three times on each attempt. He nearly fell several times and the test was stopped for safety reasons. Finger to Nose: Suspect missed the tip of his nose on all six attempts. His arm movements were slow and rigid.

8. **CLINICAL INDICATORS:** The suspect exhibited six clues of HGN with an immediate angle of onset. VGN and Lack of Convergence were present. His pulse rates, blood pressure, and body temperature were all elevated and above the DRE average ranges.

9. **SIGNS OF INGESTION:** Nothing observed or detected.

10. **SUSPECT’S STATEMENTS:** The suspect did not respond when asked about drug use.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of a Dissociative Anesthetic and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A urine sample was collected from the suspect.

13. **MISCELLANEOUS:**

Rev. 10/15
Session 17

Narcotic Analgesics
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Upon successfully completing this session the participant will be able to:

- Explain a brief history of the Narcotic Analgesic category of drugs.
- Identify common drug names and terms associated with this category.
- Identify common methods of administration for this category.
- Describe the symptoms, observable signs and other effects associated with this category.
- Describe typical time parameters, i.e. onset and duration of effects, associated with this category.
- List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of this category of drugs.
- Describe the procedures for examining and determining the ages of injection sites.
- Correctly answer the “topics for study” questions at the end of this session.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES

A. Overview of the Category ............................................................. Instructor-Led Presentations
B. Possible Effects ........................................ Review of Drug Evaluation; Classification Exemplars
C. Onset and Duration............................................................... Reading Assignments
D. Overdose Signs and Symptoms.................................................. Video Presentations
E. Expected Results of the Evaluation............................................. Slide Presentations
F. Injection Site Examination
G. Expected Location of Injection Marks
H. Conclusion
I. Classification Exemplar
A. Overview of the Category

Narcotic Analgesics

The term “Opioid,” however, most correctly refers to the synthetic subcategory of Narcotic Analgesics.

Narcotic Analgesic Defined

A medical term, not a legal or police term.

An “Analgesic” is a medication or drug that relieves pain. It differs from an anesthetic, in that it lowers one’s perception or sensations of pain, rather than stopping nerve transmission.

Non-Narcotic Analgesics, such as Aspirin, Tylenol, and Motrin, relieve pain, but do NOT produce narcosis, which means numbness or sedation.

Clarification: non-Narcotic Analgesics relieve pain, but do not alter mood. Therefore, they, in small amounts, are not psychoactive and are not abused for their mind or mood altering actions.

A Narcotic is a drug derived from Opium, or produced synthetically that relieves pain, but also induces euphoria, alters mood, and produces sedation.
There are two subcategories of Narcotic Analgesics:

- Opiates
- Synthetics

Opiates: drugs that either contain or are derived from Opium.
Natural alkaloids of Opium.
The term “main ingredient” can be used as a synonym for “alkaloid.”

*The Natural Alkaloids*
Alkaloids and the Opium derivatives all come from Opium, which is sap from the seed pods of a particular type of poppy.

*The Opium poppy is also called “papaver somniferum” (somniferum in Latin means “carrier of sleep”)*

*Opium Derivatives*
Opium derivatives are obtained by chemically treating the Opium alkaloid. Opium derivatives are therefore derived from Opium.

*Synthetics*
Synthetics, which do not derive from Opium at all, have similar or identical effects as Opium alkaloids and derivatives.
Narcotic Analgesics all share three characteristics:

- They all relieve pain.
  Clarification: They produce analgesia.

- They will produce withdrawal signs and symptoms when the user is physically dependent, and drug use is stopped.
  Clarification: Physical dependence results from “chronic administration.” This means that the drug has been taken at fairly regular intervals for a period of time.

- They will suppress the withdrawal signs and symptoms of chronic Narcotic Analgesic administration.
  Clarification: This means that the various Narcotic Analgesics can be substituted for each other to relieve withdrawal symptoms.

Morphine is typically used as the standard for comparison with other Narcotic Analgesics.
Some Commonly Abused Opiates

**Powdered Opium**

Powdered Opium (also known as smoking Opium).

A simple refinement of raw Opium.

Used medically to treat diarrhea (administered orally).

The development of more effective opiates and synthetics has virtually eliminated its use medically. In recent years, there has been little street use of Opium. It is important to realize, however, that drug use trends can and do change.

Remains popular as a drug of abuse (smoked) among some Asian-American communities.

**Morphine**

Morphine, the principal natural alkaloid of Opium.

Morphine was first isolated from Opium in 1805.

Used medically to suppress severe pain (e.g., with terminal cancer patients).

Highly addictive.

Morphine was widely used during the Civil War. Morphine addiction was termed “Soldier’s disease.”

At one time, Morphine was the most commonly abused Narcotic Analgesic.
**Codeine**

Codeine is another natural alkaloid of Opium.

Its technical name is Methylmorphine.

First isolated in 1832.

Codeine’s pain killing ability is much weaker than Morphine’s.

Used medically to suppress coughing or minor pain.

Clarification: Narcotic Analgesic addicts often turn to Codeine when they cannot get more popular drugs.

Codeine is definitely an addictive drug.

---

**Heroin**

Heroin is the most commonly abused illicit Narcotic Analgesic.

Derived from Morphine in 1874.

Heroin was first thought to be a non-addictive substitute for Morphine.

It was approved for general use by the American Medical Association in 1906.

By the 1920’s it was evident that Heroin was much more addictive than Morphine.

Importation and manufacture of Heroin have been illegal in this country since 1925.

Heroin is a Schedule I drug, which means it has no legitimate medical uses in the United States.

---

**Dilaudid**

Dilaudid is another derivative from Morphine.

Technical Name: Hydromorphone Hydrochloride.

First produced in 1923.

Sometimes called “drug store Heroin,” since it is commercially available from medical and pharmaceutical sources.

Dilaudid has the same addictive liabilities as does Heroin or Morphine.

Used medically for short term relief of moderate to severe pain, and to suppress severe, persistent coughs.
Can be ingested via injection, orally or in suppositories. Sometimes abused by addicts who are unable to obtain Morphine or Heroin.

---

**Hydrocodone**

Hydrocodone is derived from Codeine but is more closely related to Morphine in its pharmacological profile.

Examples include:

- Hycodan
- Vicodin (*Vicodin is a commonly prescribed pain reliever containing Hydrocodone and Acetaminophen.*)
- Lortab

---

**Thebaine**

An opiate alkaloid derived from opium.

Not used therapeutically.

Converted into several drugs including oxycodone and oxymorphone.

**Numorphan**

Technical Name: Oxymorphone.

Used medically for the relief of chronic pain.

Sold in ampules (injection) and in suppositories.

Previously (pre-1972) it was sold in tablets, and was a favorite substitute for Heroin among addicts; addicts now generally prefer Dilaudid as a Heroin substitute.

A derivative of Thebaine (source: “Disposition of Toxic Drugs and Chemicals in Man” 9th edition, R. Baselt)
**Oxycodone**

Oxycodone is a semi-synthetic narcotic produced by chemically treating Thebaine. It is somewhat less addictive than Morphine, but more than Codeine.

Two examples are:

Brand Name: OxyContin.

Percodan is one of the most commonly prescribed Narcotic Analgesics.

It is also produced under the brand name of “Percocet”, which is Percodan combined with Acetaminophen, such as Tylenol.

OxyContin is a controlled release tablet that contains large amounts of Oxycodone (10-160mg). Abusers learn to circumvent the slow release mechanism.

Street names: “Oxy”; “OC”; “Killer.”

---

**Buprenorphine**

Buprenorphine is a Thebaine derivative with powerful analgesia. As an analgesic it is about 25 to 40 times more potent than morphine (Source: “Disposition of Toxic Drugs and Chemicals in Man” 9th Edition, R. Baselt.)

It is an ingredient of the drug Suboxone.

Depending on the application form, buprenorphine is normally prescribed for the treatment of moderate to severe chronic pain (pain that has outlived its use to prevent injury and after three months. It is commonly used in the treatment of opioid addiction, much like methadone.

Buprenorphine hydrochloride is normally administered by intramuscular injection, intravenous infusion, via a transdermal patch, or as a sublingual (under the tongue) tablet. It is also used in the treatment of narcotic addiction.
Some Common Synthetic Opiates

Demerol

Demerol was first produced in 1939.

Technical Name: Meperidine.

Demerol is one of the most widely used Synthetic Opiates for relief of pain and for sedation. It is also one of the Narcotic Analgesics that is most frequently abused by medical personnel. Demerol is widely used as an analgesic in childbirth.

One medical advantage of Demerol is that it produces less respiratory depression than do other Narcotic Analgesics; thus, a fatal overdose is less likely with Demerol.

Medical literature sometimes indicates that Demerol does not cause pupillary constriction. Enforcement experience indicates to the contrary.
Methadone

Methadone was developed in Germany during World War II and first marketed in America in 1947.

Methadone was developed in Germany because of wartime shortages of Morphine.

Methadone’s effects are similar to Morphine’s, although they develop more slowly and last longer than do Morphine’s effects.

Methadone’s withdrawal symptoms are slower and milder than are Morphine’s.

Used extensively in “maintenance programs” as a substitute for Heroin for addicts undergoing therapy and treatment.

In theory, the daily dose of Methadone given to a Heroin addict allows the addict to function normally with no physical need for up to 24 hours. Methadone has a much longer duration of effects than Heroin and is not designed to be injected.

Methadone is also used medically to relieve moderate to severe pain, and to suppress coughing.
Fentanyl

A synthetic Narcotic Analgesic of high potency and short duration of action.

“Sublimaze” is one of numerous brand names for Fentanyl. It is a Schedule II drug. It is frequently found in overdose situations. For example, “Tango and Cash” and “Goodfellas,” which contained Fentanyl, were sold in New York City in 1990 as Heroin.

Many fatal overdoses occurred as a result.

First developed in 1963 as an intravenous anesthetic.

Legally produced as a pain killer and available in an injectable solution or transdermal patches.

The principal abused analog of Fentanyl is “3-methylfentanyl.”
Methods of Administration

Methods of administration of Narcotic Analgesics vary from one drug to another.

Some are commonly taken orally.

Some are smoked.

Some are snorted (taken intranasally).

Users have stated that the fear of contracting diseases, such as AIDS, from shared needles, has prompted them to either snort or smoke Heroin.

Some are often administered in suppositories. Medically, some Narcotic Analgesics may be administered transdermally or through the skin.

Fentanyl patches are often used for chronic pain.

Heroin and some others are usually taken by injection.
B. Possible Effects

As with nearly all drugs of abuse, the effects produced by Heroin or other Narcotic Analgesics depend on the tolerance that the user has developed for the drug.

People develop tolerance for Narcotic Analgesics fairly rapidly.

“Tolerance” means that the same dose of the drug will produce diminishing effects or conversely that a steadily larger dose is needed to produce the same effects.

A Narcotic Analgesic user who has developed tolerance and who is using his or her “normal” dose of the drug may exhibit little or no evidence of intellectual or physical impairment.

Impairment is more evident with new users, and with tolerant users who exceed their “normal” doses.

Clarification: the tolerant addict who has injected his or her “normal dose” of Heroin may appear to be much less impaired than an inexperienced user who had taken the same dose.
Observable Effects

Observable effects of Heroin and other Narcotic Analgesics.

Sedation – “On the Nod.”

The condition known as “on the nod” is a semiconscious state of deep relaxation.

The user’s eyelids become very droopy.

Their head will slump forward until the chin rests on the chest.

In this condition, the user usually can be aroused easily and will be sufficiently alert to respond to questions.
Other Effects

These effects may be dose-related, and most often occur with non-tolerant users.

- slowed reflexes
- slow and raspy speech
- slow, deliberate movements
- inability to concentrate
- slowed breathing
- skin cool to the touch
- possible vomiting
- itching of the face, arms or body
C. Onset and Duration of Effects

*Psychological Effects*

The psychological effects of Heroin begin immediately after the injection.

- A feeling of pleasure or euphoria.
- Relief from the symptoms of withdrawal.
- Relief from pain.
Onset and Duration of Effects

5-30 minutes: Onset of physical effects
• “On the nod”
• Poor motor coordination
• Depressed reflexes
• Slowed breathing

Physical effects usually are observable for up to 4-6 hours

Observable Signs

The observable signs will usually become evident within 5 – 30 minutes after the user has injected.

- User may nod head and move in and out of consciences
- User may display poor motor coordination, depressed reflexes, and slowed breathing

The effects will usually be observable for up to 4 – 6 hours.

As the drug wears off, withdrawal signs and symptoms start to develop until the addict user injects again.
As the effects of Heroin diminish, withdrawal symptoms begin.

- Aches
- Chills
- Insomnia
- Nausea

As with nearly all drugs, the withdrawal signs and symptoms are essentially the opposite of the “high” or intoxicated state.

Withdrawal signs start to become observable 8 – 12 hours following injection.

- Goose bumps (piloerection) on the skin
- Sweating
- Runny nose
- Tearing
- Vomiting
- Yawning

Withdrawal signs and symptoms closely resemble those of Influenza or the common cold.
These symptoms begin to intensify from 14 – 24 hours after injection, and may be accompanied by goose bumps (piloerection), slight tremors, loss of appetite and dilation of the pupils.

Approximately 24 - 36 hours after injection, the addicted user experiences insomnia, vomiting, diarrhea, weakness, depression and hot and cold flashes.

Withdrawal symptoms and signs generally reach their peak 2 – 3 days after injection:

- Muscular and abdominal cramps
- Severe tremors and twitching
- Elevated temperature
- Sharp loss of weight

The addicted user at this point is nauseated, gags, vomits and may lose 10 – 15 pounds within 24 hours.

The withdrawal syndrome continues to decrease in intensity over time, and is usually greatly reduced by the fifth day, disappearing in one week to 10 days. A common misconception regarding withdrawal from Narcotic Analgesics is that they may be fatal. In reality, however, although Narcotic withdrawal is extremely uncomfortable, it rarely, if ever proves fatal.
**D. Overdose Signs and Symptoms**

Narcotic Analgesics depress respiration.

In overdoses, the user’s breathing will become slow and shallow.

Death can occur from severe respiratory depression.

The danger of death is heightened by the fact that the addicted user may not know the strength of the drug he or she is taking.

Clarification: the percentage of pure Heroin in the sample the addict uses may be much higher than what the addict expects and is used to.

Other signs and symptoms of an overdose of a Narcotic Analgesic include clammy skin, convulsions and coma, blue lips and pale or blue body, extremely constricted pupils (unless there is brain damage, in which pupils may be dilated), recent needle marks, or perhaps a needle still in the user’s arm.

Narcotic Analgesic overdoses are sometimes treated by the administration of a Narcotic antagonist such as Narcan. A Narcotic antagonist works at neuron receptor sites, blocking or counteracting the effects of Narcotic Analgesics. In effect, these substances precipitate withdrawal. The short duration of effects produced by Narcotic antagonists, however, require continued medical monitoring of the user.
E. Expected Results of the Evaluation

Observable Evidence of Impairment

Neither Horizontal Gaze Nystagmus nor Vertical Gaze Nystagmus will be present. Eyes will not exhibit Lack of Convergence.

Psychophysical Tests

Performance on the Modified Romberg Balance Test will be impaired. Generally, the subject will appear drowsy, and will have a slow internal clock.

Performance on the Walk and Turn and One Leg Stand will be impaired, and will reflect the slow and deliberate movements caused by this category of drugs.

Performance on Finger to Nose will also be impaired. Generally, the subject will appear drowsy, possibly “on the nod,” and exhibit slow and deliberate movements.
**Vital Signs**

Pulse will be down.

Blood pressure will be down.

Body temperature will be down.

Muscle tone will be flaccid.

**Dark Room**

Pupil size generally will be constricted (below 3.0 mm in diameter).

Pupil reaction to light will be little or none visible.
General Indicators

- Constricted pupils (Miosis)
- Depressed reflexes
- Droopy eyelids (Ptosis)
- Drowsiness
- Dry mouth
- Euphoria
- Facial itching

Itching – caused by the release of Histamines

- Nausea
- “On the nod”
- Puncture marks
- Slowed reflexes
- Slow, low, raspy speech
- Slowed breathing
### Narcotic Analgesic Symptomatology Chart

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Description</th>
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<tbody>
<tr>
<td>HGN</td>
<td>None</td>
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<tr>
<td>VGN</td>
<td>None</td>
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<tr>
<td>Lack of Convergence</td>
<td>None</td>
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<tr>
<td>Pupil Size</td>
<td>Constricted</td>
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<tr>
<td>Reaction to Light</td>
<td>Little or None Visible</td>
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<tr>
<td>Pulse Rate</td>
<td>Down</td>
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<td>Blood Pressure</td>
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<tr>
<td>Temperature</td>
<td>Down</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Flaccid</td>
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</tbody>
</table>

### Symptomatology Chart
F. Injection Site Examination

Examination of subject’s injection sites can give many clues to their drug habits.

- The slang term for an injection site is a “mark.”
- Many drugs can be injected.
- The presence of injection sites doesn’t ensure the subject is under the influence of drugs. Examination of injection sites is just one of the twelve steps in the evaluation.
- Injection sites are a sign of drug abuse which may or may not be present.
- May be evidence of habitual use.
- The trauma to the skin, muscles and the blood is the basic concept of injection sites.
Drugs and medication are injected into the body in three ways:

* **Intramuscular**
  - Legal injections are usually Intramuscular.
  - Abbreviated as I/M
  - “Intramuscular” is defined as administering by entering a muscle.

* **Intravenous**
  - For medically drawing of blood or emergency medical procedures, the injection is made into a blood vessel (Intravenous). Veins are usually used. Arteries are deep, thus not lending themselves to injection.
  - Abbreviated as I/V
  - “Intravenous” defined as entering a vein.

* **Subcutaneous**
  - Subcutaneous means just under the skin.
  - Commonly referred to as “skin popping.”
The primary instrument for injection is the hypodermic syringe.

- It consists of a hollow needle, a Barrel (tube) and a plunger.
- Needles vary in size, with the primary variance being the inside diameter of the needle or the gauge.
- A 26 gauge needle is used by a diabetic.
- The greater the number the larger the gauge, the smaller the inside diameter of the needle.
- Most illegal drug users prefer a larger gauge needle.
- The hypodermic marks are smaller and are therefore, less noticeable making it more difficult for the DRE to see them.
The user’s equipment is commonly referred to as a “hype kit” or “works.”

- The kit contains a “cooker” which is any device such as a bottle cap, a metal spoon, etc., that is used to heat the drug with water to form an injectable solution. Other parts of the “kit” include:
  - A handle to hold the “cooker” over the flames.
  - Matches, lighters (primarily disposable, adjustable flame types) used to heat the substance in the “cooker.”
  - A tourniquet, which can be a rubber tubing, a tie, belt, etc. It is tied around the arm, above the injection site, to cause the vein to bulge or rise, thus making it easier to inject.
  - “Cottons” are the cotton balls or cigarette filters used to “purify” the drug. The user places the “cottons” into their cooker and draws the drug up through the cottons.
  - The cottons are saved for later use since they contain some of the drug.
As a DRE, you may be asked in court to describe the difference between a medical and non-medical injection site.

A medical injection is usually intramuscular

Some exceptions would be in a blood donation, an emergency or a lab test.

There may be multiple injections, if the technician is unable to find a vein during the first try. There may also be bruising near the site.

The injection mark for medical purposes can be described as:

- Clean
- No scarring or scabbing

Most intramuscular medical injections will not be evident during a DRE evaluation.

- Usually there will be only one mark and it will be larger than the typical non-medical injection.
- Medical injections are made with new, sterile needles.
Non-Medical Injection Site

- Non-Medical (illicit) mark is usually over a vein
- Usually multiple marks in various stages of healing
- Use of same needle over and over again causes them to be dull or barbed
- Injection sites may be jagged

The non-medical (illicit) mark is usually over a vein.

- There will usually be multiple marks in various stages of healing. It takes approximately two weeks for a “mark” to totally heal.
- For example, the Heroin addict will inject approximately four to six times each day (every four to six hours). Therefore, they will inject approximately 2,000 times in one year.
- Users frequently use the same needle over and over again. Thus making it become dull or barbed.
- Frequently the needles are carried in pockets or socks and the rubbing against clothing causes them to be dull or barbed.
- Since the used needles make it more difficult to pierce the skin and vein, the injection sites may be jagged.
- A barbed needle may tear the skin on the way in and on the way out.
- Use of old, dirty and shared needles cause the spread of infections and diseases such as AIDS.

ALWAYS WEAR PROTECTIVE GLOVES PRIOR TO CONDUCTING THE EXAMINATION.
Users may frequently use the same spot to inject, as an attempt to reduce their likelihood of detection.

The veins may become hard and thick from continuous injections and makes them difficult to find. This is an obstruction by a clot of coagulated blood shutting off the passage of blood.

- The technical term is “Thrombosed.”

After about 10 to 20 injections, a large sore forms causing the site to enlarge and bruise. Upon close examination, the site reveals there are numerous puncture wounds in the same area, overlapping each other.

- This is referred to as “tunnel” or “corn.”
Basic Principles of Puncture Healing

The healing is greatly retarded.

Any needle that punctures the skin leaves a scab. A scab is simply a crust formed by the drying of the discharge from the puncture.

Scab is the dried remains of blood, plasma (a cellular, colorless fluid part of the blood), lymph fluid (a thin fluid that bathes all the tissues of the body) and puss (a thick yellowish/greenish fluid that forms at an injection(s) site).

These dried remains fill the gap caused by the puncture of the skin. As the fluids dry they harden (clot and gel).

Users will sometimes peal a corner of a healing scab up and inject into that area then cover the injection site with the scab.

This injecting under a scab to hide multiple puncture wounds is referred to as “Trap Dooring.”

Puncture Healing Timetable

There are no exact timetables for wounds to heal, but there are some general guidelines.

- Chronic disease, poor nutrition and etc. retard the puncture healing process.
- Scabs develop within about 18 – 24 hours after a puncture.
- A general rule: when the scab first forms, it is bright red. With age, the color gets darker and darker.

After about 14 days a scab usually starts to peel or flake and then falls off. The skin under the scab is shriveled and is lighter in color than the surrounding tissue.
There is no exact science to classifying the age of puncture wounds. Some general guidelines are:

- **Fresh puncture wounds** are defined as under 12 hours after injection and will be a red dot and have an oozing appearance or blood crater with no scab formation.
- **Early puncture wound** is 12 – 96 hours (half day to 4 days) after injection. It will have a light scab, light bruise, reddened border and a crater appearance.
- **Late puncture wound** is 5 – 14 days old and will have a dark scab, dark bruise and the crater will flatten.
- **Healing puncture wound** is over 14 days. The scab will be flaking and falling off with shriveled light colored skin underneath.
Other Indicators of Injection Sites

In an attempt to hide puncture wounds, users may inject into tattoos.

Tattoos that are designed to hide puncture wounds are frequently colored and found on the inner arms.

- Tattooing also refers to dark carbon deposits that result from using a flame to “sterilize” a needle. Carbon deposits on the needle are then injected into the skin, causing a tattoo effect.

- A “track” is a hardened part of a vein where numerous injections have been administered. The entire vein becomes scarred and hardened and with time may no longer be able to inject into. The area becomes silvery-blue in color and raised. This is referred to as “silver streaks.”

- AS A GENERAL RULE: one inch of tracks indicates that approximately 50 – 100 separate injections have been administered in this area.
G. Expected Location of Injection Marks

Prior to conducting the injection site examination, always remember to wear gloves.

Injection sites may be located anywhere on the subject’s body.

Conduct a thorough, slow, methodical examination of the subject’s arms beginning with the left.

- Using a magnifying light or “ski light” examine the inner arm as it is extended with the palm facing you.
- Beginning at the bicep, slowly examine the arm. Document the findings of your examination.
- Ask the subject to contract the arm, grasping their shoulder. Starting at the wrist, slowly examine the arm to the elbow documenting the results.
- This forces the individual’s veins to protrude.
- Next examine the outer arm as it is extended palm facing downward. Start the examination at the shoulder moving to the wrist.
- Subject should extend and spread his/her fingers when examining the hands. Examine both sides of the hands, with particular attention to the areas between the fingers, under watch bands and rings.
- Conduct the entire procedure for the right side.
Ankles are a common injection area.

- Subject should be instructed to remove their shoes and socks to allow the DRE to examine them for puncture wounds.

- The most common area is on the foot or the ankle.

Subject’s sometimes hide hypodermic needles in their socks, shoes and the heel compartments of their shoes.

On a case by case basis, the DRE may need to examine other parts of the body for marks. Another such area may be the legs.

- **ALWAYS follow your Agency’s rules, policies and procedures and laws regarding invasive type searches.**
H. Conclusion
The injection site examination may reveal evidence of recent use.

The presence of marks, however, doesn’t mean drug influence or impairment at the time of the evaluation.

Conducting an injection site examination is a skill.
As with all skills, such as taking blood pressure, competency improves with practice.
I. Classification Exemplar
TOPICS FOR STUDY

1. What are the two subcategories of Narcotic Analgesics?

2. What three distinguishing characteristics do all Narcotic Analgesics share?

3. Consider this situation: A heroin addict injects what is, for him, a “normal” dose of the drug. One hour later a DRE examines the addict and finds that he is not impaired. What is the most likely explanation for this?

4. What is another, more common, name for the drug called Diacetyl Morphine?

5. What is Methadone?

6. An analgesic is a drug that ______?

7. What is Oxycodone?
**DRUG INFLUENCE EVALUATION**

**Evaluator:**
Officer Charles Sheffield  
Reno PD

**Arrestees Name:**
Schmack, Charley J

**Date Examined:**
12/23/14

**Recorder/Witness:**
Officer Karl Nieberlein  
Sparks PD

**Date of Birth:**
05/14/70

**Date of Arrest:**
12/23/14

**Arresting Officer:**
Sgt. Mike Edgell  
Nevada HP

**Date and Time of Arrest:**
05/14/70

**Location:**
9013 Washoe Co. Jail

**Protocol:**
Session XVII-

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**Modified Romberg Balance Test:**
- Walk and Turn Test:
  - Start time: 1518
  - End time: 1550
  - Steps taken: 9

**Finger to Nose Test:**
- Start time: 0110
  - End time: 0050

**Pupil Size:**
- Left Eye:
  - Room Light: 2.0
  - Darkness: 2.5
  - Direct: 2.0
- Right Eye:
  - Room Light: 2.0
  - Darkness: 2.5
  - Direct: 2.0

**Blood Pressure:**
110/60

**Temperature:**
97.0°

---

**Conclusion:**
- Minimal influence of drugs.
- suspicion of alcohol.

---

**Opinion of Evaluator:**
- Not Impaired
- Not under the influence of alcohol.
- Co-Other.
- Not under the influence of drugs.
- No record of any drug use.

---

**Chemical Test:**
- Negative

---

**Additional Notes:**
- No evidence of intoxication.
- No physical defects.
- Normal performance.
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Schmack, Charley

1. **LOCATION:** The evaluation was conducted at the Washoe County Jail.

2. **WITNESSES:** Officer Charles Sheffield of the Reno PD recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was requested to contact Sgt. Edgell of the NV HP at the Washoe County Jail for a drug evaluation. Sgt. Edgell advised that the suspect was operating a stolen vehicle, and was involved in a non-injury crash. The suspect’s speech was slow and thick. His coordination was poor and he was swaying as he stood. His pupils were constricted, and he was frequently licking his lips. He performed poorly on the SFST’s and was arrested for DUI.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the booking room at the County Jail. He appeared to be “on the nod.” His eyes were partially closed, his head kept nodding forward, and his breathing was slow and shallow. The suspect responded to questions slowly, and his speech was thick and slurred. He had a dry mouth and was licking his lips. His movements were slow and deliberate.

6. **MEDICAL PROBLEMS AND TREATMENT:** None noted or stated.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: The suspect swayed approximately 2” front to back and 3” side to side. He estimated 30 seconds in 54 seconds. Walk & Turn: The suspect lost his balance three times during the instructions stage, stopped while walking twice, missed touching heel to toe twice, stepped off the line once, and raised his arms for balance three times. One Leg Stand: Suspect swayed, used his arms for balance and put his foot down twice on each foot. Finger to Nose: Suspect missed the tip of his nose on four of the six attempts. His hand and arm movements were slow and deliberate.

8. **CLINICAL INDICATORS:** The suspect’s pulse, blood pressure, and temperature were below the DRE average ranges. His pupils were constricted in all three lighting levels with no visible reaction to light. His eyelids were droopy, and his muscle tone was flaccid.

9. **SIGNS OF INGESTION:** A red injection mark was located on the suspect’s left arm.

10. **SUSPECT’S STATEMENTS:** The suspect denied using drugs and said he was just tired.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of a Narcotic Analgesic and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A urine sample was collected from the suspect.

13. **MISCELLANEOUS:** Suspect was also charged with UUMV and DWS.
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Wynn, Hara

1. LOCATION: The evaluation was conducted at the Albany OSP Patrol Office.

2. WITNESSES: Sgt Mike Iwai of the Oregon State Police recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Sergeant Iwai and Trooper McKay for a drug evaluation at the Albany State Police Office. Trooper McKay advised that the suspect had failed to stop at a stop sign and had nearly crashed into his patrol vehicle. The suspect had slow and deliberate movements. His speech was slow, slurred, and raspy. His pupils were constricted. He was unable to perform the SFST’s as directed, and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at the Patrol Office. He was repeatedly scratching his face and neck. His head kept nodding forward and he appeared to be “on the nod.” His voice was raspy and low. His pupils appeared to be constricted and his coordination and movements were slow.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 3” front to back and side to side. He estimated 30 seconds in 44 seconds. Walk & Turn: Suspect lost his balance during the instructions, stopped while walking one time on the first nine steps and twice on the return. He stepped off the line once, and raised his arms for balance. One Leg Stand: He counted slowly, swayed, and used his arms for balance. He put his foot down twice while standing on his left foot and once while standing on his right foot. Finger to Nose: Suspect missed the tip of his nose on all six attempts.

8. CLINICAL INDICATORS: Suspect’s pulse, blood pressure, and body temperature were below the DRE average ranges. His pupils were constricted in all three lighting levels.

9. SIGNS OF INGESTION: Suspect had scars on his right inside forearm and fresh puncture wounds on the inside of his left arm. The marks were photographed.

10. SUSPECT’S STATEMENTS: The suspect refused to answer questions about drug use.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a Narcotic Analgesic and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS:
**DRUG INFLUENCE EVALUATION**

Evaluator: Officer Peter Manukas, Raleigh PD

DRE #: 14031
Rolling Log #: 14-11-210
Case #: 14-88754
Session XVII - #3

Recorder/Witness: Lt. Tim Tomczak, Raleigh PD

Date examined: 05/16/76

Given by: Officer Peter Manukas, Raleigh PD

Date of Birth: 05/16/76

M B

Arresting Officer (Name, ID#): Trooper Kendall Jackson #14576

Arresting agency: North Carolina HP

Date Warrant Issued: 11/07/14

Time Location: Raleigh Intake Center

Breath Results: 32215

Chemical Test: Blood

Date of Arrest: 11/07/14

Precinct/Station: Raleigh Intake Center

**Comments:**

Are you under the care of a doctor or dentist?

Are you taking any medication or drugs?

If you have taken pain pills or other medication, please list them and the date:

Are you under the care of a doctor or dentist?

Are you taking any medication or drugs?

Are you taking any prescription or nonprescription medication that may affect your driving ability?

Are you under the care of a doctor or dentist?

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Are you under the care of a doctor or dentist?
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Cotton, Ozzie

1. LOCATION: The evaluation was conducted at the Raleigh Police Department.

2. WITNESSES: Lt. Tim Tomczak of the Raleigh PD recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Trooper Jackson for a drug evaluation. Trooper Jackson advised me that the suspect was observed drifting in and out of his traffic lane and driving 20 mph under the posted speed on Highway 64. The suspect’s coordination was poor and he had slow and deliberate movements. His speech was slow, thick, and slurred. His pupils were constricted. He had difficulties performing the SFST’s and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at RPD. He was sitting at the interview table and was continually scratching his face and arms. He had a dry mouth and smacked his lips when he spoke. His movements were slow and deliberate, and he was unstable when he stood. He also stated he was cold.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 2” front to back and side to side, and estimated 30 seconds in 55 seconds. Walk & Turn: The suspect lost his balance twice during the instructions stage, missed heel to toe three times, stepped off the line three times, and raised his arms for balance six times. One Leg Stand: Suspect counted slowly, swayed while balancing, used his arms to balance, and put his foot down twice on the left and once on the right foot. Finger to Nose: Suspect had slow hand movements, and missed the tip of his nose on three of the six attempts.

8. CLINICAL INDICATORS: Two of the suspect’s three pulse rates were below the DRE average ranges. His blood pressure and temperature were below the DRE average ranges. His pupils were constricted in all lighting levels with little to no visible reaction to light.

9. SIGNS OF INGESTION: None evident.

10. SUSPECT’S STATEMENTS: Suspect denied drug use but said he used to take pain pills.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a Narcotic Analgesic and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS: An empty container of Vicodin was located in the suspect’s vehicle.

Rev. 10/15
Participant Manual

Drug Recognition Expert Course

Session 18
Practice: Test Interpretation

45 Minutes
[This page is intentionally left blank]
Learning Objectives

• Analyze the results of a complete drug influence evaluation and identify the category or categories of drugs affecting the individual examined

• Articulate the bases for the drug category identification

Upon successfully completing this session the participant will be able to:

• Analyze the results of a complete drug influence evaluation and identify the category or categories of drugs affecting the individual examined.

• Articulate the bases for the drug category identification.

CONTENT SEGMENTS .................................................................. LEARNING ACTIVITIES
A. Interpretation Demonstrations .............................................. Instructor-Led Demonstrations
B. Interpretation Practice............................................................... Small-Group Practice
.................................................................................................. Participant-Led Presentations
A. Interpretation Demonstrations

Case No.1: “Subject Martinez”

Preliminary Examination

• Review the results of the preliminary examination of Subject Martinez.

Eye Examinations

• Review the results of the eye examination of Subject Martinez.

Psychophysical Tests

• Review the results of the psychophysical tests of Subject Martinez.

Vital Signs Examinations

• Review the results of the vital signs examinations of Subject Martinez.

Dark Room Examinations

• Review the results of the dark room examinations of Subject Martinez.
Other Evidence

- Review the results of the examinations for injection sites and muscle rigidity, and of the final interview of Subject Martinez.

Opinion of the Evaluator
Case No. 2: "Subject Groves"

**Direct participants to review the “Subject Groves” exemplar.**

**Preliminary Examination**
- Review the results of the preliminary examination of Subject Groves.

**Eye Examination**
- Review the results of the eye examinations of Subject Groves.

**Psychophysical Tests**
- Review the results of the psychophysical tests of Subject Groves.

**Vital Signs Examinations**
- Review the results of the vital signs examinations of Subject Groves.

**Dark Room Examinations**
- Review the results of the dark room examinations of Subject Groves.
Other Evidence

• Review the results of the examinations for injection sites and muscle rigidity, and of the final interview of Subject Groves.

Opinion of the Evaluator
B. Interpretation Practice

Team Practice

Review and Discussion of Exemplars by Teams

Feedback of Results
**DRUG INFLUENCE EVALUATION**

Evaluator: Sgt. Scott Peters  
Riverton PD  
DRE #: 16855  
Rolling Log #: 14-08-024  
Case #: 14-69815  
Session XVIII - #1

Date Examined: 08/22/14  
Location: 2330 County Jail Intake

Date of Birth: 05/20/80  
Sex: M  
Race: H

Arresting Officer (Name, ID#):  
Lt. Ben Schlosser  
Wyoming HP  
#16843

Arresting Agency:  
Martinez, Juan  
05/12/0180 M  
H Laramie PD

**Evaluator's Comments:**

- **Date Examined:** 08/22/14  
- **Location:** 2330 County Jail Intake

**Address:**

- **Street:** S  
- **Number:** 14-89815  
- **City:** Riverton PD

**Evaluator's Signature:**

- **Crime:** Officer Bartel  
- **Date:** 08/04/14  
- **Languages:** English  
- **Age:** 38  
- **Height:** 5'9"  
- **Weight:** 150 lbs

**Medical:**

- **Address:** Laramie PD  
- **City:** Riverton PD

**Evaluator’s findings:**

- **Date:** 08/22/14  
- **Time:** 2210

**Drug Influence Evaluation:**

<table>
<thead>
<tr>
<th>Pupil Size</th>
<th>Room Light</th>
<th>Darkness</th>
<th>Direct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Eye</td>
<td>5.0</td>
<td>6.5</td>
<td>4.0</td>
</tr>
<tr>
<td>Right Eye</td>
<td>5.0</td>
<td>6.5</td>
<td>4.0</td>
</tr>
</tbody>
</table>

**Blood Pressure:**

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>156 / 98</td>
<td>102</td>
</tr>
</tbody>
</table>

**Muscle Tone:**

- **Normal**  
- **Flaccid**  
- **Rigid**

**Comments:**

- **What drugs or medications have you been using?**
- **How much?**
- **Time of use?**
- **Where were the drugs used? (Location)**
- **Date/Time of arrest:** 08/22/14  
- **Time DRE was notified:** 2210  
- **Evaluation start time:** 2330  
- **Evaluation completion time:** 2430

**Officer’s Signature:**

- **DRE #:** Reviewed/approved by / date:

**Opinion of Evaluator:**

- **Not Impaired**  
- **Medical**  
- **CNS Stimulant**  
- **Dissociative Anesthetic**  
- **Inhalant**  
- **Alcohol**  
- **CNS Depressant**  
- **Hallucinogen**  
- **Narcotic Analgesic**  
- **Cannabis**
Suspect: Martinez, Juan

1. **LOCATION:** The evaluation was conducted at Albany County Jail.

2. **WITNESSES:** Lt. Ben Schlosser of the Wyoming HP recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was requested to contact Officer Bartel at the County Jail for a drug evaluation. When contacted, he advised that he had observed the suspect’s vehicle drifting over the lane divider line on Hwy 287 and nearly hit a vehicle head-on. When contacted, the suspect appeared dazed and confused. He had a blank stare, was non-responsive at times, and sweating profusely. He had six clues of HGN, VGN, and did poorly on the SFST’s, and was arrested for DUI.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the booking area. He appeared to be disoriented and had a fixed, blank stare. He responded very slowly to questions. His speech was slow and slurred. His face was flushed and he was sweating.

6. **MEDICAL PROBLEMS AND TREATMENT:** None noted or stated.

7. **PSYCHOPHYSICAL TESTS:**
   - Modified Romberg Balance: The suspect swayed approximately 3” side to side, and estimated 30 seconds in 38 seconds.
   - Walk & Turn: Suspect lost his balance twice during the instructions stage, stopped walking three times, stepped off the line twice, and raised his arms for balance five times.
   - One Leg Stand: Suspect put his foot down twice while standing on his left foot and three times on the right and nearly fell. The test was stopped for safety reasons.
   - Finger to Nose: Suspect missed the tip of his nose on four of the six attempts. His arm movements were very slow and rigid.

8. **CLINICAL INDICATORS:** Suspect had six clues of HGN and exhibited an early onset of Nystagmus. VGN and LOC were also present. The suspect’s pulse rates, blood pressure, and temperature were all elevated and were above the DRE average ranges.

9. **SIGNS OF INGESTION:** The suspect had a chemical-like odor on his breath.

10. **SUSPECT’S STATEMENTS:** The suspect did not respond to questions about drug use.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of a _______________ and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A urine sample was collected from the suspect.

13. **MISCELLANEOUS:** A glass vial with an unknown liquid was located in the suspect’s vehicle. He claimed he got it from a friend and didn’t know what it was.
**DRUG INFLUENCE EVALUATION**

**Evaluator:** Sgt. Sam Ketchum
**Sgt.** Idaho State Police

**Trooper Chris Glenn**
**Officer Robert R.**
**Officer Tim Riha**

**Date Examined:** 09/15/14
**Time Examined:** 1730
**Location:** Nampa PD

**Chemical Test:** Urine [ ] Blood [ ]
**Test Refused:** [ ]
**Test or tests refused:** [ ]

**Date of Birth:** 08/10/87
**Sex:** M
**Race:** W
**Property:** None

**Groves, Robert R.**
**Sgt. Sam Ketchum Idaho State Police**

**Date of Arrest:** 09/15/14
**Time of Arrest:** 1640
**Precinct/Station:** Nampa Police Department

**Number not impaired:** [ ]
**Alcohol:** [ ]
**CNS Stimulant:** [ ]
**Dissociative Anesthetic:** [ ]
**Inhalant:** [ ]
**Medical:** [ ]
**CNS Depressant:** [ ]
**Hallucinogen:** [ ]
**Narcotic Analgesic:** [ ]
**Cannabis:** [ ]

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**Modified Romberg Balance and Tum Test**

<table>
<thead>
<tr>
<th>Walk and Turn Test</th>
<th>HGN</th>
<th>Right Eye</th>
<th>Left Eye</th>
<th>Convergence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fails 1st 9</td>
<td>Lack of Smooth Pursuit</td>
<td>None</td>
<td>Left Eye</td>
<td>Right eye</td>
</tr>
<tr>
<td>Fails 2nd 9</td>
<td>Maximum Deviation</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

**Internal clock:** estimated as 30 seconds

**Blood pressure:** 118 / 62
**Temperature:** 97.4 o

**Pupil Size:**
- Room Light: 2.5 - 5.0
- Darkness: 5.0 - 8.5
- Direct: 2.0 - 4.5

**Pupil Size:**
- Left Eye: 2.0
- Right Eye: 2.0

**Rebound Dilation:**
- [X] Yes [ ] No

**Eye Lid:** Normal

**Reaction to Light:**
- [X] Little to None

**Nasal area:**
- Clear

**Oral cavity:**
- Clear

---

**Comments:**
- Slow hand and arm movements. Searched for nose.
- Nothing detected.

---

**Opinion of Evaluator:**
- Not Impaired [ ]
- Alcohol [ ]
- CNS Stimulant [ ]
- Dissociative Anesthetic [ ]
- Inhalant [ ]
- Medical [ ]
- CNS Depressant [ ]
- Hallucinogen [ ]
- Narcotic Analgesic [ ]
- Cannabis [ ]

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**Rev. 02/15**
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Groves, Robert

1. LOCATION: The evaluation was conducted at the Nampa Police Department.

2. WITNESSES: Trooper Chris Glenn of the Idaho SP recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Officer Riha of the Nampa Police Department for a drug evaluation. Officer Riha advised that he had observed the suspect’s vehicle drifting over the center line and traveling 15 mph under the posted speed on N. Midland Blvd. When stopped, the suspect had slow, slurred speech. His balance and coordination were poor, and he was unable to complete the SFST’s as directed and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the Interview Room at the PD. He appeared sleepy, and his head was nodding forward. His speech was slow and slurred. When he stood up, he lost his balance and used the desk to steady himself.

6. MEDICAL PROBLEMS AND TREATMENT: Suspect said he twisted his back about two weeks ago and a friend gave him some pills for it. He did not seek medical treatment.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 3” front to back and side to side, and estimated 30 seconds in 42 seconds. Walk & Turn: Suspect lost his balance twice during the instructions stage, missed heel to toe three times, stepped off the line four times, and raised his arms for balance five times. One Leg Stand: Suspect swayed while balancing, used his arms to balance, and put his foot down twice while standing on each foot. Finger to Nose: Suspect missed the tip of his nose on all six attempts. He searched for his nose, and his movements were slow and deliberate.

8. CLINICAL INDICATORS: The suspect’s pulse rates were all at the low end of the DRE average ranges. His blood pressure and temperature were below the DRE average ranges. His pupils were constricted in all three lighting levels with little to no reaction to light.

9. SIGNS OF INGESTION: None were evident.

10. SUSPECT’S STATEMENTS: Suspect admitted taking a “couple pills” earlier in the day.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a ____________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS:
**DRUG INFLUENCE EVALUATION**

Evaluator: Deputy Dallas Gotts  
Maricopa Co. SO  

Date Examined: 07/22/14  

Recorder: Det. Kemp Layton  
Phoenix PD  

Date of Birth: 07/13/79  
Sex: M  
Race: H  

Arrestee's Name: Halos, Carlos  

Date Examined: 07/22/14  
Location: 2110 4th Ave. Jail  

Test Refused: No  

Chemical Test: Blood  

Chemical Test: 0.00  

Chemical Test Results: None  

Arresting Officer Agency: Glendale PD  

Arresting Officer Name, ID#: Officer Chip Haas #13073  

Taco: About 5 pm  

How much?: Water 2 bottles  

Time of Last Drink: N/A  

Location: 15690  

What have you been drinking?: How much?: Water 2 bottles  

Time of use?: N/A  

Where were the drugs used?: (Location)  

Date of arrest: 07/22/14  
Time DRE was notified: 2050  

Evaluation start time: 2110  
Evaluation completion time: 2200  

Precinct/Station:  

Officer's Signature: Reviewed/approved by / date:  

Opinion of Evaluator: Not Impaired  
Alcohol: No  
CNS Stimulant: No  
Dissociative Anesthetic: No  
Inhalant: No  
Medical: No  
CNS Depressant: No  
Hallucinogen: No  
Narcotic Analgesic: No  
Cannabis: No  

**Finger to Nose**  
(Draw lines to spots touched)  

- R  
- P  
- 2  
- 4  
- 5  
- 3  
- 1  
- M  
- S  

Blood pressure: 156 / 98  
Temperature: 99.8 °

Muscle tone: Normal  

Flaccid: No  
Rigid: No  

Comments:  

What drugs or medications have you been using?  

"No man, I'm clean."  

How much?: N/A  

Time of use?: N/A  

Where were the drugs used? (Location)  

Deputy Dallas Gotts  
Maricopa Co. SO

Corrective Lenses:  

- None  
- Contacts, if so:  
- Hard: No  
- Soft: Yes  

Pupil Size:  

- Equal: No  
- Unequal (explain): None  

Pulse and time:  

1. 108 / 2122  
2. 106 / 2135  
3. 106 / 2150  

Modified Romberg Balance:  

- Lower body tremors.  
- 2" 2" 3" 3"  
- Sways while balancing  
- Uses arms to balance  
- Puts foot down  

Walk and Turn Test:  

- Starts too soon  
- Misses heel-toe  
- Steps off line  
- Raises arms  

Actual steps taken: 9  

Convergence:  

- Left Count: 38  
- Right Count: 41  

Type of footwear: Lace-up boots without laces  

**NURSE**

- Right Eye: None  
- Left Eye: None  

PUPIL SIZE:  

Room Light: 2.5 - 5.0  
Darkness: 5.0 - 8.5  
Direct: 2.0 - 4.5  

Left Eye: 6.5  
Right Eye: 6.5  

Reaction to Light: Normal  

Rebound Dilation:  

- Yes: No  
- No: Yes  

Pupillary Unrest:  

- Yes: No  
- No: Yes  

Reaction to Light: Normal  

- Left Eye: None  
- Right Eye: None  

Nasal area: Redness  
Oral cavity: Clear  

Type of foot:  

- L: None  
- R: None  

Internal clock: estimated as 30 seconds  

Finger to Nose:  
(Draw lines to spots touched)  

- R  
- P  
- 2  
- 4  
- 5  
- 3  
- 1  
- M  
- S  

Jerky, fast movements. Used pads of fingers.  

Blood pressure: 156 / 98  
Temperature: 99.8 °
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Hatos, Carlos

1. LOCATION: The evaluation was conducted in a holding cell at the 4th Ave Jail.

2. WITNESSES: Detective Kemp Layden of the Phoenix PD recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to meet Officer Haas of the Glendale PD for a drug evaluation. Officer Haas advised me that he had observed the suspect’s vehicle traveling at a high rate of speed on Indian School Road. When stopped, the suspect appeared nervous, was very talkative and could not stand still, and his pupils were dilated. He performed poorly on SFST’s and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the booking area at the jail. He was very talkative and was repeatedly shifting his weight from foot to foot. He was making abrupt, quick hand movements, and appeared animated and restless. When not speaking, he appeared to be grinding his teeth, and his pupils appeared to be dilated.

6. MEDICAL PROBLEMS AND TREATMENT: None noted and none stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 2” front to back and 3” side to side. He estimated 30 seconds in 23 seconds and had body tremors. Walk & Turn: Suspect lost his balance during the instructions stage, stopped while walking twice, missed touching heel to toe four times, and raised his arms for balance four times. His steps were quick and he slammed his heel to his toes on each step. One Leg Stand: Suspect swayed while balancing, used his arms for balance, and put his foot down once while standing on each foot. He also counted quickly. Finger to Nose: He missed the tip of his nose on five of the six attempts, and used the pads of his fingers on five attempts.

8. CLINICAL INDICATORS: Suspect’s pulse and blood pressure were elevated and above the DRE average ranges. His pupils were dilated in all lighting levels. Due to the suspect’s dark colored eyes, a U.V. Light was utilized for the Near Total Darkness measurement.

9. SIGNS OF INGESTION: Suspect’s nasal area was red and he had a bloody left nostril.

10. SUSPECT’S STATEMENTS: Suspect stated “I’m clean” and that he quit doing drugs.

11. DRE'S OPINION: In my opinion, the suspect is under the influence of a ________________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect at 2300 hours.

13. MISCELLANEOUS: 

Rev. 10/15
# Drug Influence Evaluation

**Evaluator:** Officer Virgil Miller  
**Wichita PD**

**DRE #:** 10828  
**Recording Log #:** 14-07-035  
**Case #:** 14-96875  
**Session:** XVIII - #4

**Detective / Witness:** Det. Karinna Brasser  
**Sedgwick CO SO**

**Officer:** Virgil Miller  
**Wichita PD**

**Arresting Officer:** Trooper Mark Crump  
**Kansais Highway Patrol**

**Session:** XVIII - #4

**Date Examined:** 07/18/2014  
**Location:** Sedgwick County Jail

**Results:**
- Test Refused
- Chemical Test: Blood

**Arrestee's Name:** Jackson, Scott M.  
**Date of Birth:** 06/15/78  
**Gender:** M  
**Race:** W  
**Agency:** Kansas Highway Patrol

**Date Examined:** 07/18/14  
**Time:** 20:20  
**Location:** Sedgwick County Jail

**Results:** 0.00  
**Instrument #:** 13240  
**Test refused:** D

**Miranda Warning Given:** Yes

**What have you eaten today?**  
- Ham sandwich Noon

**What have you been drinking?**  
- Coffee 2 cups

**Time of last drink:** 10 pm

**Last night?**  
- 7 hours

**Are you sick or injured?**  
- Yes (Asthma)

**Are you diabetic or epileptic?**  
- No

**Do you take insulin?**  
- No

**Are you taking any medication or drugs?**  
- Yes (L prednisone, chloroquine)

**Are you under the care of a doctor or dentist?**  
- Yes (Dentist)

**Attitude:** Cooperative, Passive

**Coordination:** Poor, Unsteady

**Speech:** Slow, Thick

**Corrective Lenses:** None

**Pupil Size:** Equal

**Vertical Nystagmus:** None

**Corrective Lenses:** None

**Rebound Dilation:** None

**Pupillary Unrest:** None

**Reaction to Light:** Little to None

**Blood pressure:** 122 / 88

**Temperature:** 98.0

**Nasal area:** Clear

**Oral cavity:** Clear

**Nail area:** Clear

**Room Light Pupil Size:** 2.5 - 5.0
**Darkness Pupil Size:** 5.0 - 8.5
**Direct Pupil Size:** 2.0 - 4.5

**Finger to Nose:**  
- (Draw lines to spots touched)

**Type of footwear:** Lace-up shoes

**Modified Romberg Balance:**
- 3" 3" 3" 3"

**Walk and Turn Test:**
- Starts too soon
- Misses heel-toe
- Steps off line
- Raises arms
- Actual steps taken: 9

**Internal clock:**
- Estimated as 30 seconds

**Type of footpath:**
- N/A

**Date / Time of arrest:** 07/18/14 19:15

**Time DRE was notified:** 19:48

**Evaluation start time:** 20:20

**Evaluation completion time:** 21:05

**Precinct/Station:**

**Opinion of Evaluator:** Not Impaired
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Jackson, Scott M.

1. LOCATION: The evaluation was conducted at the Sedgwick County Jail.

2. WITNESSES: Detective Karrina Brasser witnessed and recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Trooper Crump at the Sedgwick County Jail for a drug evaluation. Trooper Crump advised that he observed the suspect’s vehicle traveling E/B on Highway 54 near the Garden Plain exit traveling under the posted speed limit and drifting in and out of his lane. When Trooper Crump attempted to stop the suspect, he continued for over a mile before stopping. The suspect’s speech was thick and slow. The suspect had poor coordination, was unable to complete SFST’s as directed, and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at the jail. He was cooperative, and had slow, thick, slurred speech. He responded slowly to questions. He was unstable on his feet and nearly fell several times when walking.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 3” front to back and side to side. He estimated 30 seconds in 42 seconds. Walk & Turn: Suspect lost his balance during the instructions stage, stopped while walking twice, missed heel to toe six times, stepped off the line three times, and raised his arms for balance when he walked. He made an improper turn by walking around in a half circle using both feet. One Leg Stand: Both tests were stopped for safety reasons after he put his down three times on each attempt and nearly fell. Finger to Nose: The suspect had slow hand movements, and he missed the tip of his nose on five of the six attempts.

8. CLINICAL INDICATORS: The suspect’s pulse rates and blood pressure were below the DRE average ranges. His pupils were constricted in all three of the lighting levels.

9. SIGNS OF INGESTION: The suspect had two fresh injection marks on his left forearm.

10. SUSPECT’S STATEMENTS: The suspect denied using drugs.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a _____________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS: 

Rev. 10/15
**DRUG INFLUENCE EVALUATION**

Evaluator: Sgt. Paul Kotter, Utah Highway Patrol

Trooper: Jason Marshall, Utah HP

Stevens, William A

Date Examined/Tone Location: 11/17/14 County Intake

Results: Breath Test Refused

Results: 0.00

Instrument #: 14-90987

 Arresting Officer: Trooper Janet Miller, Utah Highway Patrol

Recorded By: Wilness

Time Examined: 10:00

Breath Results: Test Refused

Results: 0.00

Instrument #: 14-90987

Arresting Officer: Trooper Janet Miller, Utah Highway Patrol

Date of Birth: 04/11/47

Name: Stevens, William A

Date of Birth: 04/11/47

Name: Stevens, William A

Time of Last Drink: 6:30 pm/ 1915

Time of Last Drink: 6:30 pm/ 1915

Given by: Tpr. Miller

Given by: Tpr. Miller

Breath Odor: Slurred, Thick

Breath Odor: Slurred, Thick

Corrective Lenses: None

Corrective Lenses: None

Eye: Reddened Conjunctiva

Eye: Reddened Conjunctiva

Eyes: Nonnal

Eyes: Nonnal

Pupil Size: Equal

Pupil Size: Equal

Rebound Dilation: Pupillary Unrest

Rebound Dilation: Pupillary Unrest

Nasal area: Clear

Nasal area: Clear

Oral cavity: Clear

Oral cavity: Clear

Type of footwear: Dress shoes

Type of footwear: Dress shoes

Muscle tone: Normal

Muscle tone: Normal

Comments: Finger to Nose (Draw lines to spots touched)

Comments: Finger to Nose (Draw lines to spots touched)

Blood pressure: 156/98

Blood pressure: 156/98

Temperature: 99.8

Temperature: 99.8

What drugs or medications have you been using? "Some medicine for anxiety. That's it man."

What drugs or medications have you been using? "Some medicine for anxiety. That's it man."

How much? Just a couple

How much? Just a couple

Time of use? Home

Time of use? Home

Where were the drugs used? Location)

Where were the drugs used? Location)

Date/Time of arrest: 11/17/14 1810

Date/Time of arrest: 11/17/14 1810

Time DRE was notified: 1845

Time DRE was notified: 1845

Evaluation start time: 1910

Evaluation start time: 1910

Precinct/Station:

Precinct/Station:

Opinion of Evaluator: Not Impaired

Opinion of Evaluator: Not Impaired
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Stevens, William A.

1. LOCATION: The evaluation was conducted at the Salt Lake County Jail.

2. WITNESSES: Trooper Jason Marshall of the Utah H.P. witnessed the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Trooper Miller for a drug evaluation. Trooper Miller advised she had located the suspect’s vehicle stopped partially in the travel lane of Highway 48. The suspect was sitting in the driver’s seat and had a drunk-like appearance. His speech was thick, slurred, and slow. He had six clues of HGN and VGN, but no odor of an alcoholic beverage was detected. He had difficulty performing the SFST’s and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at the County Jail. He was cooperative and was slow to answer questions. His speech was slow, thick, and slurred. His balance was poor, and he staggered when he walked.

6. MEDICAL PROBLEMS AND TREATMENT: The suspect stated he was seeing Dr. Frank at the Clinic who had prescribed him something for anxiety.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 2” in a circular motion, and he estimated 30 seconds in 38 seconds. Walk & Turn: Suspect lost his balance twice during the instructions, stopped while walking twice, missed heel to toe three times, stepped off the line twice, and raised his arms for balance five times. He also lost his balance when he turned and took an extra step on the second nine steps. One Leg Stand: Suspect swayed while balancing, used his arms to balance, hopped once on his left foot, and put his foot down twice standing on each foot. Finger to Nose: The suspect missed the tip of his nose on three of the six attempts and had slow arm movements.

8. CLINICAL INDICATORS: The suspect had six clues of HGN with a 30 degree angle of onset. VGN and Lack of Convergence were also present. His pulse rates and blood pressure were below the DRE average ranges. His pupils were all within the DRE average ranges.

9. SIGNS OF INGESTION: Nothing observed or detected.

10. SUSPECT’S STATEMENTS: Suspect admitted taking 2 “anxiety pills” earlier in the day.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of a ______________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:

Rev. 10/15
Mid-Course Review

Review of Drugs, Drug Categories, and the Drug Influence Evaluation
MID-COURSE REVIEW

CONTENT SEGMENTS    LEARNING ACTIVITIES

A. Drugs, Drug Categories and the.............................................Instructor / Participant Dialogues
............................................................................................................Drug Influence Evaluation

B. Eyes and Vital Signs.................................................................Participant-Led Demonstrations

C. Physiology

D. Questions and Answers
A. Drugs, Drug Categories, and the Drug Influence Evaluation

Define the word “drug.”

- Any substance that, when taken into the human body, can impair the ability of the person to operate a vehicle safely.

Name the seven drug categories.

- CNS Depressants, CNS Stimulants, Hallucinogens, Dissociative Anesthetics, Narcotic Analgesics, Inhalants, and Cannabis

Name the six subcategories of Depressants.

- Barbiturates, Non-Barbiturates, Anti-Anxiety Tranquilizers, Anti-Depressants, Anti-Psychotic Tranquilizers, and Combinations of the first five

Name three subcategories of CNS Stimulants.

- Cocaine, the Amphetamines, and “Others.”

Name two sub-categories of Narcotic Analgesics.

- Opiates and Synthetics
Identify the category for each of the listed drugs:

Desoxyn
  • CNS Stimulant

Secobarbital (Seconal)
  • CNS Depressant (Barbiturate)

Dilaudid
  • Narcotic Analgesic

Alprazolam (Xanax)
  • CNS Depressant (Anti-Anxiety)

Phenyl Cyclohexyl Piperidine
  • Dissociative Anesthetics

“Ecstasy” (MDMA)
  • Hallucinogen

ETOH
  • CNS Depressant

Numorphan
  • Narcotic Analgesic

Psilocybin
  • Hallucinogen
List the twelve components of the Drug Influence Evaluation in the proper sequence.

1. Breath Alcohol Test
2. Interview of Arresting Officer
3. Preliminary Examination
4. Eye Examinations
5. Divided Attention Tests
6. Vital Signs Examinations
7. Darkroom Examinations
8. Check for Muscle Tone
9. Injection Sites Inspection
10. Statement of Suspect
11. Evaluator’s Opinion
12. Toxicological Examination
• Demonstrate the Preliminary Examination.
• Demonstrate the Eye Examinations.
• Demonstrate the Administration of the Divided Attention Tests.
• Demonstrate the Vital Signs Examinations.
• Demonstrate the Darkroom Examinations.
• Demonstrate the Check for Muscle Tone and the inspection for Injection Sites.
Identify the category for each of the listed drugs:

Demerol
- Narcotic Analgesic

Adderall
- CNS Stimulant

Chlordiazepoxide
- CNS Depressant

Ketamine
- Dissociative Anesthetics

Percodan
- Narcotic Analgesic

Ritalin
- CNS Stimulant

Isopropanol
- CNS Depressant

Bufotenine
- Hallucinogen

Methaqualone
- CNS Depressant
B. Eyes and Vital Signs

Name the three clues of Horizontal Gaze Nystagmus
Lack of smooth pursuit, distinct and sustained nystagmus at maximum deviation, angle of onset
Name the categories of drugs that will cause Horizontal Gaze Nystagmus.
CNS Depressants, Dissociative Anesthetics, Inhalants
Name the categories that will cause Vertical Gaze Nystagmus.
  • CNS Depressants, Dissociative Anesthetics, Inhalants
Name the test that is always administered immediately after Vertical Gaze Nystagmus.
  • Lack of Convergence
Name the categories of drugs that usually will cause Lack of Convergence.
  • CNS Depressants, Dissociative Anesthetics, Inhalants, Cannabis
Name the lighting conditions under which we make estimations of pupil size.

- Room light, near-total darkness, direct light

Name the other things a DRE looks for while shining the light directly into the subject’s eye.

- Pupil reaction to light and rebound dilation

How quickly must the pupil start to constrict if it is considered to exhibit normal reaction to light?

- Within one second

Define Rebound Dilation.

- A period of pupillary constriction followed by a period of pupillary dilation where the pupil steadily increases in size and does not return to its original constricted size.

State the normal ranges of pupil size for the three lighting conditions.

- Room light: 2.5 – 5.0 mm.
- Near Total Darkness: 5.0 – 8.5 mm.
- Direct Light: 2.0 – 4.5 mm.
What Do These Words Mean?

- Miosis
- Mydriasis
- Ptosis

Pupil Dilation and Constriction

- What categories of drugs will cause dilation of the pupils?
- What categories of drugs will cause constriction?

Define each of the listed terms:

- Miosis
  Abnormally constricted pupils
- Mydriasis
  Abnormally dilated pupils
- Ptosis
  Droopy eyelids

What categories of drugs will cause dilation of the pupils?
- CNS Stimulants, Hallucinogens, Cannabis (although sometimes only slight dilation, if any)

What categories of drugs will cause constriction?
- Narcotic Analgesics
More Drugs to Categorize

- Oxycodone
- Halcion
- Librium
- Peyote
- Adderall
- Diazepam
- Dexedrine
- Hycodan

Identify the category for each of the listed drugs:

Oxycodone
  - Narcotic Analgesic
Halcion
  - CNS Depressant
Librium
  - CNS Depressant
Peyote
  - Hallucinogen
Adderall
  - CNS Stimulant
Diazepam
  - CNS Depressant
Dexedrine
  - CNS Stimulant
Hycodan
  - Narcotic Analgesic
Klonopin
  - CNS Depressant
Define “Pulse.”

• The expansion and contraction of an artery, generated by the pumping action of the heart.

(Also acceptable: the expansion and contraction of an artery, caused by the surging flow of blood)

Define “Pulse Rate.”

• The number of pulsations in an artery per minute

Define “Artery.”

• A strong, elastic blood vessel that carries blood from the heart to the body tissues.

Define “Vein.”

• A blood vessel that carries blood back to the heart from the body tissues.
Identify the location of each listed pulse point:

Radial
• In the wrist, at the base of the thumb

Brachial
• In the crook of the arm

Carotid
• In the neck, on either side of the center of the throat

State the normal range of adult human pulse rate.
• 60 – 90 beats per minute

Name the drug categories that usually cause elevated pulse rate.
• CNS Stimulants, Hallucinogens, Dissociative Anesthetics, Inhalants, Cannabis

Name the drug categories that usually cause lowered pulse rate.
• CNS Depressants, Narcotic Analgesics
Define “Blood Pressure.”
• The force exerted by blood on the walls of the arteries

How often does a person’s blood pressure change?
• It is always changing, from instant to instant.

When does the blood pressure reach its highest value?
• When the heart is fully contracted, and blood is sent rushing into the arteries.

When does the blood pressure reach its lowest value?
• When the heart is fully expanded, just before it starts to contract for the next “pumping” action.

Name the two medical instruments that are used to measure blood pressure.
• SPHYGMOMANOMETER and STETHOSCOPE

Name the sounds that we hear through the stethoscope when we take a blood pressure measurement.
• KOROTKOFF SOUNDS
What does this “Hg” mean?

- Chemical symbol for the element Mercury; abbreviation for the Latin word Hydrargyrum, meaning “Mercury.”

In what units is blood pressure measured?

- Millimeters of Mercury

Suppose that, at some particular instant, a person has a blood pressure of 120 mmHg. What does that “120 mmHg” mean?

- It means the pressure would be strong enough to push a column of liquid Mercury up a glass tube to a height of 120 millimeters.

Name the drug categories that usually cause a lowered blood pressure.

- CNS Depressants, Narcotic Analgesics, and the Anesthetic Gases subcategory of Inhalants

Name the drug categories that elevate blood pressure.

- CNS Stimulants, Hallucinogens, Dissociative Anesthetics, Cannabis, and the other two subcategories (Volatile Solvents and Aerosols) of Inhalants
Some Technical Terms to Define

- Systolic
- Diastolic
- Bradycardia
- Tachycardia
- Hypertension
- Hypotension

Blood Pressure Measurement

- State the normal range of systolic blood pressure:
- State the normal range of diastolic blood pressure:

State the meaning of each of the listed terms:

Systolic
- The highest value of blood pressure

Diastolic
- The lowest value of blood pressure

Bradycardia
- Abnormally slow heart rate, pulse rate below the normal range

Tachycardia
- Abnormally rapid heart rate, pulse rate above the normal range

Hypertension
- Abnormally high blood pressure

Hypotension
- Abnormally low blood pressure

State the normal range of systolic blood pressure.
- 120 – 140 mmHg

State the normal range of diastolic blood pressure.
- 70 – 90 mmHg
C. Physiology

Define “Physiology.”

- Physiology is the branch of biology dealing with the functions and activities of life or living matter and the physical and chemical phenomena involved.

What is the expression we use to remember the names of the ten major body systems?

- MURDERS INC
- Muscular (have a student print out each name)
- Urinary
- Respiratory (or, reproductive)
- Digestive
- Endocrine
- Reproductive (or, respiratory)
- Skeletal
- Integumentary
- Nervous
- Circulatory
State the word that means “dynamic balance involving levels of salts, water, sugars and other materials in the body’s fluids.”

- Homeostasis

Which artery carries blood from the heart to the lungs?

- Pulmonary

What is unique about the Pulmonary artery, compared to all other arteries?

- It is the only artery that takes blood from the right side of the heart
- It is the only artery that carries deoxygenated blood (i.e., blood that is depleted of oxygen)

What are the Pulmonary veins?

- The veins that carry blood back to the heart from the lungs

What is unique about the Pulmonary veins?

- They are the only veins that bring blood to the left side of the heart
- They are the only veins that carry oxygenated blood
Name the various types of nerves.

- Sensory nerves, carry messages to the brain. Also known as Afferent Nerves
- Motor nerves, carry messages from the brain. Also known as Efferent Nerves
- Voluntary nerves are motor nerves that carry messages to the muscles that we consciously control.
- Autonomic nerves are motor nerves that carry messages to the muscles and organs we do not consciously control.
- Sympathetic nerves are autonomic nerves that carry messages commanding the body to react to fear, stress, excitement, etc. Clarification: Sympathetic nerves carry the brain’s “fire alarms” and “wake up calls”.
- Parasympathetic nerves are autonomic nerves that carry messages to produce relaxed and tranquil activities. Clarification: Parasympathetic nerves carry the brain’s “all clear” and “at ease” messages.
Define each of the listed terms:

**Neuron**
- A nerve cell, the basic “building block” of a nerve

**Synapse**
- The gap or space between two nerve cells

**Neurotransmitter**
- A chemical that flows across the synapse, to carry a message from one neuron to the next

**Axon**
- The end of a neuron that sends out the neurotransmitter

**Dendrite**
- The end of a neuron that receives the neurotransmitter
D. Questions and Answers

QUESTIONS?
Participant Manual

Drug Recognition Expert Course

Session 19
Inhalants

1 Hour and 35 Minutes

Session 19 - Inhalants
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Learning Objectives

- Explain a brief history of the Inhalant category of drugs
- Identify common drug names and terms associated with this category
- Identify common methods of administration for this category
- Describe the symptoms, observable signs, and other effects associated with this category

Learning Objectives

- Describe the typical time parameters, i.e. onset and duration of effects associated with this category
- List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of this category of drugs
- Correctly answer the “topics for study” questions at the end of this session

Upon successfully completing this session the participant will be able to:

- Explain a brief history of the Inhalant category of drugs.
- Identify common drug names and terms associated with this category.
- Identify common methods of administration for this category.
- Describe the symptoms, observable signs and other effects associated with this category.
- Describe the typical time parameters, i.e. onset and duration of effects associated with this category.
- List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of this drug category.
- Correctly answer the “topics for study” questions at the end of this session.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES

A. Overview of the Category ............................................................. Instructor-Led Presentations
B. Possible Effects .................................................. Review of the Drug Evaluation and Classification Exemplars
C. Onset and Duration of Effects .......................................................... Reading Assignments
D. Overdose Signs and Symptoms......................................................... Video Presentations
E. Expected Results of the Evaluation..................................................... Slide Presentations
F. Classification Exemplar
A. Overview of the Category

Inhalants are breathable chemicals that produce mind altering results.

Inhalants vary widely in terms of the chemical involved and the specific effects produced.

Depending on the nature of the particular Inhalant, the effects produced may be similar to those of CNS Stimulants, Depressants or Hallucinogens.
There are three major subcategories of Inhalants:

- Volatile Solvents
- Aerosols
- Anesthetic Gases

“Volatile” Solvents

The Volatile Solvents include a large number of readily available substances, none of which are intended by their manufacturers to be used as drugs.

“Volatile” means that they evaporate easily to produce fumes.

One widely abused Volatile Solvent is plastic cement, or “model airplane glue.”

Plastic cement includes the following volatile chemicals:

- Toluene
- Acetone
- Naphtha
- Aliphatic Acetates (straight-chained hydrocarbons)
- Hexane
- Cyclohexane
- Benzene
Other frequently abused Volatile Solvents include:

- Fingernail polish remover (contains Acetone)
- Household cements and glues (rubber cements contain Benzene)
- Lighter fluid (contains Naphtha)

Petroleum products:

- Various glues (model airplane glue)
- Gasoline
- Kerosene
- Dry cleaning fluids
- Paints (particularly oil or solvent based)
- Paint thinners
- Spray paints
- Liquid correction fluid
- Engine degreasers
**Aerosols**

Aerosols are chemicals discharged from a pressurized container by the propellant force of a compressed gas.

Commonly abused Aerosols include hair sprays, deodorants, insecticides, glass chillers (freeze spray), and vegetable frying pan lubricants.

All of these abused Aerosols contain various hydrocarbon gases that produce drug effects.

The overwhelming majority of abusers of Volatile Solvents and Aerosols are pre-teens and teenagers.

Some reasons:

- These substances appear in nearly every household.
- They are inexpensive and readily accessible.
Anesthetic Gases

The third subcategory is Anesthetic Gases. Anesthetic gases are drugs that abolish pain. They are used medically during surgical procedures such as childbirth, dental surgery, etc.

Adults may be more frequent users of the anesthetic gases subcategory than of the Aerosols or Volatile Solvents.

Anesthetic gases that sometimes are abused as Inhalants:

• Ether
• Nitrous Oxide

Many of these substances have a long history of medical and illicit use, e.g., Ether abuse dates to the 1790’s in England.

Nitrous Oxide has been used since 1845. It is still used in certain dental procedures.

Nitrous Oxide is a propellant for whipped cream. Drug paraphernalia stores often sell Nitrous Oxide in cartridges that are identical to carbon dioxide containers. They are termed by users “whippets,” and are allegedly sold to purchasers as devices to propel whipped cream.
Other common Inhalants in this subcategory are:

- Amyl Nitrite
- Butyl Nitrite (Isobutyl Nitrite)

Nitrites are vasodilating substances used medically to relieve angina pectoris (heart-related chest pain) and for treatment of cyanide poisoning. In angina, the nitrites work by dilating blood vessels near the heart so that more blood can reach the heart.

Nitroglycerin, ordinarily not abused as an intoxicant, is also used for this purpose.

Isobutyl Nitrite and Butyl Nitrite have essentially identical effects of Amyl Nitrite.

Anesthetic gases can dilate the blood vessels around the heart thus causing a lowered blood pressure.

Common slang and brand names for the nitrites are: “Rush” and “Locker Room.”

Examples: Amyl Nitrite and Butyl Nitrite are sold in small glass bottles or bulbs. The user simply opens the bottle and breathes in the fumes. They have been marketed in drug paraphernalia stores as room deodorizers.
Inhalants obviously are ingested by breathing, or inhaling the fumes.

- Some are ingested directly from the source.
- Some are soaked into rags, handkerchiefs, or tissue paper for repeated inhalation.
- Some are placed in paper or plastic bags which the user places over the face or head. These may be placed in twist lock beverage containers.
- Some are used by breathing the fumes or vapors from balloons.

Some common street names that Inhalant users use are: huffing, hacking, ballooning and glading.
B. Possible Effects

The effects of Inhalants vary somewhat from one substance to another. In fact, many of the Inhalants are classified as Depressants in medical texts. Their effects, consequently, often mirror alcohol intoxication.

Common effects of Inhalants include:

- Altered shapes and colors
- Antagonistic behavior
- Bizarre thoughts
- Distorted perceptions of space and time
- Dizziness and numbness
- Drowsiness and weakness
- Floating sensations
- Inebriation similar to alcohol intoxication
- Intense headaches
- Light headedness
- Nausea and excessive salivation
- Possible hallucinations

Persons under the influence of Inhalants generally will appear confused and disoriented, and their speech will be slurred.
C. Onset and Duration of Effects

Inhalants’ effects are felt virtually immediately.

Duration depends on the particular substance.

• The effects of nitrous oxide last 5 minutes or less.
• Amyl Nitrite and Isobutyl Nitrite produce effects that last a few seconds up to 20 minutes.

Users claim these substances enhance sexual excitement. This may occur from dilation of genital arteries (vasodilation) and relaxation of other smooth muscles.

Inhalation of these produces a distinct “rush” similar to that of the related substance, Nitrous Oxide.

Glue, paint, gasoline and other commonly abused Inhalants produce effects that last several or more hours. (Generally 6-8 hours for most volatile solvents depending on exposure).
D. Overdose Signs and Symptoms

There is a risk of death due to overdose of Inhalants.

All volatile solvents make the heart more sensitive to adrenaline. This sometimes causes a dangerous cardiac arrhythmia. The term “sudden sniffing death” (SSD) has been used to describe death resulting from physical exertion and the breathing of Inhalants in an enclosed, poorly ventilated space.

Some Inhalants will depress the Central Nervous System to the point where respiration ceases. Others can produce instant death from heart failure.

Overdoses of Inhalants frequently induce severe nausea and vomiting. If the user vomits while he or she is unconscious, death can result from aspiration of the vomitus.

Death can also result indirectly, if a person places a plastic bag over the head, loses consciousness and suffocates.

Long term abuse of Inhalants can cause permanent damage to the Central Nervous System, and greatly reduce mental and physical abilities.

Evidence also exists of liver, kidney, bone and bone marrow damage resulting from long term Inhalant abuse.

There are no well-defined withdrawal symptoms for these substances. Physical dependence has not been documented, although habituation is common.
E. Expected Results of the Evaluation

With Inhalants, there is significant variation in effects from one substance to another.
Evaluation of Subjects Under the Influence of Inhalants

- HGN - Present
- VGN - Present (high dose for that individual person)
- Lack of Convergence - Present
- Impaired performance will be evident on Modified Romberg Balance, Walk and Turn, One Leg Stand, and Finger to Nose tests

Observable Evidence of Impairment

Eye Exam

- HGN: Horizontal Gaze Nystagmus will generally be present.
- VGN: Vertical Gaze Nystagmus may be present.
- LOC: Lack of Convergence will be present.

Psychophysical Exercise

Drug Evaluation Tests

Performance on the Modified Romberg Balance, Walk and Turn, One Leg Stand, and Finger to Nose tests will be impaired.
Vital Signs

Pulse will be up.

Pulse increase is due to many factors, including oxygen displacement. The heart may beat faster in order to supply body tissues with a sufficient supply of oxygen.

Blood pressure will be up or down.

The lowering of blood pressure by Anesthetic Gases is due to their vasodilation effect. The heart compensates for this vasodilation by increasing its heart rate.

Effect on body temperature may be up, down or normal range.

Dark Room

Pupil size will be normal (DRE Average Ranges) but may be dilated.

Reaction to light generally will be slow.

Anesthetic gases may produce some dilation, although usually not to the extent seen with CNS Stimulants or Hallucinogens. No Inhalants produce pupillary constriction.
General Indicators

- Bloodshot, watery eyes
- Confusion
- Disoriented
- Flushed face
- Intense headaches
- Lack of muscle control
- Non-communicative
- Normal or Flaccid muscle tone
- Odor of the inhaled substance
- Possible nausea
- Residue of the substance around the face and nose and on the hands or clothing
- Slow, thick, slurred speech

Speech usually clears up quickly when substance is no longer being inhaled.
### Inhalants Symptomatology Chart

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGN</td>
<td>Present</td>
</tr>
<tr>
<td>VGN</td>
<td>Present (High dose for that individual)</td>
</tr>
<tr>
<td>Lack of Convergence</td>
<td>Present</td>
</tr>
<tr>
<td>Pupil Size</td>
<td>Normal ((^4))</td>
</tr>
<tr>
<td>Reaction to Light</td>
<td>Slow</td>
</tr>
<tr>
<td>Pulse Rate</td>
<td>Up</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Up or Down ((^5))</td>
</tr>
<tr>
<td>Temperature</td>
<td>Up, Down or Normal</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Normal or Flaccid</td>
</tr>
</tbody>
</table>

\(^4\) Normal but may be dilated  
\(^5\) Down with anesthetic gases – Up with volatile solvents and aerosols

---

### F. Classification Exemplar

**Inhalants**

- Present
- Up with anesthetic gases
- Down with volatile solvents and aerosols
- Slow
- Up
- Up or Down
- Up, Down or Normal
- Normal or Flaccid

---

Drugs Recognition Expert Course  
Session 19 - Inhalants  
Revised: 10/2015  
Page 16 of 17
Topics for Study
1. What are the three major subcategories of Inhalants?

2. What are some of the principal active ingredients in many volatile substances?

3. In what important respect do the effects of Anesthetic Gases differ from the effects of Volatile Solvents and Aerosols?

4. Do any of the subcategories of Inhalants cause pulse rate to decrease?

5. The effects of Amyl Nitrite and Butyl Nitrite last from a few seconds to up to ______ minutes.
**DRUG INFLUENCE EVALUATION**

**Evaluator:** Sgt. Joe Armstrong  
**Missouri HP**  
DRE # 11850  
**Case #** 14-314775  
Session XIX - #1

**Recorder/Witness:** Tpr. Adams  
Missouri Highway Patrol

**Arrrestee’s Name (Last, First, Middle):** Whippets, Walter L.

**Date of Birth:** 06/08/96  
**Sex:** M  
**Race:** W  
**Arresting Officer (Name, ID#):** Trooper Blaine Adams #7134

**Date Examined / Time Location:** 09/04/14  
**Union PD**

**Date of Birth:** 09/04/14  
**Sex:** M  
**Race:** W

**Chemical Test: Urine Blood**

**Test or tests refused:**

**Breath Results:**

- **Test Refused:** 0.00
- **Instrument #:** 32444

**Blood pressure:** 180/92  
**Temperature:** 98.6°

**Pupil Size:** Normal
- **Equal Vertical Nystagmus**

**Droopy**

**Corrective Lenses:** None

**Contacts if so:**

**Blindness:**
- **Tracking:**
  - **Normal**
  - **Unequal**

**HGN:**
- **Lack of Smooth Pursuit**
- **Maximum Deviation**

**Coordination:**
- **Poor, Unsteady**

**Coordination:**
- **Poor, Unsteady**

**Operation of Vehicle:**
- **In the Park and in my car.**

**Precinct/Station:**
- **2205**
- **2255**

**Evaluation start time:** 2145  
**Evaluation completion time:** 09/04/14 2135  
**Time DRE was notified:** 2105

**Internal clock estimated as 30 seconds**

**Type of footwear:** Skate Shoes

**Tired or Intoxicated:**
- **No**

**Finger to Nose (Draw lines to spots touched):**

**Blood pressure:** 158 / 92

**Muscle tone:** Normal

**Comments:**
- **Gold paint on hands.**

**Blood pressure:** 158 / 92  
**Temperature:** 98.6°

**PUPIL SIZE**

- **Room Light:** 2.0 - 5.0
- **Darkness:** 5.0 - 8.5
- **Direct:** 2.0 - 4.5

- **Left Eye:**
  - **4.0**
  - **7.0**
  - **3.5**

- **Right Eye:**
  - **4.0**
  - **7.0**
  - **3.5**

**Rebound Dilation:**
- **None**
- **Yes**

**Pupillary Unrest:**
- **None**
- **Yes**

**Reaction to Light:**
- **Slow**

**Gold paint on hands.**

**Date / Time of arrest:** 09/04/14  
**Time DRE was notified:** 2135  
**Date of arrest:** 09/04/14  
**Time DRE was notified:** 2105

**Signatures:**

- **Officer’s Signature:**
- **Witness:**

**Opinion of Evaluator:**
- **Not Impaired**
- **Alcohol**
- **CNS Stimulant**
- **CNS Depressant**
- **Hallucinogen**
- **Narcotic Analgesic**
- **Inhalant**
- **Medical**
- **Not Impaired**
- **Alcohol**
- **CNS Stimulant**
- **CNS Depressant**
- **Hallucinogen**
- **Narcotic Analgesic**
- **Inhalant**
- **Medical**
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Whippets, Walter

1. LOCATION: The evaluation was conducted at the Union Police Department.

2. WITNESSES: Sgt. Art Amato of the Union PD recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Trooper Adams for a drug evaluation. Trooper Adams advised he had observed the suspect’s vehicle drifting over the lane divider line and traveling 15 mph under the posted speed on I-50. When stopped, the suspect had extremely bloodshot eyes. He exhibited six clues of HGN, but no alcoholic beverage was detected on his breath. His balance and coordination were poor. He did poorly on the SFST’s and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the Interview Room at the PD. His speech was slurred and he was mumbling his words. He was unsteady while standing and several times he used the wall to steady himself.

6. MEDICAL PROBLEMS AND TREATMENT: None were noted and none reported.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect attempted the test but got dizzy, nearly fell, and the test was stopped for safety reasons. Walk & Turn: While in the instructions stage, the suspect lost his balance three times, and the test was stopped for safety reasons. One Leg Stand: While attempting to stand on his left foot, the suspect put his foot down three times and fell against the wall. The test was stopped for safety reasons. Finger to Nose: For safety reasons the suspect attempted the test seated. He was unable to touch the tip of his nose as directed on all six attempts.

8. CLINICAL INDICATORS: Six clues of HGN and a Lack of Convergence were observed. The suspect’s pulse rates and blood pressure were above the DRE average ranges. His pupil sizes were all within the DRE average ranges.

9. SIGNS OF INGESTION: The suspect had a paint-like odor on his breath. Gold paint residue was located on of his both hands. His nasal area was red and inflamed.

10. SUSPECT’S STATEMENTS: Suspect admitted “huffing some gold” in the park.

11. DRE'S OPINION: In my opinion, the suspect is under the influence of an Inhalant and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS:
Evaluator: Trooper Derek Brown Montana HP  
Arrestee's Name (Last, First, Middle): Poppers, Jack B.  
Date Examined/ Time: 06/24/14 0130  
Recorder:  
Miranda Warning Given: Yes  
Given by: Officer Waln D  
Officer's Signature: D Glasses  
Date of Birth: 09/01/95  
Sex: M  
Race: N.A.  
Arresting Officer (Name, ID#): Officer Brad Waln #18031  
Arresting Officer Agency: Missoula PD  

**Modified Romberg Balance**

- **Walk and Turn Test:**  
  - Cannot keep balance  
  - Starts too soon  
  - Misses heel-toe  
  - Raises arms  
  - Actual steps taken: 9  

- **Finger to Nose:**  
  - Left Eye  
  - Right Eye  

- **Blood pressure:** 144 / 94  
- **Temperature:** 99.0°  

- **Muscle tone:** Rigid  

- **Breath Odor:** Chemical  

- **Chemical Test:** Refused  

**Drug Influence Evaluation**

- **Type of footwear:** Lace-up boots  

- **Nasal area:** Redness, Runny nose  

- **Oral cavity:** Redness  

**Other Observations:**  

- **Wrong hand on attempts 5 and 6.**
Suspect: **Poppers, Jack**

1. **LOCATION:** The evaluation was conducted at the Missoula City Police Department.

2. **WITNESSES:** Sergeant Kurt Sager of the Montana HP recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was requested to contact Officer Waln of the Missoula PD for a drug evaluation. It was determined that he had observed the suspect’s vehicle drifting over the center divider line numerous times on S. 3rd Street. The suspect was slow to respond to questions. He appeared to be confused and disoriented. Officer Waln detected six clues of HGN, but no alcoholic beverage was detected on the suspect’s breath. He performed poorly on the SFST’s and was arrested for DUI. Several empty bottles of “Rush” were located in the suspect’s vehicle.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the interview room at MPD. His speech was slow and slurred. His was having difficulties with his coordination and staggered several times. His eyes were watery and bloodshot.

6. **MEDICAL PROBLEMS AND TREATMENT:** Suspect stated he felt light-headed.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: The suspect had an approximate 4” front to back and side to side sway. He estimated 30 seconds in 38 seconds. Walk & Turn: The suspect lost his balance three times during the instructions, stopped while walking three times, missed heel to toe four times, and stepped off the line three times, and staggered when he turned. One Leg Stand: After putting his right foot down three times and nearly falling, the test was stopped. Finger to Nose: Suspect touched the tip of his nose on one of the six attempts. He also used the wrong hand on attempts #5 and #6.

8. **CLINICAL INDICATORS:** The suspect had six clues of HGN with a 35 degree angle of onset. Lack of Convergence was also present. His pupils were within the DRE average ranges. His pulse rates and blood pressure were above the DRE averageranges.

9. **SIGNS OF INGESTION:** Suspect had a chemical odor on his breath and a red nasal area.

10. **SUSPECT’S STATEMENTS:** Suspect admitted being around some friends using “Locker Room” and “Rush” but claimed he didn’t use any because it made him light-headed.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of an Inhalant and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A blood sample was collected from the suspect.

13. **MISCELLANEOUS:**
**DRUG INFLUENCE EVALUATION**

**Evaluator:** Trooper Mark Griggs  
**Iowa State Patrol**  
**DRE #** 8102  
**Rolling Log #** 14-06-188  
**Case #** 14-80975  
**Session XIX - #2**

**Recorder/Witness:** Trooper Todd Olmstead  
**Iowa State Patrol**  
**Date Examined:** 06/14/14  
**Time Examined:** 2135  
**Location:** Des Moines PD  
**Test Refused:** No  
**Chemical Test:** Test or tests refused  
**Blood:** N/A

**Witness Crash:** 1:8:1 None  
**Arresting Officer (Name, ID#):** Officer Mike Dixson #15801  
**Date of Birth:** 09/10/95  
**Sex:** F  
**Race:** White  
**Height:** 5'10"  
**Weight:** 175 lbs  
**Occupation:** Driver  
**Drug or Medication:** None  
**History:** No  
**Refractive Error:** None  
**Corrective Lenses:** None  
**Pupil Size:** Equal  
**Vertical Nystagmus:** No  
**Convergence:** Present  
**Coordination:** Poor  
**Attitude:** Cooperative, Indifferent  
**Speech:** Slurred, Rambling  
**Breath Odor:** Chemical-like  
**Eye:** Reddened Conjunctiva  
**Eyes:** Normal  
**Vertical Nystagmus:** No  
**Coordination:** Poor  

**Blood pressure:** 110/62  
**Temperature:** 98.0°  
**Muscle tone:** Normal  
**Nasal area:** Redness  
**Oral cavity:** Red  
**Reaction to Light:** Slow

**Right Eye**  
- **Heel-Toe:** Yes  
- **Step Off Line:** No  
- **Raise Arms:** Yes  
**Right Count:** 9  
**Left Count:** 9

**Left Eye**  
- **Heel-Toe:** Yes  
- **Step Off Line:** No  
- **Raise Arms:** Yes  
**Right Count:** 9  
**Left Count:** 9

**Glasses:** None  
**Contacts, if so:** Soft  
**Pupillary Unrest:** Rebound  
**Pupil Size:** 5.0  
**Red:** Yes  
**Reaction to Light:** Slow  
**Type of footwear:** Athletic running shoes

**Room Light**  
- **Left Eye:** 5.0  
- **Right Eye:** 5.0

**Darkness**  
- **Left Eye:** 7.5  
- **Right Eye:** 7.5

**Direct**  
- **Left Eye:** 3.5  
- **Right Eye:** 3.5

**Internal clock estimated as 30 seconds**  
- **Describe Turn:** Slow and deliberate  
- **Cannot do test (explain):** Pronounced sway.

**Finger to Nose**  
- **Left Eye:** Rebound  
- **Right Eye:** Rebound  

**Modified Romberg Balance**  
- **5.0 5.0 5.0**

**Walk and Turn Test**  
- **1st Nine:** Present  
- **2nd Nine:** Present  
- **3." 3." 3."**

**HGN**  
- **Right Eye:** Present  
- **Left Eye:** Present  
- **Lack of Smooth Pursuit:** Yes  
- **Maximum Deviation:** Yes  
- **Angle of Onset:** 35  
- **Right Count:** 9  
- **Left Count:** 9

**Convergence**  
- **Present:** Right eye  
- **Present:** Left eye

**PUPIL SIZE**  
- **Room Light:** 2.6 - 6.0  
- **Darkness:** 5.0 - 8.5  
- **Direct:** 2.0 - 4.5

**Actual steps taken:** 9  
**Test stopped after she nearly fell.**

**Finger to Nose (Draw lines to spots touched)**

**Right Arm**  
- **Type of footwear:** Athletic running shoes

**Left Arm**  
- **Type of footwear:** Athletic running shoes

**Blood pressure:** 110/62  
**Temperature:** 98.0°  
**Muscle tone:** Normal  
**Rigid Comments:**

**What drugs or medications have you been using?**  
- **"I did a little nitrous with friends."**  
- **"I don't remember."**

**How much?**  
- **"About 8 pm?"**

**Where were the drugs used? (Location):** At friends house.

**Date / Time of arrest:** 06/14/14 2055  
**time DRE was notified:** 2115  
**Evaluation start time:** 2135  
**Evaluation completion time:** 2215  
**Precinct/Station:** Des Moines PD  

**Opinion of Evaluator:**  
- **Not Impaired**  
- **Alcohol**  
- **CNS Stimulant**  
- **Dissociative Anesthetic**  
- **Inhalant**  
- **Medical**  
- **CNS Depressant**  
- **Hallucinogen**  
- **Narcotic Analgesic**  
- **Cannabis**

**Officer's Signature:**  
**DRE #:**  
**Reviewed/approved by:**  
**Date:**

**What drugs or medications have you been using?**  
- **"I did a little nitrous with friends."**  
- **"I don't remember."**

**How much?**  
- **"About 8 pm?"**

**Where were the drugs used? (Location):** At friends house.

**Date / Time of arrest:** 06/14/14 2055  
**time DRE was notified:** 2115  
**Evaluation start time:** 2135  
**Evaluation completion time:** 2215  
**Precinct/Station:** Des Moines PD  

**Opinion of Evaluator:**  
- **Not Impaired**  
- **Alcohol**  
- **CNS Stimulant**  
- **Dissociative Anesthetic**  
- **Inhalant**  
- **Medical**  
- **CNS Depressant**  
- **Hallucinogen**  
- **Narcotic Analgesic**  
- **Cannabis**

**Officer's Signature:**  
**DRE #:**  
**Reviewed/approved by:**  
**Date:**

**What drugs or medications have you been using?**  
- **"I did a little nitrous with friends."**  
- **"I don't remember."**

**How much?**  
- **"About 8 pm?"**

**Where were the drugs used? (Location):** At friends house.
Suspect: **Huffer, Misty**

1. **LOCATION:** The evaluation was conducted at the Des Moines Police Department.

2. **WITNESSES:** Trooper Todd Olmstead of the Iowa SP recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was requested to contact Officer Dixson for a drug evaluation at the Des Moines PD. Officer Dixson advised he had observed the suspect fail to stop at red light on Hickman Road. The suspect was slow to respond to his emergency lights, and was unable to maintain a signal lane of travel. The suspect had six clues of HGN, but no alcoholic beverage was detected on her breath. She was unable to perform the SFST’s as directed and was arrested for DUI.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the Interview Room. She appeared to be disoriented and she responded very slowly to questions. Her speech was slurred and rambling. Her face was flushed, and she had bloodshot eyes.

6. **MEDICAL PROBLEMS AND TREATMENT:** None noted or stated.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: The suspect swayed approximately 3” front to back and side to side. She estimated 30 seconds in 22 seconds. Walk & Turn: Suspect lost her balance twice during the instructions, started too soon once, stopped while walking five times, and raised her arms for balance seven times. One Leg Stand: Suspect put her foot down twice while standing on her left foot. She nearly fell while attempting to stand on her right foot and the test was stopped for safety reasons. Finger to Nose: Suspect missed the tip of her nose on all six attempts, and had a pronounced sway.

8. **CLINICAL INDICATORS:** The suspect had six clues of HGN and exhibited an early onset of nystagmus. Lack of Convergence was also present. The suspect’s pulse rates were elevated. Her blood pressure was below the DRE average ranges.

9. **SIGNS OF INGESTION:** Suspect’s nasal and oral cavities were red and inflamed.

10. **SUSPECT’S STATEMENTS:** The suspect stated she did “a little nitrous with some friends” just before she got stopped. She said she likes it because it relaxes her.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of an Inhalant and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A urine sample was collected from the suspect.

13. **MISCELLANEOUS:** Two empty Nitrous Oxide containers were located in her vehicle.
Session 20
Practice: Vital Signs Examinations
Learning Objectives

- Conduct examinations of pulse, blood pressure and temperature
- Describe the vital signs examination procedures
- Document the results of the vital signs examinations

Upon successfully completing this session the participant will be able to:

- Conduct examinations of pulse, and blood pressure.
- Describe the vital signs examination procedures.
- Document the results of the vital signs examinations.

CONTENT SEGMENTS .................................................. LEARNING ACTIVITIES
A. Procedures for this Session ............................................ Instructor-Led Presentations
B. Pulse Measurements .................................................... Participant Hands-On Practice
C. Blood Pressure Measurements ......................................... Instructor-Led Coaching
D. Temperature
E. Session Wrap-Up ......................................................... Participant-Led Coaching
A. Procedures for this Session

Team Assignments

Participants will work in three or four member teams.

At any given time, one member of the team will be engaged in conducting and recording vital signs examinations of another member.

The remaining member(s) will help coach and critique the participant who is conducting the examinations.

Participants will take turns serving as test administrator, test subject, and coach.

Participants will record their measurements using the Vital Signs Examination Data Sheet.
B. Pulse Measurements

_Vital Signs Practice_

Teams initially will practice taking one another’s pulse.

---

**Pulse Measurements**

---

C. Blood Pressure Measurements

---

D. Temperature

---
E. Session Wrap-Up
# Vital Signs Examinations Data Sheet

**Examiner's Name:** ________________________________

**Date:** _______ / ______ / ______

### Pulse Measurements

<table>
<thead>
<tr>
<th>Subject's Name</th>
<th>Time</th>
<th>Pulse Point Used</th>
<th>Beats Per Minutes</th>
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<tr>
<th>Subject's Name</th>
<th>Time</th>
<th>Pulse Point Used</th>
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<tbody>
<tr>
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<table>
<thead>
<tr>
<th>Subject's Name</th>
<th>Time</th>
<th>Pulse Point Used</th>
<th>Diastolic</th>
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### Blood Pressure Measurements

<table>
<thead>
<tr>
<th>Subject's Name</th>
<th>Time</th>
<th>Pulse Point Used</th>
<th>Systolic</th>
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<table>
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<tr>
<th>Subject's Name</th>
<th>Time</th>
<th>Pulse Point Used</th>
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Revised: Drug Recognition Expert Course
10/2015 Vital Signs Examination Data Sheet Page 1 of 1
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Session 21
Cannabis
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Upon successfully completing this session the participant will be able to:

- Explain a brief history of Cannabis.
- Identify common names and terms associated with Cannabis.
- Identify common methods of administration for Cannabis.
- Describe the symptoms, observable signs and other effects associated with Cannabis.
- Describe the typical time parameters, i.e. onset and duration of effects associated with Cannabis.
- List the clues that are likely to emerge when the drug influence evaluation is conducted for a person under the influence of Cannabis.
- Correctly answer the “topics for study” questions at the end of this session.

CONTENT SEGMENTS ................................................................................................................ LEARNING ACTIVITIES
A. Overview of the Category ................................................................. Instructor-Led Presentations
B. Possible Effects of Cannabis ........ Review of the Drug Evaluation and Classification Exemplars
C. Onset and Duration of Effects ............................................................. Reading Assignments
D. Overdose Signs and Symptoms............................................................ Video Presentation
E. Expected Results of the Evaluation....................................................... Slide Presentations
F. Classification Exemplars

Revised: 10/2015
Drug Recognition Expert Course
Cannabis
Session 21
Page 1 of 18
A. Overview of the Category

“Cannabis” is a category of drugs derived primarily from various species of plants, such as Cannabis Sativa, which generally grow tall and thin, outdoors, and Cannabis Indica plants, which generally grow short and wide, and are better grown indoors. Cannabis grows readily throughout the temperate zones of the world.

It has been cultivated for centuries.

Example: At the first English settlement in America, Jamestown, VA, it was grown to produce hemp.

The primary psychoactive ingredient in Cannabis is Delta-9 Tetrahydrocannabinol.

THC is found principally in the leaves and flowers of the plant, rather than in the stem or branches.

Different varieties of the Cannabis have different concentrations of THC.

Source: Drug Identification Bible, 20014/2015.

One variety that has a relatively high concentration of THC is Sinsemilla, which is the unfertilized female Cannabis Sativa plant.

Explanatory note: “Sinsemilla” in Spanish means “without seeds.”
Forms of Cannabis

There are four principal forms of Cannabis.

- Marijuana – the dried leaves of the plant.
- Hashish – a form of Cannabis made from the dried and pressed resin of a marijuana plant.
- Hash Oil – sometimes referred to as “marijuana oil,” it is a highly concentrated syrup-like oil extracted from Marijuana. It is normally produced by soaking Marijuana in a container of solvent, such as acetone or alcohol for several hours until the solvent has evaporated. A thick syrup-like oil is produced with a higher THC content. The average THC content of hash oil seized in the U.S. in 2010 was 30.3%.
- Marinol (or Dronabinol) – a synthetic form of THC. This is a prescription drug used to treat nausea and vomiting. It is prescribed for certain cancer patients undergoing chemotherapy.
  - “Dronabinol” is the generic or chemical name for the synthetic THC.
  - “Marinol” is a trade name for Dronabinol.
  - “Nabilone – an analog of Dronabinol used as an anti-vomiting agent. Trade name: Cesamet"
Sources indicate that “waxy marijuana or wax marijuana is the purest form of cannabis. It contains anywhere from 82-99% THC making it several times more potent than a marijuana bud on a cannabis plant which usually contains 5-28% THC. One hit of wax is supposedly equal to 1-2 full cannabis joints and is reported as being more clear and longer lasting than average marijuana. Wax marijuana is also a medical marijuana product. Typical wax marijuana is golden in color and crumbly; though texture may vary based on type.”
**Synthetic Cannabinoid Products**

Synthetic cannabinoid products typically include olive colored herbs, combination of herbs, or plant materials enhanced with a delta-9-tetrahydrocannabinol (THC) synthetic analog. When smoked, synthetic cannabinoid products can produce stimulant and/or hallucinogenic effects.

**Synthetic Cannabinoid Products Effects**

They have many adverse effects that include:

- Panic attacks
- Agitation
- Tachycardia (range of 110 to 150 BPM)
- Elevated blood pressure
- Anxiety
- Pallor (pale appearance)
- Numbness and tingling

User report effects lasting between 30 minutes and 2 hours.

Common brand names for synthetic cannabinoids include K2, Spice, Spice Gold, Spice Diamond, Yucatan fire, Solar Flare, K2 Summit, Genie, PEP Spice, and Fire n Ice, to name a few.
Possible Cannabis Applications

Cannabis may have some limited medical applications, however, many experts vary in their opinions on them. Some possible applications may include:

- Lowering of intraocular pressure, which can be helpful for glaucoma patients. "Intraocular" – within the eyeball.
  Cannabis lowers the intraocular pressure by dilating in size the blood vessels of the eyes (more size – less pressure). This causes reddening of the conjunctiva. Conjunctiva is the clear membrane of the sclera (white portion of the eye) and lines the inside of the eyelids and is made of lymphoid tissue. Conjunctivae refers to both eyes. Conjunctiva is singular.

- Suppressing nausea, and sometimes is recommended for cancer patients to relieve the nausea accompanying chemotherapy.

- Cannabidiol, a non-psychoactive ingredient found in Cannabis, is used in treating Epilepsy; it helps to inhibit seizures.
**Potency, Purity and Dose**

Average THC concentration in marijuana:

- Marijuana – 13.0% (2013)
- Hash – 30 – 50% (2013)
- Hash Oil – 68.4% (2013)
- Concentrates - Vary

*Source: Drug Identification Bible, 2014/2015*

**THC levels can vary greatly depending upon areas of the country.**

Recreational doses are highly variable.

The lower the THC, the more hits required to achieve desired effects.

Marijuana usually is smoked.

Marijuana, Hash and Hash Oil also can be ingested orally, for example, baked in cookies or brownies and eaten.

THC can also be absorbed through the skin using transdermal absorption patches.

Research related to passive inhalation of marijuana smoke causing behavioral effects as well as measurable amounts in toxicology samples is mixed, and is generally dependent on the amount of smoke inhaled.

*Source: Drug Identification Bible, 2014/2015*
B. Possible Effects of Cannabis

One major effect of Cannabis is that it appears to interfere with a person’s ability to divide attention.

People under the influence of Cannabis have difficulty paying attention, with brief attention spans.

In particular, they do not divide their attention very successfully.

Clarification: They have a difficult time dealing with more than one or two tasks at once.

This can make them very unsafe drivers, since driving requires the ability to divide attention among many simultaneous tasks.

Loss of depth perception would be demonstrated by stopping improperly.

Short attention span would be indicated by erratic speeds, failing to maintain a single lane and stopping for a red light then continuing on.

People under the influence of Cannabis may attend to one or a few of these driving tasks, but simply ignore the other tasks.

Because Cannabis impairs attention, Standardized Field Sobriety Tests like Walk and Turn and One Leg Stand are excellent tools for recognizing people under the influence of Cannabis.
Effects of Cannabis:

Effects will vary with dose, route of administration, experience of user, and other factors. At recreational doses, effects include:

- Relaxation
- Euphoria
- Relaxed inhibitions
- Disoriented
- Altered time and distance perception
- Lack of concentration
- Impaired memory
- Alterations in thought process
- Drowsiness
- Sedation
- Mood Changes

Other characteristic indicators:

- Odor of Marijuana
- Marijuana debris in the mouth
- Possible green coating on the tongue
- Reddening of the conjunctivae
- Body tremors
- Eyelid tremors

Revised: Drug Recognition Expert Course  
10/2015 Cannabis  
Page 9 of 18
C. Onset and Duration of Effects

Effects from smoking Cannabis are felt within minutes and reach their peak in 10-30 minutes. Typical marijuana smokers experience a high that lasts approximately 2 hours. Most behavioral and physiological effects return to baseline within 3-5 hours after drug use, although some residual effects in specific behaviors can last up to 24 hours.

The effects reach their peak within 10–30 minutes.

- A 1985 Stanford University study showed that pilots had difficulty in holding patterns and in lining up with runways for up to 24 hours after using Marijuana.

Depending on the amount smoked and on the concentration of THC in the Marijuana, the person will continue to feel and exhibit the effects for 2–3 hours.

- In 1990, a second Stanford University study showed: Marijuana impaired performance at .25, 4, 8, and 24 hours after smoking. While 7 of the 9 pilots showed some degree of impairment at 24 hours after smoking Cannabis, only one reported any awareness of the drug's effects.

Generally, the person will feel “normal” within 3–5 hours after smoking Marijuana.

- The user may be impaired long after the euphoric feelings have ceased.
- Blood tests may disclose Marijuana use for at least 3 days after smoking.
- Urine tests may indicate the presence of metabolites of THC for a month or more.
There are two important metabolites, or chemical byproducts of THC.

- Hydroxy THC, which causes the user to feel euphoric.
- Carboxy THC, there is no evidence at this time that it is psychoactive.
- Hydroxy THC usually is eliminated from the blood plasma within six hours.
- Carboxy THC may be found in the blood plasma for several days following Marijuana use.

Cannabis is a fat soluble (i.e. it dissolves easily into fatty tissue); therefore, it can remain for long periods in the brain tissue, which is about one-third fat.

Cannabis principally is eliminated from the body in feces and urine.
D. Overdose Signs and Symptoms

Excessive or long term use of Marijuana can have very undesirable consequences. Marijuana has been observed to produce sharp personality changes, especially in adolescent users. Overdose signs and symptoms can include paranoia and possible psychosis.

Long term effects include:

- Lung damage
- Chronic Bronchitis
- Lowering of Testosterone (male sex hormone)
- Possible birth defects, still births and infant deaths
- Acute anxiety attacks
- Chronic reduction of attention span

Research indicates that life threatening overdoses rarely if ever occur.

Withdrawal – is similar to alcohol dependence withdrawal

Physical dependence can occur with chronic use
Evaluation of Subjects
Under the Influence of Cannabis

- HGN - None
- VGN - None
- Lack of Convergence - Present
- Impaired performance will be evident on Modified Romberg Balance, Walk and Turn, One Leg Stand and Finger to Nose

E. Expected Results of the Evaluation

Observable Evidence of Impairment

Clinical Indicators

- Neither Horizontal Gaze nor Vertical Gaze Nystagmus will be present.
- Lack of Convergence generally will be present.
- Performance on the Modified Romberg Balance, Walk and Turn, One Leg Stand, and Finger to Nose tests will be impaired.
Evaluation of Subjects Under the Influence of Cannabis

Vital Signs:
- Pulse - Up
- Blood pressure - Up
- Body temperature - Normal
- Muscle tone - Normal

Dark Room:
- Pupil size - Dilated (6)
- Pupil reaction to light - Normal

(6) Possibly normal

Vital Signs:
- Pulse generally will be elevated.
- Blood pressure generally will be elevated.
- Body temperature will be normal.
- Muscle tone will be normal.

Pupil size generally will be dilated or possibly normal (within DRE average ranges).
- The content and potency could effect pupil size. The higher THC content will increase the likelihood of pupil dilation. However, Cannabis does not cause pupil constriction.
- Government-grown Cannabis has low THC levels. Studies using it tend to show a normal range for pupil size.

Pupil reaction to light will be normal.
DREs report a phenomenon termed “Rebound Dilation” in subjects under the influence of Cannabis.

Clarification: “Rebound Dilation” is a period of pupillary constriction followed by a period of pupillary dilation where the pupil steadily increases in size and does not return to its original constricted size.
General Indicators

- Body tremors
- Disoriented
- Debris in the mouth (possible)
- Eyelid tremors
- Altered perception of time and distance
- Increased appetite
- Marked reddening of eyes

Visine causes vasoconstriction in the eyes and is often used to reduce reddening.

- Odor of Marijuana
- Possible paranoia
- Relaxed inhibitions
- Lack of concentration
- Impaired memory
- Alterations in thought process
- Drowsiness

Source: Drugs and Human Performance Fact Sheets, April 2014
Symptomatology Matrix

F. Classification Exemplar
TOPICS FOR STUDY

1. What is the active ingredient in Cannabis?

2. Why are the Walk and Turn and the One Leg Stand tests excellent tools for recognizing persons under the influence of Marijuana?

3. What is Marinol?

4. What is Sinsemilla?

5. Name two important metabolites of THC, and describe how they affect the duration and perception of the effects of Cannabis.
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Blunt, Mary Jane

1. LOCATION: The evaluation was conducted at the Toms River Police Department.

2. WITNESSES: Trooper Thomas Snyder of the NJ State Police recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was a 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was contacted and requested to meet Trooper Gibson at the Toms River PD for a drug evaluation. Trooper Gibson advised he stopped the suspect after observing her vehicle westbound on Hwy 37 drifting out of her traffic lane numerous times. When stopped, the suspect seemed unconcerned about her driving. She told Trooper Gibson that she was just tired and trying to make it home. An odor of marijuana was detected coming from her vehicle. The suspect had difficulty performing the SFST’s, and she was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the Interview Room at the PD. She was laughing, and several times said, “You know I’m not drunk.” She appeared lethargic and carefree acting. She had a noticeable reddening of the conjunctiva.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Suspect had a circular sway of approximately 3” and estimated 30 seconds in 43 seconds. Eyelid tremors were present. Walk & Turn: She lost her balance once during the instructions stage. She missed touching heel to toe three times on the first nine steps and four times on the second nine steps. She raised her arms for balance and at the turn, she stopped, and asked what she was supposed to do. One Leg Stand: She swayed while balancing, and used her arms to balance on both attempts. She laughed several times while trying to complete the test, and leg tremors were present. Finger to Nose: The suspect missed the tip of her nose on four of the six attempts.

8. CLINICAL INDICATORS: Suspect’s pupils were above the DRE average ranges. Rebound dilation and LOC were present. Her pulse and B/P were above the DRE ranges.

9. SIGNS OF INGESTION: The suspect had a greenish coating on her tongue.

10. SUSPECT’S STATEMENTS: Suspect stated, “I smoke a little pot. What’s the big deal?”

11. DRE’S OPINION: In my opinion, the suspect is under the influence of Cannabis and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:
**DRUG INFLUENCE EVALUATION**

**Evaluator:** Sgt. Robert Hayes, Albany PD

**Recorder:** [Blank]

**Witness:** Sgt. Tim Plummer, Oregon State Police

**Arrestee's Name:** Taker, Bud

**Date Examined:** 09/21/14

**Time:** 1945

**Location:** Linn Co. Jail

**DRE#:** 6606

**Crash:** No

**Fatal:** No

**Injury:** No

**Property:** [Blank]

**Arresting Officer:** Sr. Trooper Steve Webster, Oregon State Police

**Sex:** M

**Race:** W

**Date of Birth:** 02/21/85

**Breath Results:** 0.00

**Rolling Log#**

**Case#**

14-09-025 14-42845 Session XXI - #2

**Chemical Test:** Urine

**Test refused:** [Blank]

**Test or tests refused:** [Blank]

**Instrument#**

66454

**Test refused:** [Blank]

**Test or tests refused:** [Blank]

**Miranda Warning Given:** Yes

**Given by:** Sr. Tpr. Webster

**What have you eaten today? When?** Couple burgers 5 pm

**What have you been drinking? How much?** Coke N/A

**Time of last drink?** N/A

**Time now**

**Actual When did you last sleep? How long?** About 6 pm?/1950 Last night About 7-8 hours

**Are you sick or injured? Are you diabetic or epileptic?** Yes No

**Do you take insulin?** [Blank]

**Do you have any physical defects? Are you under the care of a doctor or dentist?** Yes No

**Are you taking any medication or drugs?** "I smoke pot." Carefree, Cooperative

**Attitude:** Slow

**Face:** Normal

**Corrective Lenses:** None

**Glasses**

- [ ] Soft
- [ ] Hard
- [ ] Contacts, if so

**Pupil Size:** Equal

**Unusual (explain):** [Blank]

**Breath Odor:** Marijuana

**Corrective Lenses:** None

**Eyes:** Reddened

**Conjunctiva:** Blindness:

- [ ] None
- [ ] Left
- [ ] Right
- [ ] Equal
- [ ] Unequal

**HGN**

- Right Eye
- Left Eye

**Convergence**

- Right eye
- Left eye

**Modiﬁed Romberg Balance**

- Walk and Turn Test
- Circular sway. Eyelid tremors

- Internal clock
- estimated as 30 seconds

**Finger to Nose**

(Draw lines to spots touched)

**PUPIL SIZE**

- Room Light
- Darkness
- Direct

- 2.5 - 5.0
- 5.0 - 8.5
- 6 - 7.5
- 8 - 10

**Left Eye**

- 6.5
- 9.0
- 6 - 7.5

**Right Eye**

- 6.5
- 9.0
- 6 - 7.5

**Rebound Dilation:**

- [ ] Yes
- [ ] No

**Pupillary Unrest:**

- [ ] Yes
- [ ] No

**Reaction to Light:**

- Slow

**Type of footwear:** Lace-up boots

**Nail area:** Clear

**Oral cavity:** Dry mouth.

**Green Coating.**

**Cannabis**

**Blood pressure**

148 / 100

**Temperature**

98.4°F

**Muscle tone:**

- [ ] Normal
- [ ] Rigid
- [ ] Flaccid

**Comments:**

- What drugs or medications have you been using? "Pot. It's legal man."
- How much? "About a bowl."
- Time of use? About 4 or 5 pm
- Where were the drugs used? (Location) In my car

**Date/Time of arrest:** 09/21/14

**Time DRE was notified:** 1905

**Evaluation start time:** 1930

**Evaluation completion time:** 2040

**Precinct/Station:** 1945

**Officer's Signature:** [Blank]

**Review/approved by/date:** [Blank]

**Opinion of Evaluator:**

- [ ] Not impaired
- [ ] Alcohol
- [ ] CNS Stimulant
- [ ] Dissociative Anesthetic
- [ ] Inhalant
- [ ] Narcotic Analgesic
- [ ] Hallucinogen
- [ ] Miscellaneous

**Medical**

- [ ] Not impaired
- [ ] Alcohol
- [ ] CNS Stimulant
- [ ] Dissociative Anesthetic
- [ ] Inhalant
- [ ] Narcotic Analgesic
- [ ] Hallucinogen
- [ ] Miscellaneous

**Narcotic Analgesic**

- [ ] Not impaired
- [ ] Alcohol
- [ ] CNS Stimulant
- [ ] Dissociative Anesthetic
- [ ] Inhalant
- [ ] Narcotic Analgesic
- [ ] Hallucinogen
- [ ] Miscellaneous

**Hallucinogen**

- [ ] Not impaired
- [ ] Alcohol
- [ ] CNS Stimulant
- [ ] Dissociative Anesthetic
- [ ] Inhalant
- [ ] Narcotic Analgesic
- [ ] Hallucinogen
- [ ] Miscellaneous

**Miscellaneous**

- [ ] Not impaired
- [ ] Alcohol
- [ ] CNS Stimulant
- [ ] Dissociative Anesthetic
- [ ] Inhalant
- [ ] Narcotic Analgesic
- [ ] Hallucinogen
- [ ] Miscellaneous

**Opinion of Evaluator:**

- Not impaired
- Alcohol
- CNS Stimulant
- Dissociative Anesthetic
- Inhalant
- Narcotic Analgesic
- Hallucinogen
- Miscellaneous

- Medical
- Narcotic Analgesic
- Hallucinogen
- Miscellaneous

- Overall opinion:
- Not impaired
- Alcohol
- CNS Stimulant
- Dissociative Anesthetic
- Inhalant
- Narcotic Analgesic
- Hallucinogen
- Miscellaneous

- Medical
- Narcotic Analgesic
- Hallucinogen
- Miscellaneous

- Overall opinion:
- Not impaired
- Alcohol
- CNS Stimulant
- Dissociative Anesthetic
- Inhalant
- Narcotic Analgesic
- Hallucinogen
- Miscellaneous
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Toker, Bud

1. LOCATION: The evaluation was conducted at the Linn County Jail.

2. WITNESSES: Sgt. Tim Plummer of the Oregon State Police recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Sr. Trooper Webster at the Linn County Jail for a drug evaluation. It was determined that the suspect had been reported as a possible DUI and was unable to maintain a single lane of travel on I-5. When contacted by Sr. Tpr. Webster, the suspect appeared relaxed, carefree, and was unconcerned about being stopped. He had poor balance and coordination, had difficulty completing the SFST’s, and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the Booking Room at the jail. He was lethargic acting and appeared relaxed. He was unsteady on his feet and was swaying as he stood. His eyes appeared to be bloodshot and his pupils were dilated.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Suspect had an approximate 3” circular sway, and estimated 30 seconds in 18 seconds. Walk & Turn: Suspect lost his balance during the instructions stage and stopped while walking twice. He missed touching heel to toe twice on the first nine steps, and once on the second nine steps. One Leg Stand: Suspect swayed while balancing, used his arms for balance, and put his foot down once while standing on each foot. Leg tremors were present during both tests. Finger to Nose: Suspect missed the tip of his nose on all six attempts, and exhibited eyelid tremors.

8. CLINICAL INDICATORS: Suspect had a Lack of Convergence and Rebound Dilation. His pupils were dilated in all three lighting levels and were above the DRE average ranges. His pulse rates and blood pressure were elevated and also above the DRE average ranges.

9. SIGNS OF INGESTION: The suspect had a green coating on his tongue and a dry mouth.

10. SUSPECT’S STATEMENTS: When asked about smoking marijuana, the suspect stated, “Hey, its legal man.” He admitted smoking about a bowl of marijuana earlier in the day.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of Cannabis and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS: Suspect was also charged with possession of marijuana.
Suspect: Duby, Sharon A.

1. **LOCATION:** The evaluation was conducted at the Lynnwood Police Department.

2. **WITNESSES:** Sergeant Mark Crandall of the WSP recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was requested to contact Officer Breault for a drug evaluation. Officer Breault advised that he arrested the suspect after her vehicle had rear-ended another vehicle at a stop light on Highway 99. An odor of marijuana was detected coming from the suspect’s vehicle. The suspect had poor balance and coordination. She was unable to complete SFST’s as directed. She possessed a medical marijuana card and admitted smoking marijuana prior to the crash.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the interview room at LPD. She appeared to be very relaxed and had a dazed appearance. She was unstable on her feet and several times used a chair to steady herself.

6. **MEDICAL PROBLEMS AND TREATMENT:** Suspect advised that she experiences occasional migraine headaches and uses medical marijuana as treatment for them.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: Suspect had an approximate 2” front to back and side to side sway. She estimated 30 seconds in 22 seconds. Walk & Turn: Suspect lost her balance once during the instructions stage, stopped while walking twice, stepped off the line once, and raised her arms for balance five times. Leg tremors were present throughout the test. One Leg Stand: Suspect swayed while balancing, and used her arms for balance. Leg tremors were present throughout the test. Finger to Nose: The suspect missed the tip of her nose on four of the six attempts using the pads of her fingers. She exhibited eyelid tremors throughout the test and laughed out loud several times.

8. **CLINICAL INDICATORS:** Lack of Convergence was present. Her pupils were dilated, and above the DRE average ranges in Room Light and Direct Light. Her pulse rates were elevated and above the DRE average ranges.

9. **SIGNS OF INGESTION:** The suspect had a green coating on her tongue with heat bumps.

10. **SUSPECT’S STATEMENTS:** Suspect stated she smokes medical marijuana daily.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of Cannabis and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A blood sample was collected from the suspect.

13. **MISCELLANEOUS:** The suspect was in possession of a valid medical MJ card.

Rev. 10/15
Session 22

Overview of Signs and Symptoms
[This page is intentionally left blank]
<table>
<thead>
<tr>
<th>MAJOR INDICATOR</th>
<th>POSSIBLE EFFECTS</th>
<th>CNS DEPRESS</th>
<th>CNS STIM.</th>
<th>HALLUC</th>
<th>DISS. ANESTETIC</th>
<th>NARC ANALGESIC</th>
<th>INHALANT</th>
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<td>MUSCLE TONE</td>
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Upon successfully completing this session the participant will be able to:

- Describe the possible effects that may be observed in each major indicator of drug impairment.
- Identify the effects that will most likely be observed with subjects under the influence of each drug category.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES

A. The Major Indicators and their Possible Effects.............................. Instructor-Led Presentations
B. Effects Associated with the Drug Categories.................................Interactive Discussions
DRE Major and General Indicators

- **Major Indicators: Physiological Indicators**
- **General Indicators: Observational and Behavioral Indicators**

For DRE purposes, Major Indicators are physiological signs that are specifically addressed and are, for the most part, involuntary; reflecting the status of the Central Nervous System homeostasis.

For DRE purposes, General Indicators are behaviors or observations of the subject that are observed and not specifically tested for.

Both are of equal value in making a decision in the totality of the evaluation.
A. The Major Physiological Indicators and Their Possible Effects

**Major Physiological Indicators of Drug Impairment**

The major physiological indicators of drug impairment are (point to the major indicators on the matrix):

- Horizontal Gaze Nystagmus
- Vertical Gaze Nystagmus
- Lack of Convergence
- Pupil Size
- Reaction to Light
- Pulse Rate
- Blood Pressure
- Body Temperature
- Muscle Tone
**Possible Effects: HGN**

Possible effects that might be observed with **Nystagmus**. With Horizontal Gaze Nystagmus, there are only two possible effects that might be observed.

- Either HGN will be **present**;
- Or it will be **none (meaning that it is not present)**.

There is no drug that stops Horizontal Gaze Nystagmus. Some drugs cause HGN to be present, others do not; but there is no drug that “cures” HGN.

**Possible Effects: VGN**

With Vertical Gaze Nystagmus, there are also only two possible effects.

- Either it will be **present**;
- Or it will be **none (meaning that it is not present)**.
Possible Effects: LOC

What effects might we observe with Lack of Convergence?

- For Lack of Convergence, there are also only two possible effects.

- Either Lack of Convergence will be present;
- Or it will be none (meaning that it is not present).

Just as with Nystagmus, there is no drug that “cures” Lack of Convergence.
Possible Effects: Pupil Size

What effects might we observe with Pupil Size?

• For Pupil Size, there are three possible effects

For Pupil Size, there are three possible effects that might be seen.

• The pupils might be normal (within the DRE average ranges);
• Or, the pupils might be dilated;
• Or, they might be constricted.
Possible Effects: Pupil Size

What effects might we observe with the pupils’ Reaction to Light?
- There are three effects that might be observed in the pupils’ Reaction to Light

Possible Effects: Reaction to Light

There are a number of effects that might be observed in the pupils’ Reaction to Light.
- The pupils might react in a normal manner, i.e. by constricting somewhat in one second or less.
- Or, the pupils might react slow, i.e. by constricting somewhat, but requiring more than one second to do so.
- Or, little to none visible.

In some instances, you may observe very little, or no visible Reaction to Light. If there is a visible reaction of the pupils, it is possible that Rebound Dilation was seen.
Possible Effects: Vital Signs

For each of the Vital Signs, there are three possible effects:

- The pulse rate, or blood pressure, or body temperature could be Normal (within the DRE average ranges)
- Or, it could be UP;
- Or, it could be DOWN.
Possible Effects: Muscle Tone

What effects might we observe with muscle tone?

• There are three possible effects that might be seen

Possible Effects: Muscle Tone

Ask participants: What effects might we observe with muscle tone?

For **Muscle Tone**, there are three possible effects that might be seen.

• Normal (meaning nothing unusual)
• Flaccid
• Rigid
B. Effects Associated with the Drug Categories

CNS Depressants

- HGN: present
- VGN: present (i.e. at high doses for that individual)
- Lack of Convergence: present
- Pupil Size: normal (within the average DRE ranges) except Soma, Quaaludes (Methaqualone) and some anti-depressants usually dilate pupils.
- Reaction to Light: slow
- Pulse Rate: down except Quaaludes (Methaqualone), ETOH and possibly some anti-depressants may elevate.
- Blood Pressure: down
- Body Temperature: normal (within the average DRE ranges)
- Muscle Tone: flaccid
CNS Stimulant Effects

CNS Stimulants

- HGN: none (Not present)
- VGN: none (Not present)
- Lack of Convergence: none (Not present)
- Pupil Size: dilated
- Reaction to Light: slow
- Pulse Rate: up
- Blood Pressure: up
- Body Temperature: up
- Muscle Tone: rigid
Hallucinogens

- HGN: none (Not present)
- VGN: none (Not present)
- Lack of Convergence: none (Not present)
- Pupil Size: dilated
- Reaction to Light: normal, certain psychedelic amphetamines may cause slowing.
- Pulse Rate: up
- Blood Pressure: up
- Body Temperature: up
- Muscle Tone: rigid
**Dissociative Anesthetic Effects**

**Dissociative Anesthetics**

- HGN: *present*
- VGN: *present* (i.e. at high doses; however, it is more common to see Vertical Gaze Nystagmus in someone under the influence of a **Dissociative Anesthetic**)
- Lack of Convergence: *present*
- Pupil Size: *normal* (within the DRE average ranges)
- Reaction to Light: *normal*
- Pulse Rate: *up*
- Blood Pressure: *up*
- Body Temperature: *up*
- Muscle Tone: *rigid*
Narcotic Analgesics

- HGN: **none** (Not present)
- VGN: **none** (Not present)
- Lack of Convergence: **none** (Not present)
- Pupil Size: **constricted**
- Reaction to Light: **little or none visible**
- Pulse Rate: **down**
- Blood Pressure: **down**
- Body Temperature: **down**
- Muscle Tone: **flaccid**
Inhalant Effects

**Inhalants**

- HGN: **present**
- VGN: **present** (high dose for that individual)
- Lack of Convergence: **present**
- Pupil Size: **normal (within the DRE average ranges) but may be dilated**
- Reaction to Light: **slow**
- Pulse Rate: **up**
- Blood Pressure: **up/down** (the Volatile Solvents and the Aerosols usually cause blood pressure to be **above the average ranges**; but the Anesthetic Gases can cause blood pressure to be **below the average ranges**, even though they **elevate** the pulse rate)
- Body Temperature: **up/down/normal**
- Muscle Tone: **normal or flaccid**
Cannabis

- HGN: none (not present)
- VGN: none (not present)
- Lack of Convergence: present
- Pupil Size: dilated or possibly normal (within the DRE average ranges)
- Reaction to Light: normal
- Pulse Rate: up
- Blood Pressure: up
- Body Temperature: normal (within the DRE average ranges)
Drug Symptomatology Sources

Literature on LOC was approved for addition into the addendum by the IACP Technical Advisory Panel (TAP), November 2008.
COMPARISON OF DRE SYMPTOMATOLOGY
WITH CROSS SECTION OF DRUG SYMPTOMATOLOGY SOURCES

CNS DEPRESSANTS:

DRE Symptomatology:
Nystagmus       Decreased pulse
Decreased blood pressure Uncoordinated
Disoriented     Sluggish
Thick slurred speech Drunk-like appearance


Nystagmus          Strabismus
Difficulty in visual Accommodation
Vertigo            Gait ataxia
Positive Romberg sign Hypotonia
Dysmetria          Diplopia
Sluggishness       Difficulty in thinking
Slowness, slurring of speech Poor comprehension
Poor memory        Faulty judgement
Emotional lability


Drug Abuse and Dependence, Grinspoon, Lester,MD; Bakalar,James B., Harvard Medical School Mental Health Review No. 1 (1990), page 11: sedative hypnotics same as alcohol and other depressants

Drugs of Abuse, Giannini, A. James, M.D.; Slaby, Andrew E. M.D., Ph.D. Medical Economics Books, Oradell, New Jersey (1989), page 72: Benzodiazepines same as barbiturate effects; pages 247; 292): Barbiturates:
Nystagmus  Depressed pulse
Depressed blood pressure  Diminished concentration
Incoordination  Decreased reaction time


Diagnostic and Statistical Manual of Mental Disorders (Third Ed, Revised), American Psychiatric Association (1987), p. 159

Maladaptive behavioral changes, e.g., disinhibition of sexual or aggressive impulses, mood lability, impaired judgment, impaired social or occupational functioning.

Slurred speech  Incoordination
Unsteady gait  Impairment in attention or memory

CNS STIMULANTS:
DRE Symptomatology:
Dilated pupils  Increased pulse rate
Increased temperature  Increased blood pressure
Body tremors  Restlessness
Excited  Euphoric
Talkative  Exaggerated reflexes
Anxiety  Grinding teeth
Redness to nasal area  Runny nose
Loss of appetite  Insomnia
Increased alertness


Medical Toxicology-Diagnosis and Treatment of Human Poisoning, Ellenhorn, Matthew J., Barceloux, Donald G. Elsevier Science Pub. Co. 1988, Amphetamines, Page 634:
### Mild influence:

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mydriasis</td>
<td>Hyperreflexia</td>
</tr>
<tr>
<td>Restlessness</td>
<td>Talkativeness</td>
</tr>
<tr>
<td>Irritability</td>
<td>Insomnia</td>
</tr>
<tr>
<td>Tremor</td>
<td>Flushing</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>Combativeness</td>
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<tr>
<td>Nausea</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Pallor</td>
<td>Dry mucous membranes</td>
</tr>
</tbody>
</table>

### Moderate:

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperactivity</td>
<td>Confusion</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Tachypnea</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Premature ventricular contraction</td>
</tr>
<tr>
<td>Chest discomfort</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>Profuse diaphoresis</td>
</tr>
<tr>
<td>Mild temperature</td>
<td>Elevation</td>
</tr>
<tr>
<td>Repetitive behavior</td>
<td>Impulsivity</td>
</tr>
<tr>
<td>Panic reactions</td>
<td>Hallucinations</td>
</tr>
</tbody>
</table>

### Serious:

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delirium</td>
<td>Marked Hypertension/Tachycardia</td>
</tr>
<tr>
<td>Hyperreflexia</td>
<td>Convulsions</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Coma</td>
</tr>
</tbody>
</table>

Cocaine, page 650-659

### Early Stimulation:

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euphoria</td>
<td>Garrulity</td>
</tr>
<tr>
<td>Excitement</td>
<td>Apprehension</td>
</tr>
<tr>
<td>Irritable behavior</td>
<td>Mydriasis</td>
</tr>
<tr>
<td>Sudden headache</td>
<td>Nausea</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Dizziness</td>
</tr>
<tr>
<td>Twitching of small muscles</td>
<td>Tics</td>
</tr>
<tr>
<td>Tremor</td>
<td>Jerks</td>
</tr>
</tbody>
</table>
Cocaine psychosis
Elevation of pulse

**Advanced:**

- Convulsions
- Decreased consciousness

Later Stages:

- Hypotension
- Dyspnea et al

---


**Amphetamines and cocaine (CNSS):**

- Dilation of pupils
- Slight tremor
- Agitation

**Drug and Alcohol Abuse, A Clinical Guide to Diagnosis and Treatment**, (3rd Ed., Schuckit, M.D., Mark A. Plenum Medical Book Co, New York 1989, page 99:

**CNSS cause:**

- Dilation of pupils
- Elevation of blood pressure
- Increased body temperature


**Amphetamine:**

- Dilation of pupils
- Blood pressure
- Teeth grinding
- Tremors

---

Revised: Drug Recognition Expert Course
10/2015 Comparison of DRE Symptomology Sources Page 1 of 18
Dilation of pupils      Increased heartbeat
Increased temperature  Similar to amphetamine

**Drug Abuse and Dependence**, Grinspoon, Lester, MD; Bakalar, James B., Harvard Medical School Mental Health Review No. 1 (1990), pages 8 and 10

**Cocaine and Amphetamine:**
- Dilated pupils          Increased pulse
- Increased blood pressure Vasoconstriction
- Agitation tremors       Increased temperature

**Drugs of Abuse**, Giannini, A. James, M.D.; Slaby, Andrew E. M.D., Ph.D. Medical Economics Books, Oradell, New Jersey (1989), page 29

**Amphetamines:**
- Pupil dilation (Mydriasis) Increased pulse rate
- Elevated blood pressure Hyperactive
- Talkative               Irritable
- Restless               Anorexia
- Tremors                Urinary retention
- Teeth grinding (Bruxism) Fidgety, jerky, random motions
- Illogical, loose thoughts

**Page 295: Cocaine:**
- Dilated pupils          Tachycardia
- Increased blood pressure Vasoconstriction
- Hyperpyrexia


**Amphetamine:**
- Increased pulse         Increased blood pressure
- Possibly increased temperature Increased wakefulness
- General increase in psychomotor activity
Cocaine

Mydriasis (dilated pupils); May cause psychosis
Euphoria Agitation


Cocaine:
Maladaptive behavioral changes, e.g., euphoria, fighting, grandiosity, hyper-vigilance, psychomotor agitation, impaired judgment, impaired social or occupational functioning.

Pupillary dilation Tachycardia
Elevated blood pressure Perspiration or chills
Nausea or vomiting Visual or tactile hallucinations

Amphetamine:
Maladaptive behavioral changes, e.g., fighting, grandiosity, hyper-vigilance, psychomotor agitation, impaired judgment, impaired social or occupational functioning.

Pupillary dilation Tachycardia
Elevated blood pressure Perspiration or chills
Nausea or vomiting

HALLUCINOGENS:
DRE Symptomatology:
Dilated pupils Increased pulse rate
Increased blood pressure Increased temperature
Dazed appearance Body tremors
Synesthesia Hallucinations
Paranoia Uncoordinated
Nausea Disoriented
Difficulty in speech Perspiring
Poor perception of time/distance

Pupillary dilation  Increased blood pressure
Tachycardia  Hyperreflexia
Tremor  Nausea
Piloerection  Muscular weakness
Increased body temperature  Hallucinations
Hyper vigilance  Synesthesia
Loss of boundaries

*Medical Toxicology-Diagnosis and Treatment of Human Poisoning*, Ellenhorn, Matthew J., Barceloux, Donald G. Elsevier Science Pub. Co. 1988, LSD, pages 667-669:

Pupillary dilation  Increased heart rate
Increased body temperature  Piloerection
Weakness  Tremor
Hyperreflexia  Ataxia
Hallucinations  Depersonalization
Poor judgment  Mood swings


*Drug and Alcohol Abuse, A Clinical Guide to Diagnosis and Treatment*, (3rd Ed.), Schuckit, M.D., Mark A. Plenum Medical Book Co, New York 1989 page 160:

Dilated pupils  Increased blood pressure
Increased awareness  Faltered body images
Sensory input  Fine tremor
Flushed face  Increased body temperature


Hallucinogens:
Dilated pupils  Increased heart rate
Increased blood pressure  Increased temperature
Profuse perspiration  Loss of appetite

Revised: 10/2015

Drug Recognition Expert Course
Comparison of DRE Symptomology Sources
Hallucinations

**Drug Abuse and Dependence**, Grinspoon, Lester, MD; Bakalar, James B., Harvard Medical School Mental Health Review No. 1 (1990)

**Drugs of Abuse**, Giannini, A. James, M.D.; Slaby, Andrew E. M.D., Ph.D. Medical Economics Books, Oradell, New Jersey (1989), page 218:

**LSD:**
- Ataxia
- Hyperreflexia
- Tachycardia
  

**Diagnostic and Statistical Manual of Mental Disorders** (Third Ed, Revised), American Psychiatric Association (1987), p. 145.

Maladaptive behavioral changes, e.g., marked anxiety or depression, ideas of reference, fear of losing one's mind, paranoid ideation, impaired judgment, impaired social or occupational functioning.

Perceptual changes occurring in a state of full wakefulness and alertness, e.g., subjective intensification of perceptions, depersonalization, derealization, illusions, hallucinations, Synesthesia

- Pupillary dilation
- Sweating
- Blurring of vision
- Incoordination

**Dissociative Anesthetics (Phencyclidine)**

DRE Symptomatology:

- Nystagmus
- Increased blood pressure
- Perspiring
- Blank stare
  
- Increased pulse
- Increased temperature
- Warm to the touch
- Early onset of nystagmus
"Moon walking" Difficulty in speech
Incomplete responses Repetitive response
Repetitive speech Increased pain threshold
Cyclic behavior Confused, agitated
Hallucinations Possibly violent and combative


Nystagmus Elevated heart rate
Elevated blood pressure Feeling of intoxication
Staggering gait Slurred speech
Numbness of extremities Sweaty
Muscular rigidity Blank stare
Drowsiness Hostile behavior
Repetitive movements

Medical Toxicology-Diagnosis and Treatment of Human Poisoning, Ellenhorn, Matthew J., Barceloux, Donald G. Elsevier Science Pub. Co. 1988, PCP 768-777:

Nystagmus Miosis
Depressed light reflexes Blurred vision
Diminished pain Ataxia
Tremors Muscle weakness
Slurred speech Drowsiness
Increased pulse rate Increased blood pressure
Amnesia Anxiety/agitation
Body image distortion Euphoria
Depersonalization Disordered thought processes
Hallucinations


PCP

Revised: Drug Recognition Expert Course
10/2015 Comparison of DRE Symptomology Sources Page 1 of 18
Increased blood pressure  Blank stare
Disinhibition  Mood swings
Muscle rigidity  Agitation
Delirium excitement  Disorientation
Hallucinations  Analgesia
Speech difficulty  Pain tolerance
Elevated blood pressure

Drug and Alcohol Abuse, A Clinical Guide to Diagnosis and Treatment, (3rd Ed.), Schuckit, M.D., Mark A. Plenum Medical Book Co, New York 1989 p. 178
Sweating  Muscle rigidity
Fever convulsions  Increased blood pressure


PCP:
Nystagmus  Increased blood pressure
Increased pulse rate  Flushing
Mood swings  Hallucinations
Changes in body awareness  Speech difficulties
Violent behavior  Decreased responsiveness

Drug Abuse and Dependence, Grinspoon, Lester, M.D.; Bakalar, James B., Harvard Medical School Mental Health Review No. 1 (1990), page 25:

PCP:
Body image distortions  Increased blood pressure
Nystagmus  Muscle rigidity
Loss of muscle control  Incoherent speech
Memory loss drooling  Blank stare

Drugs of Abuse, Giannini, A. James, M.D.; Slaby, Andrew E. M.D., Ph.D. Medical Economics Books, Oradell, New Jersey(1989) page 296:
PCP:
Nystagmus  Disorientation
Hallucination  Extreme agitation
Loss of motor control  Disassociation from
Automated speech  Environment
Nystagmus at rest


PCP:
Ataxia  Tremors
Muscular hypertonicity  Hyperreflexia
Ptosis  Tachycardia
Horizontal Gaze, Vertical Gaze and Rotary Nystagmus  Elevated blood pressure
Mood swings


Maladaptive behavioral changes, e.g., belligerence, assaultiveness, impulsiveness, unpredictability, psychomotor agitation, impaired judgment, impaired social or occupational functioning.

Vertical or Horizontal Gaze Nystagmus  Increased blood pressure or heart rate
Numbness or diminished responsiveness to pain.  Ataxia
Dysarthria (slurred speech)  Muscle rigidity
Seizures  Hyperacusis

NARCOTICS:
Dre symptomatology:
Constricted pupils  Decreased pulse rate
Decreased blood pressure  Decreased temperature
Ptosis (droopy eyelids)  "on the nod"
Drowsines
Low, raspy speech
Facial itching
Fresh puncture marks

Depressed reflexes
Dry mouth
Euphoria


Medical Toxicology-Diagnosis and Treatment of Human Poisoning, Ellenhorn, Matthew J., Barceloux, Donald G. Elsevier Science Pub. Co. 1988; Heroin, pages 702-703. See also Methadone, Demerol, etc.


Morphine:
Constructed pupils
Drowsiness
Mental clouding
Depressed respiration
Euphoria
Decreased blood pressure
Dysphoria
Sedation
Analgesia

Drug and Alcohol Abuse, A Clinical Guide to Diagnosis and Treatment, (3rd Ed., Schuckit, M.D., Mark A. Plenum Medical Book Co, New York 1989

Decrease pain (p.6)


Narcotics:
Constricted pupils
Analgesia
Euphoria
Reduced heart rate
Depressed appetite
Going "on the nod"

Drug Abuse and Dependence, Grinspoon, Lester, MD; Bakalar, James B., Harvard Medical School Mental Health Review No. 1 (1990), page 14:
Constricted pupils   "nodding off"
Dreamy state        Pain suppression
Euphoria

Drugs of Abuse, Giannini, A. James, M.D.; Slaby, Andrew E. M.D., Ph.D. Medical Economics Books, Oradell, New Jersey (1989) page 293 - 294:

Miosis (constricted pupils) Bradycardia (decreased heart beat)
Hypothermia (decreased temperature) Euphoria/dysphoria
Drowsiness lethargy Confusion
Flaccid muscle tone Depressed respiration
Analgesia


Miosis (constricted pupils) Low blood pressure
Itching Flushing sweating


Maladaptive behavioral changes, e.g., initial euphoria followed by apathy, dysphoria, psychomotor retardation, impaired judgment, impaired social or occupational functioning.

Pupillary constriction Drowsiness
Slurred speech Impairment in attention or memory

INHALANTS: (Toluene)

Dre symptomatology:
Nystagmus Increased pulse rate
Increased blood pressure Residue around nose
Odor on mouth Nausea disorientation
Slurred speech Confusion
<table>
<thead>
<tr>
<th>Decreased inhibitions</th>
<th>Floating sensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drowsiness</td>
<td>Light sensitivity</td>
</tr>
<tr>
<td>Sneezing</td>
<td>Runny nose</td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Lowered inhibitions</th>
<th>Restlessness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incoordination</td>
<td>Confusion</td>
</tr>
<tr>
<td>Nausea</td>
<td>Disorientation</td>
</tr>
<tr>
<td></td>
<td>Impaired judgment</td>
</tr>
</tbody>
</table>

**Drug Abuse and Dependence**, Grinspoon, Lester, MD; Bakalar, James B., Harvard Medical School Mental Health Review No. 1 (1990)

**Drugs of Abuse**, Giannini, A. James, M.D.; Slaby, Andrew E. M.D., Ph.D. Medical Economics Books, Oradell, New Jersey (1989), pages 265, 272, 297: Toluene:

<table>
<thead>
<tr>
<th>Nystagmus</th>
<th>Ataxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tremors cerebellar</td>
<td>Irritability</td>
</tr>
<tr>
<td>Rambling speech</td>
<td>Light headedness</td>
</tr>
<tr>
<td>Tremors</td>
<td>CNS depression that mimics ataxia</td>
</tr>
<tr>
<td>Narcotic analgesics</td>
<td>Blank stare</td>
</tr>
<tr>
<td>Euphoric mood</td>
<td></td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Brief euphoria</th>
<th>Giddy intoxication, similar to alcohol</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS depression (volatile solvents/toluene)</td>
<td>Vertigo</td>
</tr>
<tr>
<td>Dizziness</td>
<td></td>
</tr>
</tbody>
</table>
Maladaptive behavioral changes, e.g., belligerence, assaultiveness, apathy, impaired judgment, impaired social or occupational functioning.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nystagmus</td>
<td>Dizziness</td>
</tr>
<tr>
<td>Incoordination</td>
<td>Slurred speech</td>
</tr>
<tr>
<td>Unsteady gait</td>
<td>Lethargy</td>
</tr>
<tr>
<td>Depressed reflexes</td>
<td>Psychomotor retardation</td>
</tr>
<tr>
<td>Tremor generalized muscle</td>
<td>Blurred vision or diplopia</td>
</tr>
<tr>
<td>Stupor or coma</td>
<td>Weakness</td>
</tr>
<tr>
<td>Euphoria</td>
<td></td>
</tr>
</tbody>
</table>

**CANNABIS**

DRE Symptomatology:

- Dilated pupils
- Marked reddening of conjunctivae
- Odor of Marijuana
- Debris in mouth
- Body tremors
- Eyelid tremors
- Relaxed inhibitions
- Increased appetite
- Paranoia
- Disorientation
- Impaired perception of time and distance


<table>
<thead>
<tr>
<th>Symptom</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euphoria</td>
<td>Short term memory impairment</td>
</tr>
<tr>
<td>Temporal disintegration</td>
<td>Balance and stance impairment</td>
</tr>
<tr>
<td>Information processing impairment</td>
<td>Increased hunger</td>
</tr>
<tr>
<td>Dry mouth</td>
<td>Additive to alcohol</td>
</tr>
</tbody>
</table>

Lower doses affects perception, impairing well beyond when subject subjectively feels effects; alters all information processing; relatively simple motor skills unaffected

<table>
<thead>
<tr>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>High doses:</td>
</tr>
<tr>
<td>Anxiety</td>
</tr>
</tbody>
</table>

Revised: Drug Recognition Expert Course
10/2015 Comparison of DRE Symptomology Sources
Increased systolic blood Pressure
Marked reddening of Conjunctiva Simple motor skills affected
Hallucinations

Medical Toxicology-Diagnosis and Treatment of Human Poisoning, Ellenhorn, Matthew J., Barceloux, Donald G. Elsevier Science Pub. Co. 1988; Cannabis, page 678-681

Reddening of Conjunctiva Motor coordination impairment
Euphoria Relaxation
Temporal distortion (time slows) Impairment of motor tasks and reaction times requires higher dosages
Loss of short term memory Systematic thinking impaired
Stimulated appetite Dry mouth


Reddening of Conjunctiva Increased blood pressure
Dry mouth Altered sensory perception

Drug and Alcohol Abuse, A Clinical Guide to Diagnosis and Treatment, (3rd Ed., Schuckit, M.D., Mark A. Plenum Medical Book Co, New York 1989, page 145:

Cannabis:
Red Conjunctiva Euphoria
Relaxation Dry mouth
Increased heart rate Possibly nystagmus
Time distortion Short term memory
Impairment in ability to do multi-step tasks Tremors
Decrease level of motor coordination


Marijuana:
Red eye Increased heart beat
Time and space distortions  Dryness of mouth and throat
Increased heart rate  Increased pulse rate
Increased appetite

**Drug Abuse and Dependence**, Grinspoon, Lester, MD; Bakalar, James B., Harvard Medical School Mental Health Review No. 1 (1990). page 19:

**Marijuana:**
- Increased appetite
- Bloodshot eyes
- Agitation
- Hallucinations
  
- Faster heartbeat
- Confusion
- Incoordination

**Drugs of Abuse**, Giannini, A. James, M.D.; Slaby, Andrew E. M.D., Ph.D. Medical Economics Books, Oradell, New Jersey (1989), page 296:

**Cannabis:**
- Red Conjunctiva
- Pleasant relaxation
- Slowed time
- Apathy
- Problems with motor coordination
  
- Increased appetite
- Intensification of sensations
- Passivity
- Tachycardia (increased heart rate)


**Cannabis:**
- Red Conjunctiva
- Changes in time sense
- Memory
- Coordination
- Balance and stance
  
- Increased hunger
- Short-term memory loss
- Dry mouth
- Tachycardia (rapid heartbeat)
- Elevated systolic pressure affected

**Diagnostic and Statistical Manual of Mental Disorders** (Third Ed, Revised), American Psychiatric
Maladaptive behavioral changes, e.g., euphoria anxiety, suspiciousness, or paranoid ideation, sensation of slowed time, impaired judgment, social withdrawal.

Red Conjunctiva  Increased appetite
Tachycardia (rapid heart)  Dry mouth

**LACK OF CONVERGENCE:**


Session 23
Curriculum Vitae Preparation and Maintenance
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Upon successfully completing this session the participant will be able to:

- Describe and discuss the purpose of the DRE Curriculum Vitae.
- Identify the elements of a DRE Curriculum Vitae.
- Prepare a basic Curriculum Vitae summarizing their relevant training, education, experience, and accomplishments to date.
- Update and extend the Curriculum Vitae, as relevant achievements continue to expand.

CONTENT SEGMENTS ............................................................ LEARNING ACTIVITIES
A. Purpose of the Curriculum Vitae .............................................. Instructor-Led Presentations
B. Preparation for Court Qualification ...........................................Group Work Session
C. Curriculum Vitae Content ...................................................... Reading Assignments
D. Guidelines for Curriculum Vitae Preparation and Maintenance
A. Purpose of the Curriculum Vitae

The basic purpose of the Curriculum Vitae is to record education, training, and experience in a single document for use in establishing qualifications when testifying in court.

Generally a witness can testify only to personal knowledge.
Basic rule is that a person skilled in some art, trade, science, or profession, having a knowledge of matters not within the knowledge of persons of average education, learning and experience, may assist the jury in arriving at a verdict by expressing an opinion on a state of facts shown by the evidence and based upon his or her special knowledge.

Source: People vs. Willis, 70 Cal APP. 465

A witness is not qualified as an expert witness unless it is shown he or she is familiar with the subject upon which he or she is asked to give an opinion.

Source: People vs. McLean, 56 Cal 2d 660

Only the court can determine whether a witness is qualified to testify as an expert.

Where a witness is qualified to give expert testimony, any question as to degree of knowledge goes to weight rather than admissibility.

Source: People vs. Perry, 44 Cal 2d 861
Witnesses’ qualification is achieved through Voir Dire Examination.

Voir Dire – literally, French for “to see, to say;” loosely translated as “to seek the truth.”
B. Preparation for Court Qualification

Being qualified as an expert may be as simple as stating your occupation, or take several hours of exhausting questioning by both the prosecutor and the defense attorney.

Although knowledge only greater than what the public has is required to qualify you as an expert, your testimony will carry much more “weight” if you have good credentials.

Accurate, up-to-date information is essential for an officer who is called upon to give his or her qualification as an expert in any field.

Drug Recognition Experts will base their expertise on the following areas:

- Formal education and training
- Relevant experience
- Outside readings and studies
### C. Curriculum Vitae Content

**Formal Education**

- High School(s) attended
  
  List dates – highlight classes which provided knowledge in the area of drugs.
  
- Colleges and Universities attended
  
  List dates, instructor, subject(s) covered, credits, etc.
  
- University level courses
  
  List dates, instructor, subject(s) covered, credits, etc.
  
- Specialized College
  
  List dates, length, major topics covered, etc. Highlight classes which provided knowledge or skills in the area of drugs.

**Formal Training**

- Police Academy (recruit training).
  
- Specialized police training or in-service training.
  
  List dates, length, instructor(s), subject(s) covered, etc. Highlight training which provided knowledge or skills in the area of drugs.
  
- Other specialized training.
  
- Military training.
  
- Lectures and seminars.
  
  List dates, length, instructor(s), subject(s) covered, etc. Highlight training which provided knowledge or skills in the area of drugs.
Experience

- Job experience – years.

List dates, division, duties, etc., include loans to specialized units.

- Assignments.
- List agencies, dates, assignments, etc.
- Prior law enforcement experience.

List employer, dates, duties, assignments, etc. which provided experience in the area of drugs.

- Other job related experience.

Drug enforcement/ evaluation experiences:

- Total vehicle stops
- Total DWI investigations
- Total DWI arrests
- Total drug evaluations
- Total filings
- Total convictions
Prior Testimony

- Municipal court
- Superior court
- Number of times qualified as an expert in drug cases
- Number of times qualified as an expert in other cases

For bulleted items above: list dates, courts, judges, charges, areas qualified, etc.

Outside Reading and Studies

- Drug related texts read.
- List title(s), author(s), subject(s), etc.
- Departmental training bulletins.
- Journals.
- Research papers.
- Drug related videos viewed.
Training or Research Conducted (if applicable)

List classes, briefings, training officer assignments, etc. where you served as an instructor or coach, etc. or conducted or participated in research, e.g. Alcohol Workshop.

Published Works (if applicable)

List all relevant writings that you authored or co-authored, including departmental briefing papers, training manuals/bulletins, magazine articles, books, etc.
D. Guidelines for Curriculum Vitae Preparation and Maintenance

- List information in chronological order.
- Review and update Curriculum Vitae frequently and record date of review.
The Curriculum Vitae of:

Sgt. David C. Regan
**Introduction**

Sergeant David Carroll Regan is a supervisor in the Traffic Division, Shelton Police Department. He currently commands the special Impaired Driving Enforcement Activities Squad (IDEAS), a unit he was instrumental in forming. Sgt. Regan is a 15 year veteran of law enforcement. Prior to joining the Shelton Police Department ten years ago, he served for five years as a deputy with the Fairfield County Sheriff's Department.

Sergeant Regan has been assigned to the Traffic Division since his promotion to sergeant on 11/18/YY. His duties have included coordination of speed and DWI enforcement activities, the Joint Shelton-Derby Task Force for Sobriety Checkpoints, the Officer Friendly Program, the Motorcycle Safety Education Project, and general supervision of Traffic Division officers. He also serves as the Department's principal instructor for radar speed measurement, Standardized Field Sobriety Testing and Drug Recognition Expert training.

Sergeant Regan holds a Bachelor's Degree in the Administration of Justice from Fairfield University, and currently is a candidate for a Master's Degree in Police Science and Administration at the University of Stratford. He also holds an Instructor Certificate from the State Law Enforcement Training Board.

Sergeant Regan has served on two committees of the Governor's Task Force to Prevent Drunk Driving: The Standardized Field Sobriety Tests Committee and The Paperwork Reduction Committee. The one page Standard Notetaking Guide for Field Sobriety Testing that is employed by all departments statewide was designed by him.

**Law Enforcement Experience**

<table>
<thead>
<tr>
<th>Date Range</th>
<th>Position</th>
</tr>
</thead>
<tbody>
<tr>
<td>11/18/YY to Present</td>
<td>Sergeant, Traffic Division</td>
</tr>
<tr>
<td></td>
<td>Shelton Police Department Supervisor, IDEAS Unit</td>
</tr>
<tr>
<td></td>
<td>Drug Recognition Expert Program Coordinator</td>
</tr>
<tr>
<td>7/8/ZZ to 11/17/YY</td>
<td>Patrol Officer First Class</td>
</tr>
<tr>
<td></td>
<td>Training and Operations</td>
</tr>
<tr>
<td></td>
<td>Shelton Police Department</td>
</tr>
<tr>
<td></td>
<td>Unit Supervisor, Traffic Law Enforcement Training Branch</td>
</tr>
<tr>
<td>9/11/XX to 7/7/ZZ</td>
<td>Patrol Officer</td>
</tr>
<tr>
<td></td>
<td>Third Precinct, Motorcycle</td>
</tr>
<tr>
<td></td>
<td>Shelton Police Department</td>
</tr>
</tbody>
</table>
Sgt. David C. Regan

Law Enforcement Experience (continued)

11/5/MM to 9/10/XX  Patrol Officer
First Precinct
Shelton Police Department

10/10/NN to 11/4/MM  Deputy
Traffic Patrol
Fairfield County Sheriff's Department

Special Police Training

10/XX  NHTSA/IACP

**DRE Instructor Training**
(Certified as a DRE Instructor on 11/12/XX)

8/XX  Drug Enforcement Administration

**Drug Interdiction Seminar**

11/YY  NHTSA/IACP

**Drug Evaluation and Classification Training: DRE School**
(Certified as a DRE on 1/28/XX)

10/YY  NHTSA/IACP

**Drug Evaluation and Classification Training: PRE School**

3/YY  Southeastern University Institute of Police Technology

**Special Conference: Managing DWI Squads**

4/ZZ  International Association of Chiefs of Police

**Instructor Training in Horizontal Gaze Nystagmus and Divided Attention Field Sobriety Tests**

10/MM  University of Stanford, Northern Police Institute

**Standardized Field Sobriety Testing**

6/NN  Acme Scientific Instruments, Inc.
(Certified to perform inspection and repair of the Intoxotector J2Z breath testing instrument on 6/22/NN)
Sgt. David C. Regan

Court Qualification Record

8/VV Qualified as Drug Recognition Expert in a case involving Phencyclidine impairment. (Judge Sally Grey, 8th District)

11/WW Qualified as Drug Recognition Expert in a case involving a combination of CNS Stimulant and Narcotic Analgesic. (Judge Lewis Buchanan, Superior Court)

3/WW Qualified as Drug Recognition Expert in a case involving Cannabis impairment. (Judge Sally Grey, 8th District)

9/UU Qualified as Drug Recognition Expert in a case involving Narcotic Analgesic impairment. (Judge Jerome Byrnes, 8th District)

Specialized Readings

<table>
<thead>
<tr>
<th>Title</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug and Alcohol Abuse</td>
<td>Marc A. Schuckit, M.D.</td>
</tr>
<tr>
<td>A Primer of Drug Action</td>
<td>Jerome Jaffee, Robert Petersen and Ray Hodgson</td>
</tr>
<tr>
<td>The Practitioner's Guide to</td>
<td>Ellen L. Bassuk, M.D. and</td>
</tr>
<tr>
<td>Psychoactive Drugs</td>
<td>Stephen C. Schoonover, M.D.</td>
</tr>
<tr>
<td>Drug Abuse: A Manual for Law</td>
<td>Smith, Kline and French (pub.)</td>
</tr>
<tr>
<td>Enforcement Officers</td>
<td></td>
</tr>
<tr>
<td>Licit and Illicit Drugs</td>
<td>Edward M. Brecher</td>
</tr>
<tr>
<td>Chocolate to Morphine</td>
<td>Andrew Weil, M.D. and Winifred Rosen</td>
</tr>
<tr>
<td>Cocaine Addiction</td>
<td>U.S. Department of Health and Human Services</td>
</tr>
<tr>
<td>Marijuana Alert</td>
<td>Peggy Mann</td>
</tr>
</tbody>
</table>
SAMPLE Curriculum Vitae NUMBER TWO

TRUMBULL POLICE DEPARTMENT

The Curriculum Vitae of:

OFFICER ANN MARIE REED
Drug Recognition Expert

Latest Update: 4/25/YY
Officer Ann M. Reed

Introduction

Officer Ann Marie Reed is an eight year veteran with the Trumbull Police Department. She is currently assigned to the Special Operations Branch of the Administrative Division, where she serves as a Narcotics Enforcement Officer. Previously, she has served in the same Branch as a Vice Enforcement Officer, and as a patrol officer in the Department's first and second precincts.

Officer Reed is a graduate of Monroe College, with the Bachelor’s Degree in Police Science and Administration. She is currently a candidate for the JD Degree at the Law School of the University of Bridgeport.

Law Enforcement Experience

5/12/VV to Present
Narcotics Enforcement Officer and Drug Recognition Expert
Special Operations Branch
Trumbull Police Department

3/26/WW to 5/11/VV
Vice Enforcement Officer Special Operations Branch Trumbull Police Department

9/23/XX to 3/25/WW
Patrol Officer
First Precinct
Trumbull Police Department

8/28/NN to 9/22/XX
Patrol Officer
Second Precinct
Trumbull Police Department

5/15/NN to 8/25/NN
Trainee
Fairfield County Regional Police Academy
(Graduated 8/25/NN)

Special Police Training

2/YY
University of Norwalk, Police Science Institute

Seminar: Packaging and Transport of Illicit Drugs

10/VV
University of Norwalk, Police Science Institute

Seminar: Suppression of Drug-related Crime

3/VV
NHTSA/IACP

Drug Evaluation and Classification Training: DRE School
(Certified as a DRE on 5/22/VV)
Officer Ann M. Reed

Special Police Training (Continued)

2/VV Fairfield County Regional Police Academy

Drug Evaluation and Classification Training: PRE-School

10/WW Fairfield County Regional Police Academy

Standardized Field Sobriety Testing

Publications Authored


Reed, Ann M., Procedures for Requesting Drug Recognition Expert Services; Training Bulletin for the Trumbull Police Department. 6/VV.

Reed, Ann M., Recognizing the Heroin Addict; Training Bulletin for the Trumbull Police Department. 1/VV.

Court Qualification Record

11/WW Qualified as an expert witness for identification of Heroin impairment. (Judge Michael Adkins, 7th District)

3/WW Qualified as a Drug Recognition Expert in a case involving a combination of CNS Stimulant and Narcotic Analgesic. (Judge Roberta Mayer, 7th District)

9/ZZ Qualified as an expert witness for identification of "track" marks. (Judge Charles Peltier, 7th District)

Specialized Readings

<table>
<thead>
<tr>
<th>Title</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Signs and Symptoms Handbook</td>
<td>Barbara McVan, M.D.</td>
</tr>
<tr>
<td>Drugs From A to Z</td>
<td>Richard R. Lingeman</td>
</tr>
<tr>
<td>Guide to Psychoactive Drugs</td>
<td>Richard Seymour and David E. Smith, M.D.</td>
</tr>
<tr>
<td>Addictions: Issues and Answers</td>
<td>Robert M. Julien, M.D.</td>
</tr>
<tr>
<td>Report on Synthetic China</td>
<td>Det. James Miller, LAPD</td>
</tr>
<tr>
<td>White: Fentanyl</td>
<td></td>
</tr>
</tbody>
</table>

Revised: 10/2015 Drug Recognition Expert Course Example Curriculum Vitae
Learning Objectives

• Explain the prevalence of polydrug use among drug impaired subjects and identify common combinations of drugs abused by those subjects
• Describe the possible effects that combinations of drugs can produce on the major indicators of drug impairment

Learning Objectives

• Define the terms “Null,” “Overlapping,” “Additive” and “Antagonistic” as they relate to polydrug effects
• Identify specific effects that are most likely to be observed in persons under the influence of particular drug combinations

Upon successfully completing this session the participant will be able to:

• Explain the prevalence of polydrug use among drug impaired subjects and identify common combinations of drugs abused by those subjects.
• Describe the possible effects that combinations of drugs can produce on the major indicators of drug impairment.
• Define the terms “Null,” “Overlapping,” “Additive” and “Antagonistic” as they relate to polydrug effects.
• Identify the specific effects that are most likely to be observed in persons under the influence of particular drug combinations.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES
A. The Prevalence of Polydrug Use ................................................... Instructor-Led Presentations
B. Possible Effects of Drug Combinations ................................................... Interactive Discussions
C. Identifying Expected Indicators of Specific Combinations ......................... Workbook Exercise
........................................................................................................................... Video Presentations
A. The Prevalence of Polydrug Use

Polydrug use means ingesting drugs from two or more drug categories.
Prevalence of Polydrug Use

It is actually more common for a DRE to encounter polydrug users than single drug users.

- In the Los Angeles Field Study (1985), 72% of the suspects had two or more drugs in them.
- If we discount alcohol, nearly half (45%) of the Field Study suspects had two or more other drugs in them.
- The National DRE database indicates that approximately 35% of all DRE reported cases revealed two or more drug categories detected.

Source: NHTSA/IACP DRE Database (2014)
Common Combinations

- Cocaine and Cannabis.
- Cocaine and Heroin.
- PCP and Cannabis.
- Alcohol and practically anything else.

Many of the subjects you examine will be exhibiting the effects of two or more drugs acting together.
B. Possible Effects of Drug Combinations

Combos

Let us examine the possible ways in which two or more drug categories might interact.

Some common combinations of drug categories and their street names include:

- Cocaine and Heroin - “Speedball”
- PCP and Heroin - “Fireball”
- Crack and PCP - “Space base”
- Crack and Marijuana - “Primo”
- Crack and Methamphetamine - “Croak”

There are four effects of drug combinations on major indicators of impairment:

- Null Effect
- Overlapping Effect
- Additive Effect
- Antagonistic Effect
Null Effect

- If neither drug affects a particular indicator of impairment, their combination also will not affect that indicator
- No action plus no action equals no action

Example #1: HGN
- If neither drug affects HGN...
  Example: Narcotic Analgesic and Cannabis
  - (Neither category affects HGN)
  - ...the combination should also not affect HGN, so HGN will not be present in this combination

Four Effects

- Null Effect

The first effect is called the “Null Effect.”
- This would be no action plus no action equals no action.

Example #1: HGN
- Neither drug affects HGN.

The combination would not result in HGN being present.

Example #1 is called the Null Effect.
Example #2: Reactions to Light

Another example of the Null Effect:

Reaction to Light: neither drug affects reaction to light. Example: a Dissociative Anesthetic and Cannabis.

Example #3: Body Temperature

Another example of the Null Effect:

Body Temperature: neither a CNS Depressant nor Cannabis usually affects body temperature; the combination of the two leaves body temperature in the DRE average range.
Overlapping Effect
The second effect is called the “Overlapping Effect.”

Example #1: Pupil Size
Example #1: one drug affects pupil size, but the other does not.
Example: CNS Stimulants and Dissociative Anesthetics. CNS Stimulants dilate pupils, Dissociative Anesthetics do not affect pupil size.
Therefore, pupils should be dilated.
Example #2: HGN

HGN: a CNS Depressant will cause HGN, but Cannabis will not cause HGN; a person under the combined influence of a CNS Depressant and Cannabis will usually have HGN.

Example #3: Lack of Convergence

Another example of the “Overlapping Effect”:

Lack of Convergence. Dissociative Anesthetics cause Lack of Convergence, Hallucinogens do not. Under the influence, lack of convergence should be present.
Additive Effect

The third effect is called the Additive Effect.

• If two drugs independently affect some indicator in the same way, their use in combination will also affect the indicator and the effect may be reinforced

  • Action plus the same action produces reinforced action

Example #1: Pulse Rate

Pulse Rate. Cannabis and Inhalants both elevate pulse rate. Therefore, pulse rate should be elevated, or up.
Example #2: Pupil Size

Pupil Size. CNS Stimulants and Hallucinogens both dilate the pupils; therefore, pupils should be dilated.

Example #3: Blood Pressure

Blood Pressure. CNS Depressants and Narcotic Analgesics both depress blood pressure. Therefore, the blood pressure should be depressed or down.
Antagonistic Effect

The fourth effect is called the Antagonistic Effect.

When two drugs produce an “Antagonistic Effect,” they tend to try to override or compete with the effect of the other drug(s) until the drug with the longest duration of effects prevails. Normally, whichever drug is more psychoactive at the time determines what we’ll see.

There is not an Antagonistic Effect for:

- HGN,
- VGN,
- Lack of Convergence and
- Reaction to Light.
Example #1: Pulse Rate

Pulse Rate. CNS Stimulants elevate pulse rate, CNS Depressants depress pulse rate; therefore, pulse rate will be up, down or within the DRE average ranges.

Example #2: Pupil Size

Pupil Size. CNS Stimulants dilate pupils, Narcotic Analgesics constrict pupils. Pupil size will be dilated, constricted or within the DRE average ranges.

Example #3: Body Temperature

Body Temperature. Hallucinogens elevate body temperature, Narcotic Analgesics depress body temperature. Body temperature will be up, down or within the DRE average ranges.

With an “Antagonistic Effect,” we just can’t predict what we will see.

Summary

When drugs from two or more drug categories are taken together, they tend to produce a combination of Null Effects, Overlapping Effects, Additive Effects and Antagonistic Effects.
**HGN**

A specific example: consider a person who is under the influence of a combination of Cannabis and a CNS Stimulant.

Neither Cannabis nor a CNS Stimulant causes HGN.

This is a case of no action plus no action equals no action.

We will not see HGN with this combination.

---

**Vertical Gaze Nystagmus**

Neither Cannabis nor a CNS Stimulant causes VGN.

This is another Null Effect.

We won’t see VGN.

---

**Lack of Convergence**

Cannabis causes Lack of Convergence; a CNS Stimulant does not.

This is a case of action plus no action equals action.

We will see Lack of Convergence with this combination.
Pupil Size
CNS Stimulants dilate pupils; Cannabis either dilates pupils or has no effect on them.
This may be a case of action plus no action equals action.
Or it may be a case of action plus same action reinforces action.
In either case, we should see dilated pupils with this combination.

Reaction to Light
CNS Stimulants slow the pupils’ Reaction to Light; Cannabis usually doesn’t affect the pupils’ reaction.
Here we have another Overlapping Effect.
We should observe a slowed reaction of the pupils.

Pulse Rate
Both Cannabis and CNS Stimulants usually elevate pulse rate.
This is an Additive Effect.
We should see a pulse rate that is up or elevated.

Blood Pressure
Cannabis usually causes blood pressure to be up or elevated; so does a CNS Stimulant.
This is another Additive Effect.
We should see a blood pressure that is up or elevated.
Body Temperature
Cannabis usually does not affect body temperature. But CNS Stimulants usually elevate temperature.
This is another case of action plus no action equals action.
We can expect to see an elevated temperature with this combination.

Muscle Tone
Cannabis usually does not affect muscle tone. CNS Stimulants cause muscle tone to be rigid.
This is another case of action plus no action equals action.
We can expect to see rigid muscle tone with this combination.
Dissociative Anesthetics and Narcotic Analgesics

Another specific example: consider a person under the influence of a combination of a Dissociative Anesthetic and a Narcotic Analgesic.

**HGN**

A Dissociative Anesthetic causes HGN, Narcotic Analgesics do not.

This is an Overlapping Effect.

We can expect to see HGN with this subject.

**Vertical Gaze Nystagmus**

A Dissociative Anesthetic should cause Vertical Gaze Nystagmus, especially at high doses. A Narcotic Analgesic will not cause Vertical Gaze Nystagmus.

This is another Overlapping Effect.

We should see Vertical Gaze Nystagmus in this subject.
Lack of Convergence

A Dissociative Anesthetic causes Lack of Convergence; Narcotic Analgesics do not.
Another Overlapping Effect.
We can expect to see Lack of Convergence.

Pupil Size

A Dissociative Anesthetic doesn’t affect pupil size, but a Narcotic Analgesic constricts pupils.
This is another Overlapping Effect.
We can expect to see constricted pupils with this subject.

Reaction to Light

A Dissociative Anesthetic doesn’t affect pupil’s Reaction to Light; but a Narcotic Analgesic usually produces a “little or none visible” reaction.
This, too, is an Overlapping Effect.
We can expect a “little or none visible” reaction in this subject’s pupils.

Pulse Rate

A Dissociative Anesthetic usually causes pulse rate to be elevated; a Narcotic Analgesic usually produces a depressed or lower pulse rate.
This is our first Antagonistic Effect.
We cannot predict what this subject’s pulse rate will be.
The pulse rate could be elevated, or depressed, or within the DRE average ranges.
This subject’s pulse rate will depend on many factors, including:

• How much of each drug was taken.
• How and when each drug was taken.
• How tolerant the subject is of each drug.
**Blood Pressure**
A Dissociative Anesthetic usually elevates blood pressure; a Narcotic Analgesic usually lowers blood pressure.
This is another Antagonistic Effect.
We can’t predict what the blood pressure will be.
It could be above DRE average ranges, below DRE average ranges, or within the DRE average ranges.

**Temperature**
A Dissociative Anesthetic usually elevates temperature; a Narcotic Analgesic usually lowers it.
This, too, is an Antagonistic Effect.
The temperature could be elevated (up), or depressed (down) or within the DRE average range.

**Muscle Tone**
A Dissociative Anesthetic usually causes rigid muscle tone. A Narcotic Analgesic usually causes flaccid muscle tone.
This could be an Antagonistic Effect.
Muscle tone could be normal, rigid, or flaccid.
**Cannabis, CNS Stimulants and Hallucinogens**

Another specific example: consider a person under the influence of Cannabis, a CNS Stimulant and a Hallucinogen.

**HGN**

None of the three categories causes HGN. This is an example of the Null Effect.

**VGN**

None of the three drug categories cause Vertical Gaze Nystagmus, another example of the Null Effect.

**LOC**

Cannabis causes a Lack of Convergence while CNS Stimulants and Hallucinogens do not. This is an example of an Overlapping Effect and Lack of Convergence should be present.
Pupil Size
Cannabis usually dilates pupils. CNS Stimulants and Hallucinogens also dilate the pupils.
This is an example of an Additive or Overlapping Effect.
The pupils should be dilated.

Reaction to Light
Cannabis does not effect the Reaction to Light. CNS Stimulants will slow down the reaction.
Most Hallucinogens, with some exceptions, will cause a normal Reaction to Light.
This is an example of either an Overlapping or Additive Effect.
We could probably see a slow Reaction to Light.

Pulse Rate
Cannabis will normally elevate the pulse rate as will CNS Stimulants and Hallucinogens.
This is an example of an Additive Effect.
The result would be an elevated pulse rate.

Blood Pressure
All three drug categories will elevate blood pressure.

This is an example of an Additive Effect.
Blood pressure should be elevated with this combination.

Body Temperature
Cannabis usually causes a body temperature in the average range. CNS Stimulants and Hallucinogens elevate body temperature.
This would be an example of an Additive or Overlapping Effect.
The body temperature should be elevated with this combination.

Muscle Tone
Cannabis causes a normal muscle tone, while CNS Stimulants and Hallucinogens will cause rigid muscle tone.
This would be an example of an Additive or an Overlapping Effect.
The muscle tone should be rigid with this combination.
Identifying Expected Indicators of Specific Combinations

The *Drug Symptomatology Matrix* outlines the expected results of the drug influence evaluation for each drug category.

C. Identifying Expected Indicators of Specific Combinations

*Drug Symptomatology Matrix*

The Matrix outlines the expected results of the drug influence evaluation for each drug category.
**Worksheet Exercises**

Worksheet #1: Dissociative Anesthetic and a Hallucinogen.
Worksheet #2: Cannabis and CNS Depressant.
Worksheet #3: CNS Depressant and CNS Stimulant.

**Discussion of Worksheets**
### INDICATORS CONSISTENT WITH DRUG CATEGORIES

<table>
<thead>
<tr>
<th></th>
<th>CNS DEPRESSANTS</th>
<th>CNS STIMULANTS</th>
<th>HALLUCINOGENS</th>
<th>DISSOCIATIVE ANESTHETICS</th>
<th>NARCOTIC ANALGESICS</th>
<th>INHALANTS</th>
<th>CANNABIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HGN</strong></td>
<td>PRESENT</td>
<td>NONE</td>
<td>NONE</td>
<td>PRESENT</td>
<td>NONE</td>
<td>PRESENT</td>
<td>NONE</td>
</tr>
<tr>
<td><strong>VGN</strong></td>
<td>PRESENT (HIGH DOSE)</td>
<td>NONE</td>
<td>NONE</td>
<td>PRESENT</td>
<td>NONE</td>
<td>PRESENT</td>
<td>NONE</td>
</tr>
<tr>
<td><strong>LACK OF CONVERGENCE</strong></td>
<td>PRESENT</td>
<td>NONE</td>
<td>NONE</td>
<td>PRESENT</td>
<td>NONE</td>
<td>PRESENT</td>
<td>PRESENT</td>
</tr>
<tr>
<td><strong>PUPIL SIZE</strong></td>
<td>NORMAL (1)</td>
<td>DILATED</td>
<td>DILATED</td>
<td>NORMAL</td>
<td>CONSTRICTED</td>
<td>NORMAL (4)</td>
<td>DILATED (6)</td>
</tr>
<tr>
<td><strong>REACTION TO LIGHT</strong></td>
<td>SLOW</td>
<td>SLOW</td>
<td>NORMAL (3)</td>
<td>NORMAL</td>
<td>LITTLE OR NONE VISIBLE</td>
<td>SLOW</td>
<td>NORMAL</td>
</tr>
<tr>
<td><strong>PULSE RATE</strong></td>
<td>DOWN (2)</td>
<td>UP</td>
<td>UP</td>
<td>UP</td>
<td>DOWN</td>
<td>UP</td>
<td>UP</td>
</tr>
<tr>
<td><strong>BLOOD PRESSURE</strong></td>
<td>DOWN</td>
<td>UP</td>
<td>UP</td>
<td>UP</td>
<td>DOWN</td>
<td>UP/DOWN (5)</td>
<td>UP</td>
</tr>
<tr>
<td><strong>BODY TEMPERATURE</strong></td>
<td>NORMAL</td>
<td>UP</td>
<td>UP</td>
<td>UP</td>
<td>DOWN</td>
<td>UP / DOWN / NORMAL</td>
<td>NORMAL</td>
</tr>
<tr>
<td><strong>MUSCLE TONE</strong></td>
<td>FLACCID</td>
<td>RIGID</td>
<td>RIGID</td>
<td>RIGID</td>
<td>FLACCID</td>
<td>NORMAL OR FLACCID</td>
<td>NORMAL</td>
</tr>
</tbody>
</table>

**FOOTNOTE:** These indicators are those most consistent with the category, keep in mind that there may be variations due to individual reaction, dose taken and drug interactions.

1. Soma, Quaaludes and possibly some anti-depressants usually dilate pupils.
2. Quaaludes, ETOH and possibly some anti-depressants may elevate.
3. Certain psychedelic amphetamines may cause slowing.
4. Normal, but may be dilated
5. Down with anesthetic gases, up with volatile solvents and aerosols.
6. Pupils possibly normal.
<table>
<thead>
<tr>
<th>MAJOR INDICATORS</th>
<th>CNS DEPRESSANTS</th>
<th>CNS STIMULANTS</th>
<th>HALLUCINOGENS</th>
<th>DISSOCIATIVE ANESTHETICS</th>
<th>NARCOTIC ANALGESICS</th>
<th>INHALANTS</th>
<th>CANNABIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>NOTE</em></td>
<td>Disoriented</td>
<td>Droopy eyes</td>
<td>Drowsiness</td>
<td>Drunk-like behavior</td>
<td>Gait ataxia</td>
<td>Slow, sluggish reactions</td>
<td>Thick, slurred speech</td>
</tr>
<tr>
<td><strong>GENERAL INDICATORS</strong></td>
<td>Anxiety</td>
<td>Body tremors</td>
<td>Dry mouth</td>
<td>Euphoria</td>
<td>Exaggerated reflexes</td>
<td>Excited</td>
<td>Eyelid tremors</td>
</tr>
<tr>
<td><strong>NOTE: With Methaqualone, (Quaaludes) pulse will be elevated and body tremors will be evident. Alcohol and Methaqualone elevate pulse. Soma and Methaqualone dilate pupils.</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>DURATION OF EFFECTS</strong></th>
<th>Ultra-short: A few minutes</th>
<th>Short: Up to 5 hours</th>
<th>Intermediate: 6-8 hours</th>
<th>Long: 8-14 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cocaine: 5-90 minutes</td>
<td>Amphetamines: 4-8 hours</td>
<td>Meth: 12 plus hours</td>
<td>Duration varies widely from one hallucinogen to another.</td>
<td>LSD: 10-12 hours</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Clammy skin</td>
<td>Coma</td>
<td>Dilated Pupils</td>
<td>Rapid, weak pulse</td>
<td>Shallow breathing</td>
<td>Agitation</td>
<td>Hallucinations</td>
<td>Increased body temperature</td>
<td>Long intense “trip”</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>OVERDOSE SIGNS</strong></th>
<th>Bloodshot eyes</th>
<th>Confusion</th>
<th>Disoriented</th>
<th>Flushed face</th>
<th>Intense headaches</th>
<th>Lack of muscle control</th>
<th>Non-communicative Odor of substance</th>
<th>Possible nausea</th>
<th>Residue of substance</th>
<th>Slow, thick, slurred speech</th>
<th>Watery eyes</th>
<th>NOTE: Anesthetic gases cause below normal blood pressure; volatile solvents and aerosols cause above normal blood pressure.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bloodshot eyes eyes Debris in mouth</td>
<td>Disoriented</td>
<td>Drowsiness</td>
<td>Eyelid tremors</td>
<td>Impaired memory</td>
<td>Increased appetite</td>
<td>Lack of Concentration Odor of Marijuana</td>
<td>Possible paranoia</td>
<td>Relaxed inhibitions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| **REVISED: 10/2015** | Drug Recognition Expert Course | Indicators Consistent with Drug Categories | Page 2 of 2 |
Specific Examples of Drug Combinations: An Exercise for the Student

On the final five pages of this session, you will find examples of specific drug combinations. The expected results for the first two of these combinations (Cannabis and Stimulants, and Dissociative Anesthetic and Narcotic Analgesic) have been worked out for you. Study those examples, then complete the work sheets for the three remaining combinations.
**CANNABIS AND CNS STIMULANT IN COMBINATION**

<table>
<thead>
<tr>
<th>Impairment Indicator</th>
<th>Effect Due To Cannabis</th>
<th>Effect Due To CNS Stimulant</th>
<th>Type Of Combined Effect</th>
<th>What Will We See</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertical Gaze Nystagmus</td>
<td>None</td>
<td>None</td>
<td>Null</td>
<td>None</td>
</tr>
<tr>
<td>Lack Of Conv.</td>
<td>Present</td>
<td>None</td>
<td>Overlapping</td>
<td>Present</td>
</tr>
<tr>
<td>Pupil Size</td>
<td>Dilated Or Normal</td>
<td>Dilated</td>
<td>Overlapping Or Additive</td>
<td>Dilated</td>
</tr>
<tr>
<td>Reaction To Light</td>
<td>Normal</td>
<td>Slow</td>
<td>Overlapping</td>
<td>Slow</td>
</tr>
<tr>
<td>Pulse Rate</td>
<td>Up</td>
<td>Up</td>
<td>Additive</td>
<td>Up</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Up</td>
<td>Up</td>
<td>Additive</td>
<td>Up</td>
</tr>
<tr>
<td>Body Temp</td>
<td>Normal</td>
<td>Up</td>
<td>Overlapping</td>
<td>Up</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Normal</td>
<td>Rigid</td>
<td>Overlapping</td>
<td>Rigid</td>
</tr>
</tbody>
</table>
## Dissociative Anesthetic and Narcotic Analgesic in Combination

<table>
<thead>
<tr>
<th>Impairment Indicator</th>
<th>Effect Due To Phencyclidine</th>
<th>Effect Due To Heroin</th>
<th>Type Of Combined Effect</th>
<th>What Will We See</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horizontal Gaze Nystagmus</td>
<td>Present</td>
<td>None</td>
<td>Overlapping</td>
<td>Present</td>
</tr>
<tr>
<td>Vertical Gaze Nystagmus</td>
<td>Present</td>
<td>None</td>
<td>Overlapping</td>
<td>Present</td>
</tr>
<tr>
<td>Lack Of Conv.</td>
<td>Present</td>
<td>None</td>
<td>Overlapping</td>
<td>Present</td>
</tr>
<tr>
<td>Pupil Size</td>
<td>Normal</td>
<td>Constricted</td>
<td>Overlapping</td>
<td>Constricted</td>
</tr>
<tr>
<td>Reaction To Light</td>
<td>Normal</td>
<td>Little Or None Visible</td>
<td>Overlapping</td>
<td>Little Or None Visible</td>
</tr>
<tr>
<td>Pulse Rate</td>
<td>Up</td>
<td>Down</td>
<td>Antagonistic</td>
<td>Down/Normal/Up</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Up</td>
<td>Down</td>
<td>Antagonistic</td>
<td>Down/Normal/Up</td>
</tr>
<tr>
<td>Body Temp</td>
<td>Up</td>
<td>Down</td>
<td>Antagonistic</td>
<td>Down/Normal/Up</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Rigid</td>
<td>Flaccid</td>
<td>Antagonistic</td>
<td>Rigid/Flaccid/Normal</td>
</tr>
<tr>
<td>Impairment Indicator</td>
<td>Effect Due To Dissociative Anesthetics</td>
<td>Effect Due To Hallucinogen (Hall)</td>
<td>Type Of Combined Effect*</td>
<td>What Will We See</td>
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<td>Horizontal Gaze Nystagmus</td>
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<td>Vertical Gaze Nystagmus</td>
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<td>Lack Of Conv.</td>
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<td>Pupil Size</td>
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<td>Reaction To Light</td>
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<tr>
<td>Muscle Tone</td>
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</tbody>
</table>

*Null; Overlapping; Additive; or, Antagonistic
## WORKSHEET #2
### CANNABIS AND CNS DEPRESSANT

<table>
<thead>
<tr>
<th>Impairment Indicator</th>
<th>Effect Due To Cannabis</th>
<th>Effect Due To Depressant</th>
<th>Type Of Combined Effect*</th>
<th>What Will We See</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horizontal Gaze Nystagmus</td>
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<td>Vertical Gaze Nystagmus</td>
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<td>Lack Of Conv.</td>
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<td>Pupil Size</td>
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<tr>
<td>Reaction To Light</td>
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<table>
<thead>
<tr>
<th>Impairment Indicator</th>
<th>Effect Due To CNS Stimulant</th>
<th>Effect Due To CNS Depressant</th>
<th>Type Of Combined Effect*</th>
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</tr>
</thead>
<tbody>
<tr>
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Participant Manual

Drug Recognition Expert Course

Session 25
Practice:
Test Interpretation
Learning Objectives

- Analyze the results of completed drug influence evaluations and identify the category or categories of drugs affecting the individual examined.
- Describe the basis for the drug category identification.

Upon successfully completing this session the student will be able to:

- Analyze the results of completed drug influence evaluations and identify the category or categories of drugs affecting the individual examined.
- Describe the basis for the drug category identification.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES
A. Interpretation Demonstrations ................................................ Instructor-Led Demonstrations
B. Interpretation Practice............................................................................... Small Group Practice
............................................................................................................ Participant Led Presentations
A. Interpretation Demonstrations

Case One: Subject Allen

Preliminary Examination

Eye Examinations

Psychophysical Tests

Vital Signs Examinations
Dark Room Examinations

Other Evidence

Review the results of the examinations for injection sites and muscle tone, and of the final

Opinions of Evaluator
Case Two: Subject Brown

Preliminary Examination

Eye Examinations

Psychophysical Tests

Vital Signs Examinations
Case Two: Subject Brown

• Dark Room Examinations
• Other Evidence
• Opinions of the Evaluator

Dark Room Examinations

Other Evidence

Opinions of Evaluator
B. Interpretation Practice

Team Practice

Feedback of Results

Session Wrap-Up
QUESTIONS?
Evaluator: Officer Ed Finnegan  
Recorded by: Lt. Tom Reagan  
Arrestee's Name: Allen, Thomas G.  
Date examined/time examined: 03/21/14 1340  
Officer's Signature:  

**DRUG INFLUENCE EVALUATION**

**Date of Birth:** 09/03/88  
**Arresting Officer (Name, ID#):** Trooper Aaron Turcotte, #11644  
**Arresting Agency:** Maine State Police  
**Session XXV - #1**

**Evaluator ORE #:** 8070  
**Rolling Log #:** 14-03-077  
**Case #:** 14-8890  
**Location:** Bangor PD  
**Chemical Test:** N/A  
**Test or tests refused:** N/A  
**Drugs or medications have you been using?** N/A  

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**Arresting Agency:** Maine State Police  
**Session XXV - #1**
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Allen, Thomas

1. LOCATION: The evaluation was conducted in the interview room at the Bangor PD.

2. WITNESSES: Lt. Tom Reagan of Bangor PD witnessed and recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was contacted by Trooper Turcotte requesting a drug evaluation. I met Trooper Turcotte at the BPD and was advised that he had arrested the suspect for DUI after observing his vehicle being operated without headlights, and traveling 15 mph under the posted speed limit. Upon contact, the suspect was disoriented, and had slow, lethargic movements. He had poor balance and coordination, and was unable to perform the SFST’s as directed. No HGN was observed, but the suspect’s eyes were red and bloodshot, and his pupils were dilated.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at BPD. He seemed disinterested and unconcerned about his arrest. He was unsteady on his feet. Several times he used the wall to steady himself. His speech was slow and thick. His eyes were bloodshot, and his pupils appeared to be dilated.

6. MEDICAL PROBLEMS AND TREATMENT: None noted or stated.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Suspect had an approximate 2” circular sway, and estimated 30 seconds in 38 seconds. Walk & Turn: Suspect lost his balance twice during the instructions stage, missed touching heel to toe five times on the first nine steps, and three times on the second nine steps. He raised his arms for balance five times, and made a slow, walking turn. Leg tremors were present throughout the test. One Leg Stand: Suspect swayed while balancing, used his arms to balance, and put his foot down once while standing on his left foot and twice standing on his right foot. Finger to Nose: Suspect missed the tip of his nose on all six attempts and eyelid tremors were present.

8. CLINICAL INDICATORS: LOC and rebound dilation were present. His pupils were dilated in all the lighting levels. His pulse and B/P were above the DRE average ranges.

9. SIGNS OF INGESTION: The suspect had a brownish-green coating on histongue.

10. SUSPECT’S STATEMENTS: The suspect denied using drugs.

11. DRE'S OPINION: In my opinion, the suspect is under the influence of _________________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the suspect.

13. MISCELLANEOUS:

Rev. 10/15
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: **Brown, Jerome A.**

1. **LOCATION:** The evaluation was conducted in Bedford PD Interview Room.

2. **WITNESSES:** Trooper Beaudoin witnessed and recorded the evaluation.

3. **BREATH ALCOHOL TEST:** The suspect’s breath test was 0.00%.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** I was contacted by Officer Humphrey requesting a drug evaluation. I met Trooper Beaudoin and Officer Humphrey at the Bedford PD where it was determined that the suspect had nearly hit a BPD officer while on a traffic stop. When stopped, the suspect was non-responsive, had a blank stare, and was sweating profusely. Six clues of HGN and VGN were observed. The suspect performed very poorly on the SFST’s, and was arrested for DUI.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the breath testing room. He was looking straight ahead with a blank stare. When asked questions he responded slowly, and at times did not respond at all. His speech was slow and thick, and several times he repeated his answers. He was unsteady on his feet. When he stood, he would stagger, and nearly fell several times. The suspect was perspiring heavily.

6. **MEDICAL PROBLEMS AND TREATMENT:** None noted or stated.

7. **PSYCHOPHYSICAL TESTS:** Modified Romberg Balance: The suspect had an approximate 3” side to side sway and estimated the passage of 30 seconds in 55 seconds. Walk & Turn: The suspect lost his balance twice during the instructions, stopped while walking three times, missed heel to toe on every step, and raised his arms for balance. His movements were rigid-like and slow. One Leg Stand: The suspect lost his balance while attempting this test and nearly fell. After several attempts, the test was stopped for safety reasons. Finger to Nose: Suspect missed the tip of his nose on each attempt. He also kept his finger in contact with his face on each attempt. His arm movements were slow and rigid.

8. **CLINICAL INDICATORS:** HGN, VGN, LOC, and Rebound Dilation were present. His pulse, blood pressure and temperature were all above the DRE average ranges.

9. **SIGNS OF INGESTION:** A green coating was observed on the suspect’s tongue.

10. **SUSPECT’S STATEMENTS:** Suspect did not respond when asked about drug use.

11. **DRE’S OPINION:** In my opinion, the suspect is under the influence of a ________________ and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** A blood sample was collected from the suspect.

13. **MISCELLANEOUS:**

Rev. 10/15
DRUG INFLUENCE EVALUATION

�luator

Officer Cullen Kau

Recorder I Witness

Sgt Ben Moszkowic:z

ame t� rm:il, 1Y1Ndle)

Cole, Ricky Lee

0200

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HPD Intake

14-05-61

I

06104/94

-se#

Ko.....,., -i:,,,.

Crash: � None
0Fata1Fi1ni...., np.....-,
Date of Birth
Sex

Honolulu PD

Date Examined f Time I Location

05107/14

I

DRc•
5992

Honolulu PD

M

Breath Results:
Results::
0.00

I

14-70785

Officer Michelle Yosh1ki

Kw

"Mavbe 3 am" I 0208 I

O Yes IE] No
Are you taking any medication or drugs?
Yes
No

D

G1

Speech:
Slow, Slurred, Thick
Corrective tenses:

I
I

Last ni�ht 4 or 5 hours

Do you take insulin?

�None

#13052

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VITIU:lr 1-\Qt!inc:y:
� luPD
Hano

Test Refused
Instrument #

unne L?tl
Blood LJ
l Test
lChemica
Test or tests refused D
What have you been drinking? How much
Time of last drink?

Miranda Warning Given
�Yes
Wrat have you eaten today? VVhen?
f
Given by;
Officer Yoshiki D No
Rice Bowl 7 pm
rnne now I Actual
VVhen did you last sleep? How long?
Are you sick or injured?

I

Session XXV - #3

Arresling Officer (Name, ICJ!ll
LJ

I

98902

Red Bull

D Yes 0No

Do you have any physical defects?

D Yes 0 No

Attitude:

Passive, Cooperative

Breath Odor:

One can

l

race:

Rancid

Poor, Staggeling at times

Flushed

Eyes: U Reddened Conjunctiva

Blindness:

I

D Nonna! 0 Bloodshot 0 Wateiy
0 NoneD LeftD Right
D �-ntacts it so DttaroDSoft
Pupil Size:
0Equal
vernca1 Nystagmus
Able to follow stimulus
Du
0Yes D No
ain)
0Yes DNo
Pulse and time
HGN
Left Eye
Right Eye
Left Count
Conve,gence
1 . ....1Q§_ I 0214
Lack of Smooth Pursuit Present
Present
2. _1QL I 0222
Maximum Deviation
Present Present
3. �/ 0240
Right eye
left eye
Angle of Onset
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Modified Romberg Balance Wal nd Tum T4i
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Cannot keep balance ,/,/
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Starts too soon

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estimated as 30 seconds

45

Steps off line

uescnoe 1um
Quick steps.

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I

Actual steps taken

I

-��

Blood pressure

Rebound Dilation:
DYes lEJNo

1
3

6

Officer's Signature:

0115

Opinion of Evaluator:

0140

DNot Impaired
DMe<fical

v'
v',/

One Leg Stand

.

I

Evaluation start time:
u"�"

DAloohol
,OCNS Depressant

(9

i

Sways while balancing
Uses arms to balance
Hops
Puts foot down

Nearly fell. Tests stopped.
Nasal area:

Redness

OraJ cavity:

-

Pupillary Un
�
0Yes
x No

aear
J

Reaction to Light

Normal
LEFT ARM

::?:".::;}

�

�

===

�

Chemical-like odor on hands and fingers

I

I NIA

Time of use?

How much?

0200

R

�

Right Count

T�pe offootwear:
F 1p..flops

4.0
4.0

� ==

DRigid

0

05/07114

6.5
6.5

�

Temperature

0Raa:id

,/,/

2.0-4.5

:I::

�

Commenls:
What drugs or mecflcations have you been using?
J
NIA
"None. -I'm not usinQ druqs.
Date I Time of arrest
Time DRE was notified:

L

00
0 0
00
DD

,/../

9

5.0-8.5

RIGHT ARM

98.8 0

__H§_/�

DNormal

4.5
4.5

Right Eye

Swaying. Opened eyes on each auempt.

Musdetone:

2.5-5.0

._•,

�

9

.,

2nd Nine

Cannot do test (explain)
NIA
Room
Light Darkness
Direct
PUPIL SIZE
left Eye

' A

, .,

'

./
,t

Raises-arms

Finger to Nose
(Draw lines to spots touched)

2

v'v'
v'.IV

Misses heel-toe

Circular sway.

mremal CIOCK

1st Nine

SIDps walking

.

Tracking:

0 Equal D Unequal
Eyelids � Notmal
Doroopy

(:3)(2;

2" 2" 2" 2"

NIA

Are you diabetic or epileptic?
D Yes IE] No
Are you under the-care of a dodor or dentist?
Oves 0 No
Coordination:

J

I NIA

Evaluation completion time:

Reviewed/approved

by,_
0255

E]C.NS Stimulant
DHallucinogen

>

I

VVhere were the drugs used? (Location}
Precinct/Station:

DDissociative Aneslhetic
ONan:oticAnalgesic

[:llnhalant

ocannabis

R<w0VI5


DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Cole, Ricky Lee

1. LOCATION: The evaluation was conducted in the Interview Room at the Honolulu PD.

2. WITNESSES: Sgt. Ben Moszkowicz of the HPD witnessed and recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was contacted by Officer Yoshiki requesting a drug evaluation. Officer Yoshiki advised that she detained the suspect after observing his vehicle fail to stop at a red traffic light at King Street and University Ave. The suspect’s speech was slow, slurred and thick. Six clues of HGN were observed, but no alcohol was detected on the suspect’s breath. He was unable to complete the SFST’s as directed. He nearly fell several times and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at HPD. He appeared passive and confused. He had poor balance and coordination. He swayed when he stood and staggered several times when he walked.

6. MEDICAL PROBLEMS AND TREATMENT: Suspect reported being lightheaded.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately 2” front to back and side to side. He estimated the passage of 30 seconds in 45 seconds. Walk & Turn: The suspect lost his balance twice during the instructions stage, stopped while walking twice on the first nine steps and three times on the second nine steps. He missed touching heel to toe five times, and stepped off the line twice. One Leg Stand: The suspect was unable to maintain his balance on either foot and nearly fell. The test was stopped for safety reasons. Finger to Nose: The suspect was unable to touch the tip of his nose on any of the six attempts. He repeatedly opened his eyes, and swayed noticeably.

8. CLINICAL INDICATORS: Suspect had six clues of HGN. VGN and LOC were also present. His pulse and blood pressure were elevated and above the DRE average ranges.

9. SIGNS OF INGESTION: The suspect had a severe redness to his nasal area. He also had a strong chemical-like odor on his clothing and hands. He had bloodshot and watery eyes.

10. SUSPECT’S STATEMENTS: Suspect denied using any medications or drugs.

11. DRE’S OPINION: In my opinion, the suspect is under the influence of an ________________ and is unable to operate a vehicle safely.

12. TOXI COLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS:
**DRUG INFLUENCE EVALUATION**

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Officer Greg Jensen</th>
<th>Lakeville PD</th>
<th>DRE # 8174</th>
<th>14-10-088</th>
<th>14-45902</th>
<th>Session XXV - #4</th>
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<td>Recorder/Witness</td>
<td>Sgt. Daniel Day</td>
<td>St. Paul PD</td>
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<td>Fatal: [ ]</td>
<td>Injury: [ ]</td>
<td>Property: [ ]</td>
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<td>(Draw lines to spots touched)</td>
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<td></td>
<td>56 / 2008</td>
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<tr>
<td>Pupil Size:</td>
<td>Equal</td>
<td></td>
<td>Unusual (explain)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Pulse and time</td>
<td>5 6 / 1940</td>
<td></td>
<td>58 / 1954</td>
<td></td>
<td>56 / 2008</td>
<td></td>
</tr>
<tr>
<td>Pupil Size:</td>
<td>Equal</td>
<td></td>
<td>Unusual (explain)</td>
<td></td>
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</tr>
</tbody>
</table>
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Davis, Paul J.

1. LOCATION: The evaluation was conducted in interview room at the Hennepin Co Jail.

2. WITNESSES: Sgt. Daniel Day of the St. Paul Police Department recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’ breath test was 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Officer Engle of the MPD for a drug evaluation. Officer Engle advised that he located the suspect slumped over the steering wheel of his vehicle parked along the shoulder of W. 13th Street. The vehicle was in gear and the suspect had his foot on the brake. The suspect’s speech was slow, low and raspy. He had slow movements and he was unstable on his feet. He had difficulty completing the SFST’s and was arrested for DUI.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the suspect in the interview room at the jail. He was having difficulties keeping his eyes open. His head was continually nodding forward, and he had droopy eyelids. When he spoke, his voice was slow, low and raspy. He was continually scratching his face and arms and he complained of being cold.

6. MEDICAL PROBLEMS AND TREATMENT: The suspect said he felt cold and nauseous but did not request or need medical assistance.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: The suspect swayed approximately two inches front to back and side to side. He estimated the passage of 30 seconds in 68 seconds. Walk & Turn: The suspect lost his balance twice during the instructions stage, stopped while walking four times, missed heel to toe three times, stepped off the line three times, and raised his arms for balance. One Leg Stand: The suspect put his foot down three times on both the left and right foot, and the tests were stopped for safety reasons when he nearly fell on each attempt. Finger to Nose: The suspect missed the tip of his nose on five of the six attempts. His arm and hand movements were slow and deliberate.

8. CLINICAL INDICATORS: The suspect’s pupils were constricted in all three lighting levels. His pulse, blood pressure and temperature were below the DRE average ranges.

9. SIGNS OF INGESTION: A fresh injection mark was located on the back of his left hand.

10. SUSPECT’S STATEMENTS: The suspect made several references to being “clean.”

11. DRE'S OPINION: In my opinion, the suspect is under the influence of a ________________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A urine sample was collected from the suspect.

13. MISCELLANEOUS:  

Rev. 10/15
DRUG INFLUENCE EVALUATION

Evaluator: Susan Reidenbach
Officer: Indianapolis Metro

Officer Zach Dodd
Hamilton Co. SO

Date Examined/Time/Location: 11/05/14, 1810 Downtown Metro Office

Recorder: Zach Dodd
Officer: Hamilton Co. SO

Date of Birth: Elliott, John B. 06/01/80

Name: Elliott, John B.
Age: 06/01/80

Date of Birth: 06/01/80

Sex: Male

Blood pressure: 176 / 118

Temperature: 99.0 o F

Muscle tone: Normal

Comments: Nothing observed.

What drugs or medications have you been using? I don't use drugs, but probably should.

How much? N/A

Time of use? N/A

Where were the drugs used? (Location) N/A

Date/Time of arrest: 11/05/14, 1722

Time DRE was notified: 1750

Evaluation start time: 1810

Evaluation completion time: 1920

Officer's Signature:

Opinion of Evaluator: Not Impaired

- Alcohol
- Medical
- CNS Stimulant
- CNS Depressant
- Hallucinogen
- Dissociative Anesthetic
- Narcotic Analgesic
- Inhaling
- Cannabis
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: Elliott, John B.

1. LOCATION: The evaluation was conducted at the Indianapolis Metro Downtown Office.

2. WITNESSES: Deputy Zach Dodd of the Hamilton County SO recorded the evaluation.

3. BREATH ALCOHOL TEST: The suspect’s breath test was a 0.00%.

4. NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER: I was requested to contact Officer Rector for a drug evaluation. According to Officer Rector, the subject was driving his vehicle without headlights, failed to stop at a red light, and severely injured a pedestrian crossing the street in the crosswalk. The subject was acting very strange and was highly emotional at times. His speech was rambling, and at times was incoherent. He had difficulties maintaining his balance and several times nearly fell when walking.

5. INITIAL OBSERVATION OF SUSPECT: I first observed the subject in the interview room at the Downtown District Office. He was having problems with his balance and was very unsteady on his feet. At times he was laughing, and then would get emotional and start crying. He was talking to himself. His speech was rambling and at times incoherent.

6. MEDICAL PROBLEMS AND TREATMENT: The subject stated that he had been seeing a doctor for some “issues” but stopped going to his appointments.

7. PSYCHOPHYSICAL TESTS: Modified Romberg Balance: Subject swayed approximately 2” front to back and side to side, and estimated 30 seconds in 32 seconds. Walk & Turn: He could not maintain his balance in the instructions stage and the test was stopped for safety reasons. One Leg Stand: He could not stand on one foot and several times nearly fell. The test was stopped for safety reasons. Finger to Nose: Subject could not touch the tip of his nose as directed, and used the pads of his fingers on all the attempts.

8. CLINICAL INDICATORS: The subject’s left pupil sizes were within the DRE average ranges and two of his right pupil sizes were outside the DRE average ranges (Room Light and Direct Light). The subject had no explanation regarding the difference in his pupil sizes. His blood pressure was above the DRE average ranges.

9. SIGNS OF INGESTION: Nothing was observed or noted.

10. SUSPECT’S STATEMENTS: The subject denied using drugs or medications. He stated that his doctor wanted to prescribe him some medications, but he refused.

11. DRE'S OPINION: In my opinion, the subject is under the influence of a ____________ and is unable to operate a vehicle safely.

12. TOXICOLOGICAL SAMPLE: A blood sample was collected from the subject.

13. MISCELLANEOUS:

Rev. 10/15
Upon successfully completing this session the participant will be able to:

- Discuss the essential elements of the drug influence evaluation report.
- Prepare a clear and concise narrative description of the results of the drug influence evaluation.

CONTENT SEGMENTS................................................................. LEARNING ACTIVITIES
A. Components of the Process............................................. Instructor-Led Presentations
B. Components of the Drug Evaluation Report .................... Interactive Discussion
C. Drug Evaluation Narrative Report Format
D. Sample Report
A. Components of the Process

The DRE Report

Successful prosecution depends on how clearly, completely and convincingly the DRE presents their observations, measurements, and conclusions.

A well written, clear, and convincing drug evaluation report increases the likelihood that the suspect will be convicted.

- A prosecutor is more likely to file the charge if the evidence is organized, clearly documented and compelling.
- The defense is less likely to contest the charge when the report is descriptive, detailed, and complete.
B. Components of the Drug Influence Evaluation Report

The Face Sheet

The Drug Influence Evaluation Face Sheet is part of your drug influence evaluation report; but it is not the entire report.

The Face Sheet contains some very important information.

Examples:

- Suspect’s pulse rate was elevated on all three measurements.
- Suspect’s eyes failed to converge.
- Suspect’s pupils were constricted.

But the Face Sheet does not contain all of the important information that is available concerning this suspect.
Most importantly, the Drug Influence Evaluation Face Sheet is a technical document.

- Trained DREs know how to complete and interpret the Face Sheet.

Examples:

- Information obtained during the interview of the arresting officer.
- Elaborate or lengthy statements made by the suspect.
- Paraphernalia found in the suspect’s possession.

Many prosecutors, judges, and jurors won’t know how to interpret the face sheet.

- It is up to you to take all of the information you work so hard to obtain, and put it into a clear, plain English, written report so that the prosecutor, the judge, and the jury will understand what you observed and what it means.
To ensure that the information contained on the Face Sheet is systematic and standardized, the results of the tests should be recorded as follows:

**Lack of Convergence**

- A dot should be made where the pupil is and draw an arrow to indicate the movement and where the pupil stops.

**Modified Romberg Balance**

- The first figure indicates the sway from front to back and should be estimated in inches from center.
- The second figure indicates the sway from side to side and is estimated in inches from center.
- Put the approximate number of inches from center the suspect sways on either end of the arrows.
- Record actual elapsed time.
**Drug Influence Evaluation Face Sheet**

**How to Record the Walk and Turn Test Results**

<table>
<thead>
<tr>
<th>Walk and Turn Test</th>
<th>Cannot keep balance</th>
<th>Starts too soon</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Walk and Turn</th>
<th>1st Nine</th>
<th>2nd Nine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Describe Turn</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Walk and Turn**

- The first two – cannot keep balance and starts too soon – are observed during the instruction stage.
- Indicate by a check mark the number of times the suspect stops, misses heel-to-toe, steps off line, or raises arms.
- Record the actual number of steps taken.
- If the suspect stops walking, indicate where with a vertical slash mark and an “S” under that mark.
- If the suspect steps off the line, indicate with half of a slash mark at an angle in the direction the step was off the line.
- If the suspect misses heel-to-toe, indicate with a vertical slash mark and an “M” under that mark.
- Describe turn.
**Drug Influence Evaluation Face Sheet**

**How to Record the One Leg Stand and the Finger to Nose tests**

<table>
<thead>
<tr>
<th>One Leg Stand:</th>
</tr>
</thead>
<tbody>
<tr>
<td>L</td>
</tr>
<tr>
<td>Sways while balancing</td>
</tr>
<tr>
<td>Hopping</td>
</tr>
</tbody>
</table>

**Type of Footwear**

- Right
- Left

Draw lines to spots touched

---

**One Leg Stand**

- Indicate in the one leg stand box the number they were counting when they put their foot down.
- Check marks should be made to indicate the number of times the suspect swayed, used arms, hopped, or put foot down.
- Indicate how far the suspect counted in 30 seconds in the top area of the box above the foot raised.

**Finger to Nose**

- A line should be drawn to the appropriate triangle or circle to indicate where the suspect touched their nose.
- Suggestion – If the DRE draws the line from the place where the suspect touches to the triangle it enables them to draw a straighter line.
C. Drug Evaluation Narrative Report Format

The Narrative Report

The typical Drug Evaluation Narrative Report format contains 13 components.

First item: Location (i.e. where the evaluation was conducted).

Second item: Witnesses

- List the person who served as the evaluator and the recorder with the complete agency name spelled out.
- Other officers who helped to conduct the evaluation.
- Others who observed the evaluation.
- Include any instructors who witnessed the evaluation.

Third item: the Breath Alcohol Test

- Indicate BAC.
- Who administered the breath alcohol test.
- Time the test was administered.

Fourth item: Notification and Interview of the Arresting Officer

- When were you first notified of the request for a drug evaluation?
- Summarize the information you were given at that time.
- Document any information provided by the arresting officer.
- Summary of your interview with the arresting officer and other witnesses.
Components of the Drug Evaluation Narrative Report

- Initial observations of the suspect
- Medical problems and treatment
- Psychophysical indicators of impairment

Fifth item: Initial Observation of the Suspect
- Where you first saw the suspect.
- Noteworthy aspects of your initial observations.
- Findings of the Preliminary Examination of the suspect.

Sixth item: Medical Problems and Treatment
- Your observations of any apparent injury or illness affecting the suspect.
- Suspect’s statements of injury or illness.
- Summary of any medical treatment provided to the suspect.

Seventh item: Psychophysical Indicators of Impairment
- Briefly summarize performance of the Modified Romberg Balance, Walk and Turn, One Leg Stand, and Finger to Nose tests.
- Include any relevant behaviors on the tests that are not included on the face sheet.
- Document any observations of eyelid tremors.
Components of the Drug Evaluation Narrative Report

- Clinical indicators of impairment
- Signs of ingestion

Eighth item: Clinical Indicators of Impairment

Eye signs

- Briefly summarize your observations of HGN, VGN, Lack of Convergence, pupil size, reaction to light, and appearance of the suspect’s eyes.

Vital signs

- Briefly summarize the suspect’s pulse rate, blood pressure, and temperature.

Ninth item: Signs of Ingestion

- Results of examinations of oral and nasal cavities.
- Results of examinations for injection marks.
- Odors detected on suspect’s breath, hands, clothing, etc.
- Physical debris of drugs or drug paraphernalia found on suspect’s person.
Components of the Drug Evaluation Narrative Report

- Suspect's statements
- DRE’s opinion

Tenth item: Suspect’s Statements.

- “Miranda” waiver and responses.
- Volunteered or spontaneous statements.
- Statements made as a result of your interview.
- Include admission or denial of drug use, time, location drugs were used, and statements relating to the suspect’s perception of their impairment, if applicable.

Eleventh item: DRE’s Opinion.

- State the category or categories of drugs that you believe is/are affecting the suspect.
- State your opinion concerning the suspect’s ability to operate a vehicle safely, if applicable to this case.
Components of the
Drug Evaluation Narrative Report

- Toxicological sample
- Miscellaneous

Twelfth item: Toxicological Sample

- State who drew the sample or observed the collection of the sample.
- State where the sample was taken and to whom it was given.
- If the suspect refused to provide a sample, state that fact.

Thirteenth item: Miscellaneous

Any other pertinent information such as drugs or drug paraphernalia found in the suspect’s possession.
D. Sample Report

A sample report is found at the end of this session, for your reference.
Evaluator: Officer Daven Byrd  
Arizona DPS  

Arrestee's Name (Last, First, Middle): Roach, Robert A

Recorder: Sgt. Paul White  
Maricopa Co. SO  

Date Examined: 10/21/14  

Miranda Warning Given: Yes  
Given by: Officer Byrd  
Arizona DPS  

Date of Birth: 04/20/88  

Corrective Lenses: None

Modified Romberg Balance Test:

- Walk and Turn Test: 5 steps  
  - Eyelid tremors observed throughout the test.  
  - Cannot keep balance

- Walk in a circle  
  - Slow movements. Eyelid tremors. Laughing.

Blood pressure: 162 / 98  
Temperature: 97.4°F

Muscle tone: Normal

Comments:
- What drugs or medications have you been using?  
  - I smoked some pot.
- How much?  
  - About 9 pm
- Where were the drugs used? (Location)  
  - At home and in my car.

Chemical Test: Urine  
Results: 3450 PBT

Opinion of Evaluator: Not Impaired

Type of footwear: Unlaced boots

PUPIL SIZE

- Room Light: 2.5 – 5.0  
  - Left Eye: 6.0  
  - Right Eye: 6.0

- Darkness: 5.0 – 8.5  
  - Left Eye: 9.0  
  - Right Eye: 9.0

- Direct: 2.0 – 4.5  
  - Left Eye: 5.0-6.5  
  - Right Eye: 5.0-6.5

Nasal area: Clear

Ocular cavity: Normal

Reaction to Light: Normal
DRUG INFLUENCE EVALUATION NARRATIVE

Suspect: **Roach, Robert A.**

1. **LOCATION:** The drug influence evaluation was conducted in the holding cell and hallway area at the Maricopa County 4th Avenue Jail. Both areas have adequate lighting and have a concrete floor with no obstructions for conducting the evaluation. The dark room examinations were conducted in the jail staff restroom.

2. **WITNESSES:** Sergeant Paul White of the Maricopa County SO witnessed and recorded the entire evaluation. The arresting officer, Officer Trevor Graff of the Arizona DPS, observed the preliminary exam and the psychophysical tests.

3. **BREATH ALCOHOL TEST:** Officer Graff obtained a breath test from the suspect prior to my arrival and obtained a 0.00 BAC result at 2050 hours.

4. **NOTIFICATION AND INTERVIEW OF THE ARRESTING OFFICER:** On 10/21/14, I was on-duty and at approximately 2115 hours was dispatched to the Maricopa County Jail to conduct a drug evaluation for Officer Graff of the Arizona DPS. I was advised by Officer Graff that he had arrested the suspect during a West Valley DUI Task Force enforcement event. According to Officer Graff, the suspect was driving over the posted speed limit on Grand Ave and failed to stop at a red light at West Greenway Road. When Officer Graff activated his emergency lights to stop the suspect, he continued on for approximately a half mile before stopping. When asked for his operator’s license and other documents, the suspect appeared confused, and had slow and deliberate movements. When the suspect exited his vehicle, he had to use the side of his vehicle to steady himself. Officer Graff administered SFST’s which the suspect was unable to perform as directed. According to Officer Graff, the suspect exhibited four clues on the Walk and Turn test and three clues on the One Leg Stand test. No clues of HGN were present and no odor of an alcoholic beverage was detected on the suspect’s breath. Officer Graff did detect an odor of marijuana coming from the suspect’s clothing and from his vehicle.

5. **INITIAL OBSERVATION OF SUSPECT:** I first observed the suspect in the booking area at the 4th Avenue Jail. The suspect was moving slowly and was unsteady of his feet. When he answered questions from the jail staff, his speech was slow, and at times difficult to hear. The suspect’s eyes were bloodshot and watery, his pupils were dilated, and his eyelids appeared to be droopy.

6. **MEDICAL PROBLEMS AND TREATMENT:** When asked about medical problems, the suspect indicated that he had a “sore back” and smoked pot to relieve the pain. When asked if he had a Medical Marijuana Card, he indicated that he did not, but was looking into it. When asked further questions about his back, the suspect indicated that it was just sore, and that he was not under a doctor’s care. He was asked if his sore back would cause any problems in performing the drug influence evaluation and he stated, “It shouldn’t.” The suspect was asked if he needed any medical assistance, and he indicated that he did not.
7. **PSYCHOPHYSICAL TESTS:** Each of the tests were explained and demonstrated to the suspect prior to him attempting them. After each demonstration, the suspect indicated that he understood the instructions. The following psychophysical tests were administered:

**Modified Romberg Balance:** During this test, the suspect exhibited a front to back sway of approximately two inches, and a side to side sway of approximately three inches. He had a slowed internal clock, estimating 30 seconds in 42 seconds. When asked how he estimated the 30 seconds, the suspect stated, “I was trying to count, but I kind of forgot.” Eyelid tremors were present throughout the test.

**Walk and Turn:** The suspect was asked if his boots would create any difficulties in performing the test. He replied that they would not. For this test, a painted line on the concrete floor was used. During this test, the suspect lost his balance twice during the instructions stage. Once he started walking, his steps were slow and deliberate. On his 8th step, the suspect stopped walking to regain his balance and raised his arms for balance. He then stopped at the turn, and appeared confused. He then made an incorrect turn by walking in a circle, using both feet. On the second nine steps, the suspect stopped while walking on his 3rd step to regain his balance. He raised his arms for balance three times during the second nine steps. Leg tremors were present throughout the test.

**One Leg Stand:** While balancing on his left foot, the suspect counted slowly, counting to 1024 when the test was stopped. He also swayed while balancing, and used his arms for balance twice. While balancing on his right foot, he put his left foot down at 1015. He also swayed while balancing, and used his arms for balance three times. His count was again slow, counting to 1022 when the test was stopped. The suspect displayed leg tremors throughout the test. He also counted incorrectly while standing on his right foot, skipping the numbers “1012” and “1019”.

**Finger to Nose:** During this test, the suspect responded to the touching sequence commands very slowly. He did not touch the tip of his nose as directed on attempts 1, 2, 4 and 5. Eyelid tremors were present throughout the test. Several times he started laughing when attempting to touch his nose.

8. **CLINICAL INDICATORS:**

Eyes: The eye examinations were conducted in the staff restroom. No clues of HGN were observed. His pupils were dilated in all three lighting levels, and were above the DRE average ranges. The DRE average ranges for pupil sizes are: 2.5 – 5.0 mm in Room Light, 5.0 – 8.5 mm in Near Total Darkness, and 2.0 – 4.5 mm in Direct Light. A Lack of Convergence and Rebound dilation were present. The suspect’s eyelids were droopy, and his eyes were bloodshot and watery.

Vital Signs: Per DRE protocol, the suspect’s pulse rates were measured three times and were 98, 96, and 98 beats per minute. All were elevated and above the DRE average ranges of 60 – 90 beats per minute. His blood pressure was measured at 162/98, which is also above the DRE average ranges for blood pressure. The DRE average ranges for blood pressure are 120 – 140 for the Systolic pressure, and 70 – 90 for the diastolic pressure.
The suspect’s body temperature was measured at 97.4, which is below the DRE average range of 98.6 plus or minus 1 degree.

The suspect was asked about his elevated pulse rates and blood pressure. He indicated that he was not aware of any issues that would cause both to be elevated.

9. **SIGNS OF INGESTION:** The suspect’s nasal area was clear. A greenish coating was present on the back of the suspect’s tongue.

10. **SUSPECT’S STATEMENTS:** The suspect admitted smoking a bowl of marijuana about 25 minutes prior to being stopped. He also admitted smoking marijuana earlier in the day at about 10 am. He also indicated that he uses marijuana frequently, and has been smoking marijuana several times a week since he was 16 years old. I asked if the marijuana he had smoked earlier in the evening had affected him. The suspect stated it was “pretty good weed” and that it gave him a “damn good buzz.” He further stated that he enjoys smoking marijuana because it relaxes him, and he thinks he drives better after smoking marijuana.

11. **DRE’S OPINION:** In my opinion as a Drug Recognition Expert, the suspect is under the influence of Cannabis and is unable to operate a vehicle safely.

12. **TOXICOLOGICAL SAMPLE:** Prior to the drug influence evaluation, a voluntary blood sample was collected from the suspect. The blood sample was collected at 2110 hours, and forwarded to the Arizona DPS Crime Lab for analysis.

13. **MISCELLANEOUS:** The suspect was also cited for Driving While Suspended.

Rev. 10/15
Session 27
Practice: Test Administration
90 Minutes
Learning Objectives

• Administer selected portions of the battery of examinations that constitute the drug influence evaluation
• Describe the evaluation procedures
• Document the results of the examinations

Upon successfully completing this session the student will be able to:

• Administer selected portions of the battery of examinations that constitute the drug influence evaluation.
• Describe the evaluation procedures.
• Document the results of the examinations.

CONTENT SEGMENTS ...................................................... LEARNING ACTIVITIES
A. Procedures for this Session ..................................... Instructor-Led Presentations
B. Hands-On Practice ................................................. Instructor-Led Coaching
C. Session Wrap-Up .................................................... Participant-Led Coaching
A. Procedures for this Session

Team Assignments

• Participants will work in two or three member teams.
• At any given time, one member of the team will be engaged in conducting and recording examinations of another member.
• The third member of the team will help coach and critique the participant who is conducting the examinations.
• Participants will take turns serving as test administrator, test subject, and coach.
B. Hands-On Practice

Drug Influence Evaluation
For this practice session, each participant will conduct a complete drug influence evaluation.

Begin with the Preliminary Examination.
Ask all of the prescribed questions.
Conduct the initial check of the eyes.
Check the pulse for the first time.
Drug Influence Evaluation

• Conduct the test of Horizontal Gaze Nystagmus, Vertical Gaze Nystagmus, and Lack of Convergence
• Administer the four divided attention psychophysical tests
• Check the vital signs

Conduct the test of Horizontal Gaze Nystagmus, Vertical Gaze Nystagmus, and Lack of Convergence.

Administer the four divided attention psychophysical tests.

• Modified Romberg Balance test
• Walk and Turn test
• One Leg Stand test
• Finger to Nose test

Check the vital signs.

• Blood pressure
• Temperature
• Check the pulse for the second time
Dark Room Examinations

- Conduct the dark room examinations.
- Check for muscle tone.
- Examine the participant (subject’s) neck, arms, and ankles for signs of injection.
- Check the pulse for the third time.
C. Session Wrap-Up

QUESTIONS?
Participant Manual

Drug Recognition Expert Course

Session 28
Case Preparation and Testimony
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Upon successfully completing this session, participants will be able to:

- Conduct a thorough pre-trial review of all evidence and prepare for testimony.
- Provide clear, accurate, and descriptive direct testimony concerning drug influence evaluations.
- Respond effectively and appropriately to cross examine in DRE cases.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES
A. Guidelines for Case Preparation ................................................... Instructor-Led Presentations
B. Guidelines for Direct Testimony ............................................... Instructor-Led Demonstrations
C. Typical Defense Tactics ................................................................. Reading Assignments
A. Guidelines for Case Preparation

Preparation

Preparation to present your case in court begins during your initial investigation. The quality of your investigation and documentation will ultimately determine your ability to accurately present information during trial.

When you receive the trial notice you should schedule a pre-trial conference with the prosecutor.

- Review all records and reports associated with the case.
- Review all evidence and your conclusion.
- Review notes with arresting officer.
- Review any weak areas.
- Clarify or resolve any discrepancies.
- Review questions the prosecutors will be asking.
- Review typical tactics the prosecutors expect the defense to use.
- Review your curriculum vitae and credentials.

If a pre-trial conference is not possible, identify the main points of the case and discuss them with the prosecutor during the few minutes before the trial.

- It is very important to meet with prosecutors that have never been exposed to the DEC Program before trial to explain that it can not be treated like a typical DUI trial. You must explain that there are different protocols for DUI vs. DRE cases.
- Excellent resources for prosecutors can be obtained through the National Traffic Law Center. Another excellent resource is your state’s Traffic Safety Resource Prosecutor (TSRP).
B. Guidelines for Direct Testimony

**Direct Testimony**

Although knowledge only greater than what the public has is required to qualify as an “expert,” your testimony will carry much more weight if you have good credentials.

Qualifications will be established during Voir Dire:

Voir Dire is a French expression literally meaning “to see, to say.” Loosely, this would be rendered in English as “to seek the truth,” or “to call it as you see it.” In a law or court context, one application of voir dire is to question a witness to assess his or her qualifications to be considered an expert in some matter pending before the court.

When testifying, relate training and experience to the type of arrest being tried (e.g., DWI, Methamphetamine, Cocaine, etc.)

Being qualified as an expert in the past does not automatically qualify you as an expert in a particular court case.

- Highlight fact that you were selected to attend specialized DRE training, not just assigned randomly.
- If possible, do not allow the defense to stipulate that you are an expert.
- Document and record all evaluations conducted. Establish ratio of evaluations that resulted in a finding that the subject was not under the influence.
- Highlight the number of times you have seen a person under the influence of the drug(s) in question and have observed the symptomatology, etc.
- Ability to answer specific questions with confidence, skill and exactness will bolster a professional image in the eyes of the judge and/or jury.
New Scientific Principle

- Remember that jurors are unfamiliar with most scientific principles.
- American courts employ either the Frye or the Daubert standards for determining the admissibility of scientific evidence.

“Frye vs. U.S.” (D.C. Cir. 1923)

Courts assess scientific testimony by considering four factors:
- Opinions that are testable.
- Peer reviewed methods/principles.
- Known error rates.
- Methodology accepted within the scientific/technical community.


Frye requires that the scientific principle or theory used to support “evidence” be in conformity with a generally accepted explanatory theory, if the “evidence” is to be admissible.

In Daubert, courts serve as a gatekeeper for all scientific evidence.


Courts assess evidence by considering four factors:
- Opinions are testable.
- Methods/principles have been subject to peer review.
- Known error rate can be identified.
- Opinions rest on methodology that is generally accepted within the relevant scientific/technical community.
General Guidelines

- Basic job – To present the findings of your investigation that the suspect was under the influence of a drug or some combination of drugs.
- Don’t be afraid to say “I don’t know.”
- Remember that some jurors focus on officer demeanor more than content of testimony.

Avoid contact with the defense attorney if possible.

Don’t be upset if prosecutor and defense attorney appear friendly to each other.
- Remember, some jurors focus on an officer’s demeanor more than content of testimony.
General Guidelines

• Review materials before court
• Use layman’s language
• Don’t testify on subject matter that was excluded
• Do not use “pass” or “fail”
• Be prepared to describe DRE terms if used

Subject’s performance is describable evidence
• All evidence taken into account before forming an opinion
• Explain “why” in great detail

Do not bring manuals or articles into court for reference.

• Review materials before court to become familiar with contents.
• Explain technical terms in layman’s language. For example, HGN means an involuntary jerking of the eyes occurring as the eyes gaze to the side.
• Pay attention to what evidence or testimony can be and is excluded.

When describing subject’s performance on SFST’s, explicitly describe exactly what the subject did or neglected to do.

• Results of subject’s performance are describable evidence.
• Be sure to emphasize that all evidence is taken into account before forming an opinion.
• If defense attorney asks a “why” question, take the opportunity to explain in great detail if appropriate.
C. Typical Defense Tactics

The defense relies on several factors to “impeach” or discredit your testimony. The defense will challenge your observations and interpretations. They will attempt to show that the signs, symptoms and behaviors observed have other explanations. Defense will challenge your credentials...a bona fide expert has both formal training resulting in a high degree of knowledge and experience in applying knowledge, resulting in a skill. By demonstrating the officer lacks depth of knowledge in the drug field by contrasting his or her knowledge with the defense expert’s knowledge.

- The trial tactic is to show that the officer does not have the expertise to accurately determine the cause of intoxication / impairment because of inadequate formal training which lessens the value of his/her field experience and increases likelihood that he/she is mistaken in his/her conclusion.
Typical Defense Tactics

Challenging your credibility through:

- Inconsistencies
- Comparison with past testimony
- Testimony at odds with other experts
- Lack of recall
- Demonstrating that parts of the drug evaluation were conducted incorrectly

Some examples of challenging your credibility are:

Inconsistencies:

- Arresting officer’s and examining officer’s testimony must be complimentary. Any differences must be explained.
- Get your facts straight and stick to them.

Comparison with past testimony:

- Try to get copies of transcripts of previous trials to review your strong/weak points. If possible, review your testimony with the prosecutor.

Testimony that is at odds with other established experts:

- Do your homework...review the literature. Explain any differences if possible.

Lack of recall:

- Try to be prepared, but don’t be afraid to say “I don’t know.” Be honest.

By demonstrating that the officer incorrectly performed part of the evaluation, resulting in an erroneous conclusion.
Role of Defense Expert

To impeach credibility of the arresting officer and/or the prosecution expert

- My expert vs. your expert. Usually they are 180 degrees apart in their opinions.

To present alternative conditions and states that could have produced the same or similar symptoms

Typical Defense Questions

Pupillary examinations:

- Where the examination took place.
- How dark was the examining room.
- The size or power of the penlight.
- Where the defendant was placed in relationship to the examiner.
- Where the penlight was directed during the examination?
- Where the defendant was looking during the examination?
- How many times each pupil was checked?
- Are there any physical illnesses or conditions that manifest the same signs as the drug(s) in question?
Suggested role play to discuss the following questions:

- What is a DRE?
- What is involved in the DEC training program?
- How do you properly identify the drug category or categories?
- How do you explain the DRE opinion?
- What are the components of a drug influence evaluation?
DRE DEFENSE CROSS EXAMINATION QUESTIONS

The following are representative of questions the defense may use to challenge the DRE’s in court. (The defendant is identified as Miss Alicia Ann Ace.)

**Missing Symptoms/Normals**

*This line of questions attempts to elicit the fact that the defendant did not have all of the expected signs or symptoms of the drug (s) in question.*

Officer, you were taught that bruxism or grinding of the teeth is a sign of CNS Stimulant influence, isn’t it? Miss Ace didn’t have that sign, did she?

*The defense may also focus on those signs or symptoms that were normal, and were therefore, not consistent with the drug in question.*

Officer, you learned the normal range of temperature in DRE training, didn’t you? And that range is 98.6 plus or minus one degree, isn’t it? What was Miss Ace’s temperature? (98) 98 is within normal ranges, isn’t it? Miss Ace’s temperature was normal, wasn’t it? CNS Stimulants cause elevated temperature, don’t they? Miss Ace’s was not elevated, was it?

**Alternative Explanations**

*The defense elicits alternative explanations for the signs and symptoms of the drug (s) in question. These alternative explanations usually deal with medical conditions, stress, a traffic crash, etc.*

Officer, an elevated pulse rate can be caused by things other than drugs, can’t it? Excitement may cause it? Stress may cause it? Being involved in a traffic crash is stressful, isn’t it? And being involved in a traffic crash may cause elevated pulse, right? Being interviewed in the early morning by three police officers is stressful? And that may also cause the pulse to be elevated, can’t it?

**Defendant’s Normals**

*The defense attempts to emphasize the fact that not everyone is so-called normal, that normal is subjective.*

Officer, you were taught the normal range for pulse in DRE training, weren’t you? And you agree that not all people fall in that normal range, don’t you? That there are people with pulse rates above normal that aren’t on drugs, right? A person’s pulse changes over time, doesn’t it? You don’t know what Miss Ace’s normal pulse is, do you? It could be in the normal range, right? But it could be above or below the normal range - normally for her, isn’t that so?
**Doctor Cop**

*The line of questioning challenges the credibility of the officer’s teachers - that they are police officers, rather than medical professionals.*

Officer, the teachers in this DRE school weren’t doctors, were they? They weren’t nurses either? Toxicologists? Pharmacologists? Paramedics? They were police officer, right?

**Just a Cop**

*This line of questioning challenges the DRE’s credentials - that they are “just a cop.” This infers that the DRE evaluation is actually a medical evaluation that should be undertaken only by a medical professional.*

Officer, you’re not a doctor, are you? A toxicologist? A pharmacologist? A nurse? A physiologist? You don’t have a degree in chemistry, do you? You’re a police officer, right?

**The Unknown**

*By causing the officer to state that they don’t know how a sign or symptom is caused, the defense attacks the officer’s credibility. This line of questioning challenges the officer’s expertise, by implying that a real expert would know these things.*

Officer, you don’t know how CNS Stimulants dilate the pupil, do you? You don’t know how alcohol supposedly causes Nystagmus, do you? You don’t know how CNS Stimulants supposedly elevate the heart rate, do you?

**Guessing Game**

*This tactic attacks the DRE’s opinion as a subjective guess, a belief, rather than objective. Guesses can be wrong.*

Officer, your opinion in a DRE case is subjective, isn’t it? It’s a belief on your part? You’ve made these beliefs in DRE cases in the past, haven’t you? A sometimes toxicology didn’t find the drug you predicted, isn’t that so? And, in fact, sometimes, toxicology didn’t find any drug, isn’t that so? And so, sometimes your opinion is not correct, right? Sometimes, you guess wrong?
Drug Recognition
Expert Course

Review of the DRE School
How do we define the term “drug” for DRE purposes?

Basic Drug Statistics

- What drug other than alcohol was found most frequently in the Los Angeles Field Validation Study?
- What does “polydrug use” mean?
**Basic Drug Statistics**

- How common was polydrug use in the LA Field Validation Study?

- How good were the DREs in the Field Validation Study?

**Basic Drug Statistics**

- In the University of Tennessee Study, what percentage of injured drivers had drugs other than alcohol in them?
Review of Symptomatology

- Name six different CNS Depressants
- Name four different CNS Stimulants
- Name two naturally-occurring Hallucinogens
- Name four different synthetic Hallucinogens
- Name a major analog of PCP
- Name the three sub-categories of Inhalants
- What is the active ingredient in Cannabis?
Review of Vital Signs

• Define “Pulse”

• True or false: Pulse rate is measured in units of “millimeters of mercury”.

• Name three different pulse points, and indicate where they are located.

• What is the “normal” range of adult human pulse rate, for DRE purposes?
Review of Vital Signs: Blood Pressure

• Define “Blood Pressure”.

• Name the instrument used to measure blood pressure.

• When does blood pressure reach its highest value? What is the highest value called?

• When does blood pressure reach its lowest value? What is the lowest value called?

• What is the “normal” range of adult human blood pressure, for DRE purposes?

• What does “Hg” stand for?
Review of the Eye Examinations: Horizontal Gaze Nystagmus

• What are the three validated clues of impairment that have been established for HGN?

• What formula expresses the approximate statistical relationship between BAC and the angle of onset of nystagmus?

• What categories of drugs usually will cause HGN?
Review of the Eye Examinations: Vertical Gaze Nystagmus

• True or False: Any drug that causes HGN may also produce Vertical Gaze Nystagmus.

• What category of drugs causes Vertical Gaze Nystagmus but not Horizontal Gaze Nystagmus?

Review of the Eye Examinations: Lack of Convergence

• True or False: Any drug that causes nystagmus will also usually cause the eyes to be unable to converge.

• What category of drugs usually causes lack of convergence but does not cause nystagmus?
Review of the Darkroom Examinations

- What are the three lighting conditions under which we must estimate the size of the suspect’s pupils?

- How long should we wait in the Darkroom before beginning to check the suspect’s pupils?

- Name the device that we use to estimate the size of the suspect’s pupils.

- What do the numbers on the Pupillometer refer to?

- In what units of measurement are those numbers given?
• For DRE purposes, what is the “normal” range of an adult pupil in room light?

• What does the term “MIOSIS” mean?

Review of the Darkroom Examinations

• What does the term “MYDRIASIS” mean?

• What category of drugs usually causes Miosis, or constricted pupils?

• What categories usually cause Mydriasis, or dilated pupils?

• What is unique about the drug Methaqualone (Quaaludes) and SOMA?
Review of the Divided Attention Tests

• Name the four Divided Attention Tests administered during the DRE drug influence evaluation.

• Why is the Modified Romberg Balance always the first test administered?
• What four validated clues of impairment have been established for the One Leg Stand Test?

• How many times is the One Leg Stand administered during the DRE drug influence evaluation?

• Which foot must the suspect stand on first when performing the One Leg Stand?
• How many validated clues of impairment have been established for the Walk and Turn test? Name them.

• In what sequence is the suspect instructed to touch the index fingers to the nose on the Finger to Nose test?
General Review Questions

• What is the medical or technical term for “droopy eyelids”?

• What does “Piloerection” mean? What drug often causes piloerection?

• What is the medical or technical term for Heroin?

• Explain the terms “Null”, “Additive”, “Antagonistic” and “Overlapping” Effect as they apply to polydrug use. Give examples
• What is “Rebound Dilation”?

• What is pupillary unrest?

• What does “Bruxism” mean?
General Review Questions

• What does the number denoting the size of a hypodermic needle refer to?

• What does “Synesthesia” mean?

• What is “Sinsemilla”?

General Review Questions

• What are the twelve major components of the DRE drug influence evaluation?
Review of Physiology

- Name the ten major body systems.

- What is the distinction between the “Smooth” muscles and the “Striated” muscles?

- What do we call the chemicals that are produced by the Endocrine System?

- What is a neuron?
Review of Physiology

• What do we call the space between two nerve cells?

• What do we call the chemicals that pass from one nerve cell to the next?

• What do we call the part of the nerve cell that sends out the neurotransmitter?

• What do we call the part of a nerve cell that receives the neurotransmitter?

• What do the Sensory Nerves do?

• What do the Motor Nerves do?
• Name the two sub-divisions of Motor Nerves.

• Name the two sub-divisions of Autonomic Nerves and describe their functions.

• What does it mean to say that a drug is “sympathomimetic”?

• What does it mean to say that a drug is “parasympathomimetic”?
• Which two categories of drugs can most appropriately be called sympathomimetic?

• Which category can most appropriately be called parasympathomimetic?

Review of Physiology

• What is an artery?

• What is a vein?
Review of Physiology

• What are the Pulmonary Arteries, and what are unique about them?

• What are the Pulmonary Veins and what is so special about them?
A SELF-TEST FOR REVIEW AND STUDY

Circle the letters corresponding to the correct answers. Note that some questions have more than one correct answer.

1. Suppose you examine a suspect that you know is under the combined influence of Demerol and Thorazine. Which of the following would you not expect to find in that suspect? (Circle all that you wouldn't expect to see.)
   A. Tachycardia is present
   B. Horizontal Gaze Nystagmus is present
   C. Hypotension is present
   D. Mydriasis is present
   E. Lack of Convergence is present

2. The Autonomic Nervous System has sympathetic nerves and nerves.
   A. parasympathetic
   B. metasympathetic
   C. postsympathetic
   D. mesosympathetic
   E. pilosympathetic

3. Suppose you examine a suspect that you know is under the combined influence of Ketamine and Methamphetamine, and you observe that he or she exhibits Horizontal Gaze Nystagmus. This is an example of ....
   A. A Synergistic Effect
   B. An Antagonistic Effect
   C. The Null Effect
   D. An Overlapping Effect
   E. An Additive Effect

4. The technical term meaning "constricted pupils" is ....
   A. Mydriasis
   B. Occulosis
   C. Miosis
   D. Bruxism
   E. Ptosis
5. **Chloral Hydrate** is an example of ....

A. a Non-Barbiturate  
B. an Anti-Psychotic Tranquilizer  
C. an Anti-Depressant  
D. a Barbiturate  
E. an Anti-Anxiety Tranquilizer

6. **Numorphan** is an example of ....

A. a Synthetic Opiate  
B. an Analog of Phencyclidine  
C. a Natural Alkaloid of Opium  
D. an Opium Derivative  
E. a non-Amphetamine-based Stimulant

7. Which of the following ordinarily **will** cause Horizontal Gaze Nystagmus? (Circle all that usually cause nystagmus.)

A. Methamphetamine  
B. Valium  
C. The combination of Cocaine and Xanax  
D. The combination of Cannabis and LSD  
E. The combination of Heroin and Dilaudid

8. **Ritalin** is an example of ....

A. a CNS Stimulant  
B. a Narcotic Analgesic  
C. a Hallucinogen  
D. a CNS Depressant  
E. an Analog of Phencyclidine

9. Suppose you examine a suspect that you **know** is under the combined influence of Heroin and PCP, and you observe that he or she exhibits **miosis**. This is most likely due to ....

A. The "Downside" of Heroin  
B. An Overlapping Effect between the two drugs  
C. An Antagonistic Effect between the two drugs  
D. An Additive Effect between the two drugs  
E. The "Downside" of PCP
10. Which of the following usually will be true in a subject who is under the influence of a Hallucinogen? (Circle all that usually will be true.)

A. Pupils will be constricted  
B. Body temperature will be elevated  
C. Eyes will be unable to converge  
D. Blood pressure will be elevated  
E. Horizontal Gaze Nystagmus will be present

11. Which of the following is not classified as a Hallucinogen? (Circle all that are not Hallucinogens.)

A. ETOH  
B. DOM  
C. MDMA  
D. 2CB  
E. THC

12. Which of the following ordinarily will leave body temperature within the DRE average range? (Circle all that usually don't affect body temperature.)

A. CNS Stimulants  
B. Dissociative Anesthetics  
C. Cannabis  
D. CNS Depressants  
E. All of the above usually do affect body temperature

13. Suppose you examine a suspect that you know is under the combined influence of Percodan and Cannabis, and you find that the suspect's pulse rate is 74 bpm. This is most likely due to ....

A. An Additive Effect between the two drugs  
B. The "Downside" of Cannabis  
C. An Overlapping Effect between the two drugs  
D. An Antagonistic Effect between the two drugs  
E. The "Downside" of Percodan

14. How many distinct, validated clues have been established for the Modified Romberg Balance test?

A. Eight  
B. Six  
C. Four  
D. Three  
E. There are no validated clues for that test.
15. A person under the combined influence of Ritalin and LSD usually will have above normal blood pressure. This is an example of ....

A. An Overlapping Effect  
B. A Synergistic Effect  
C. The Null Effect  
D. An Additive Effect  
E. An Antagonistic Effect

16. The gap between two nerve cells is called the ....

A. Vesicle  
B. Neuron  
C. Synapse  
D. Dendrite  
E. Axon

17. "Ptosis" most nearly means ....

A. Dilated pupils  
B. Grinding the teeth  
C. Constricted pupils  
D. Droopy eyelids  
E. Goose bumps

18. How many distinct, validated clues have been established for the Walk and Turn test?

A. Eight  
B. Six  
C. Four  
D. Three  
E. There are no validated clues for that test.

19. Which of the following are not subcategories of Inhalants? (Circle all that are not proper names for Inhalant Subcategories.)

A. Fluorocarbons  
B. Anesthetic Gases  
C. Aerosols  
D. Volatile Solvents  
E. Propellants
20. Phencyclidine is best described as ....
   A. parasympathomimetic
   B. an anti-depressant
   C. a cellular stimulant
   D. psychotrophic
   E. a dissociative anesthetic

21. Which of the following usually will not cause the pupils to dilate? (Circle all that usually do not cause dilation.)
   A. MDMA
   B. Methaqualone
   C. Desoxyn
   D. Peyote
   E. Ketamine

22. Which subcategory or subcategories of Inhalants usually cause blood pressure to be depressed? (Circle all that usually cause a depressed pressure.)
   A. Anesthetic Gases
   B. Propellants
   C. Volatile Solvents
   D. Aerosols
   E. Fluorocarbons

23. Which of the following are Natural Alkaloids of opium? (Circle all that are Natural Alkaloids.)
   A. Lortab
   B. Dilaudid
   C. Codeine
   D. Thebaine
   E. Hycodan

24. "Crank" is a street name for ....
   A. Heroin
   B. Cocaine
   C. PCP
   D. Methamphetamine
   E. LSD
25. Which of the following are **not validated clues** for the One Leg Stand test? (Circle all that aren't validated clues.)

A. Hopping  
B. Raising the arms  
C. Putting the foot down  
D. Failing to count out loud  
E. Swaying

26. Which of the following would be considered **sympathomimetic** drugs? (Circle all that are sympathomimetic.)

A. MDMA  
B. Dexedrine  
C. Xanax  
D. Oxycontin  
E. Desoxyn

27. Suppose you examine a suspect, and you observe all of the following: Horizontal Gaze Nystagmus is present, with an onset of approximately 30 degrees; BAC is 0.00; eyes are unable to converge; pupil size is 5.5 mm in near-total darkness and 3.5 mm in direct light; pupil reaction to light is within normal; pulse rate is 100 bpm; blood pressure is 148/96; body temperature is 99.8 degrees. In your opinion, this suspect is under the influence of ....

A. a combination of a CNS Depressant and a CNS Stimulant  
B. a CNS Depressant alone  
C. a Dissociative Anesthetic alone  
D. a combination of a Dissociative Anesthetic and a CNS Stimulant  
E. a combination of a CNS Depressant and Cannabis

28. The only artery that carries **de-oxygenated** blood is the artery.

A. Carotid  
B. Brachial  
C. Pulmonary  
D. Radial  
E. Coronal
29. Suppose a subject is under the influence of Hycodan and nothing else. Indicate whether each of the following will be true or false:

A. T F  Horizontal Gaze Nystagmus will not be present
B. T F  Pupils will be constricted
C. T F  Bradycardia will be present
D. T F  Eyes will be able to converge
E. T F  Hypotension will be present

30. "Bruxism" most nearly means ....

A. Dilated pupils
B. Grinding the teeth
C. Constricted pupils
D. Droopy eyelids
E. Goose bumps

31. Suppose a suspect is under the influence of a combination of Marijuana and Cocaine, but nothing else. Indicate whether each of the following will be true or false:

A. T F  Pulse rate will be elevated
B. T F  Pupils will be dilated
C. T F  Horizontal Gaze Nystagmus will be present
D. T F  Eyes will be able to converge
E. T F  Blood pressure will be elevated

32. How many distinct, validated clues have been established for the Finger-to-Nose test?

A. Eight
B. Six
C. Four
D. Three
E. There are no validated clues for this test.

33. The drug is an example of an Anti-Anxiety Tranquilizer. (Circle all that are Anti-Anxiety Tranquilizers.)

A. Librium
B. Valium
C. Amobarbital
D. Chloral Hydrate
E. Xanax
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ANSWER KEY FOR THE SELF-TEST

1. Correct answers are A and D.
   Demerol is a Narcotic Analgesic, Thorazine is a CNS Depressant. The combination should not produce elevated heart rate (Tachycardia) nor dilated pupils (Mydriasis). But Horizontal Gaze Nystagmus and Lack of Convergence should be present, due to the Depressant, Thorazine. And, lowered blood pressure (Hypotension) should be present as an Additive Effect of both drugs.

2. Correct answer is A, parasympathetic.

3. Correct answer is D, Overlapping.
   Ketamine is an Analog of PCP, a drug that usually does cause Horizontal Gaze Nystagmus. Methamphetamine is a CNS Stimulant, a type of drug that doesn't affect nystagmus (Dissociative Anesthetic). This is a case of action plus no action equals action, i.e., an Overlapping Effect.

4. Correct answer is C, Miosis.

5. Correct answer is A, Non-Barbiturate.

6. Correct answer is D, Opiate Derivative.

7. Correct answers are B and C.
   Valium is a CNS Depressant, which of course causes nystagmus. The combination of Cocaine and Xanax gives us a Stimulant and a Depressant (Xanax), which causes Nystagmus via an Overlapping Effect. None of the other drugs mentioned cause Nystagmus: Methamphetamine is a Stimulant; LSD is a Hallucinogen; Heroin and Dilaudid are Narcotics; Cannabis, of course, is its own category.

8. Correct answer is A, CNS Stimulant.

9. Correct answer is B, Overlapping.
   Heroin, a Narcotic, causes constriction of the pupils (Miosis); PCP does not affect pupil size. This is another case of action plus no action equals action.

10. Correct answers are B and D.
    Hallucinogens are sympathomimetic drugs, and therefore usually elevate the vital signs. But they have no effect on either Nystagmus or Lack of Convergence. And, instead of constricting the pupils, Hallucinogens usually cause pupils to dilate.
11. Correct answers are A and E. **ETOH** is the chemical name for Ethyl Alcohol, the common beverage form of alcohol that remains the most commonly-abused drug. **THC** is the primary active ingredient in Cannabis. But "MDMA" (also known as "Ecstasy") and "DOM" (also known as "STP") and 2CB are Hallucinogens.

12. Correct answers are C and D, **Cannabis and Depressants**.

13. Correct answer is D, **Antagonistic**. A pulse rate of 74 bpm is within the normal range. Percodan, a Narcotic Analgesic, usually lowers the pulse, while Cannabis usually elevates the pulse. The Antagonistic Effect of the two drugs has put this suspect's pulse into a precarious, and probably temporary, state of balance.

14. Correct answer is E, **no validated clues**. It is important to understand that, when we say there are no validated clues for Modified Romberg Balance Test, that does **not mean** that the test is invalid. It simply means that we do not have the research data to attest that specific clues on that test are statistically reliable indicators of impairment. Those kinds of research data, at the present time, are available only for Horizontal Gaze Nystagmus, Walk and Turn and One Leg Stand.

15. Correct answer is D, **Additive**. Ritalin (a Stimulant) and LSD (a Hallucinogen) both usually elevate blood pressure.

16. Correct answer is C, **Synapse**.

17. Correct answer is D, **Droopy Eyelids**.

18. Correct answer is A, **Eight**. Of the eight **validated** clues for Walk and Turn, two may be observed during the Instructions Stage of the test. They are **can't keep balance** (which means the suspect breaks away from the heel-to-toe stance) and **starts too soon**. The other six clues pertain to the Walking Stage of the test. They include:

- misses heel-to-toe
- uses arms to balance
- steps off line
- stops walking
- turns improperly
- takes the wrong number of steps

Although these eight are the only **validated** clues for Walk and Turn, they aren't the only things that might be observed that could serve as evidence of impairment. All of your observations of the suspect are important.
19. Correct answers are A and E, **Fluorocarbons and Propellants**.

The only proper names for subcategories of Inhalants are Volatile Solvents, Aerosols and Anesthetic Gases.

20. Correct answer is E, dissociative anesthetic.

21. Correct answer is E, **Ketamine**.

Ketamine is an analog of PCP, a drug that doesn't affect pupil size. MDMA and Peyote are Hallucinogens, and Desoxyn is a CNS Stimulant; all of those dilate pupils. Methaqualone is a very special CNS Depressant; unlike almost all other Depressants, Methaqualone *does* affect pupil size (by dilating the pupils).

22. Correct answer is A, **Anesthetic Gases**.

Volatile Solvents and Aerosols usually produce an elevated blood pressure. "Fluorocarbons" and "Propellants" are, of course, not proper names for subcategories of Inhalants.

23. Correct answers are C and D, **Codeine and Thebaine**.

Lortab, Dilaudid and Hycoand are all *opium derivatives*. Dilaudid derives from Morphine, and Hycoand and Lortab from Codeine.

24. Correct answer is D, **Methamphetamine**.

25. Correct answer is D, **Failing to Count Out Loud**.

Hopping, Raising the Arms, Putting the Foot Down and Swaying are the four (and only four) *validated* clues of impairment for One Leg Stand.

26. Correct answers are A, B and E: **MDMA, Dexedrine and Desoxyn**.

Dexedrine and Desoxyn are members of the Amphetamine family of CNS Stimulants. MDMA is a "Psychedelic Amphetamine" belonging to the Hallucinogens. CNS Stimulants and Hallucinogens are the two categories that make up the *sympathomimetic* drugs. That means they simulate the responses that the body makes to messages conveyed along the sympathetic nerves, i.e., elevated vital signs, dilated pupils, etc. Three other categories, namely the Inhalants, Phencyclidine and Cannabis have *some* sympathomimetic characteristics, but they are not considered to be fully sympathomimetic, and not to the degree of the CNS Stimulants and Hallucinogens. Xanax and Oxycontin aren't even close to being sympathomimetic. Xanax (a Depressant) and Oxycontin (a Narcotic) are better described as wholly or partially *parasympathomimetic*.

27. Correct answer is C, **a Dissociative Anesthetic**.

Dissociative Anesthetics, by themselves, can account for *all* of the observations listed. Dissociative Anesthetics cause Nystagmus, and Lack of Convergence; they do not affect pupil size, so the pupils remain within the normal range; they do not affect the reaction of the pupils to light; they usually elevate all three vital signs.
A Depressant, by itself, could not account for the elevated vitals, and usually would slow the pupils' reaction to light.

If we had a combination of a Depressant and a Stimulant, we'd expect to see the pupils dilated beyond the normal range (due to an Overlapping Effect), and we'd expect to see the reaction of the pupils slowed (due to an Additive Effect). Also, although it is possible that the vital signs could all be elevated with a combination of Depressant and Stimulant, we'd probably expect to see some "moderation" of the vitals due to an Antagonistic Effect.

If we had a combination of a Dissociative Anesthetic and a Stimulant, we could expect to see pupil dilation and some slowing of the reaction to light, due to Overlapping Effects.

If we had a combination of a Dissociative Anesthetic and a Stimulant, we could expect to see an elevated body temperature, since both of those drugs elevate temperature.

28. Correct answer is C, Pulmonary.

29. Correct answers are:
   (A) True: no nystagmus will be present
   (B) True: we will see miosis, or constricted pupils
   (C) True: we will find a slow pulse, or Bradycardia
   (D) True: we won't see a Lack of Convergence, so the eyes will be able to converge
   (E) True: we will find a lowered blood pressure, or Hypotension
   Hycodan is a Narcotic Analgesic, and these observations will be consistent with impairment by Narcotics.

30. Correct answer is B, Grinding the Teeth

31. Correct answers are:
   (A) True: An Additive Effect will elevate the pulse for this combo
   (B) True: pupils will dilate due to an Overlapping or Additive Effect
   (C) False: neither drug causes Nystagmus, so the Null Effect will also cause no nystagmus
   (D) False: Marijuana causes Lack of Convergence, so the Overlapping Effect means the eyes won't converge
   (E) True: An Additive Effect will elevate the blood pressure

32. Correct answer is E, no validated clues

33. Correct answers are A, B and E: Librium, Valium and Xanax
Participant Manual

Drug Recognition Expert Course

Session 29
Classifying a Suspect (Role Play)
[This page is intentionally left blank]
Upon successfully completing this session the student will be able to:

- Conduct a complete drug influence evaluation using the systematic and standardized 12-step process.
- Compile a complete, clear and accurate report documenting the results of a drug influence evaluation using the 13-step component narrative report format.

**Learning Activities**

**Content Segments**

- **A. Scenarios: Simulated Examinations**
  - Interviewing Practice
- **B. Report Preparation Practice**
  - Note-taking Practice
- **C. Report Review and Critique**
  - Small Group Work Session
  - Instructor-Led Presentations
  - Participant-Led Presentations
  - Participant-Led Critiques

---

**A. Scenarios: Simulated Examinations**

**Team Assignments**

The total number of student teams should not be more than the number of “role players” participating in this session. Otherwise, one or more teams would be unoccupied during major portions of this segment.
**Procedures**

Each team will examine as many as possible of the “role players”, until the time scheduled for this segment elapses.

Each examination will be carried out fully: nothing will be omitted except for the breath alcohol test.

At certain points in the examination, the “role player” will inform the team what to record. Example: the “role players” will instruct the teams concerning the evidence to be recorded from the Horizontal Gaze Nystagmus test.

All data will be recorded on the standard Drug Influence Evaluation Form.

- Some “role players” will be simulating the signs and symptoms of exactly one category of drugs. Clarification: “Role player Alpha” might be simulating a person who is under the influence of a CNS Stimulant only.

- “Role player Delta” might be simulating a person under the influence of an Inhalant only.

Some “role players” may be simulating the signs and symptoms of two or more categories in combination. “Role player Bravo” might be simulating someone who is under the influence of both PCP and Marijuana.

It is possible that one or more “role players” may be simulating persons who are not under the influence of any drugs.

At the completion of each examination, the team will discuss the evidence obtained and reach a consensus concerning the category or categories of drugs present.

Subsequently, each team will be assigned the responsibility of preparing and presenting a complete narrative report on one “role player.”

All students will participate in critiquing the reports.
Drug Evaluation and Classification Practice

Practice will continue for approximately 2 hours, or until each team has completed the evaluation of at least three “role players”

B. Report Preparation Practice

Team Assignments

Group Writing Exercise
C. Report Review and Critique

*Report Presentation*

- Each team should appoint a speaker to read its report. The speaker should explain exactly what led the team to its conclusion concerning the category or categories of drugs.

---

*Report Critique*

---
## DRUG INFLUENCE EVALUATION

**Evaluator**

**Recorder/Witness**

**Arrestee's Name (Last, First, Middle):** ALPHA

**Date Examined/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Urine

**Date of Birth/Sex/Race**

**Arresting Officer (Name, ID#)**

**On Scene/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Blood

**Date:**

**Time:**

**Location:**

**Date Examined/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Blood

**Date:**

**Time:**

**Location:**

**Date Examined/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Blood

**Date:**

**Time:**

**Location:**

**Date Examined/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Blood

**Date:**

**Time:**

**Location:**

**Date Examined/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Blood

**Date:**

**Time:**

**Location:**

**Date Examined/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Blood

**Date:**

**Time:**

**Location:**

**Date Examined/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Blood

**Date:**

**Time:**

**Location:**

**Date Examined/Time/Location**

**Breath Results:** Test Refused

**Chemical Test:** Blood

**Date:**

**Time:**

**Location:**
[This page is intentionally left blank]
**DRUG INFLUENCE EVALUATION**

<table>
<thead>
<tr>
<th>Officer's Signature:</th>
<th>DRE #</th>
<th>Rolling Log #</th>
<th>Case #</th>
<th>Session XXIX - 3</th>
</tr>
</thead>
</table>

**Arrestee's Name (Last, First, Middle):** Eva0111ator

**Date Examined:** 0000

**Recorder (Name, ID):**

**Arresting Officer (Name, ID):**

<table>
<thead>
<tr>
<th>Miranda Warning Given</th>
<th>What have you eaten today?</th>
<th>When?</th>
<th>What have you been drinking?</th>
<th>How much?</th>
<th>Time of last drink?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>&quot;Today&quot;</td>
<td>&quot;No&quot;</td>
<td>Unusual</td>
<td>D</td>
<td>N/A</td>
</tr>
</tbody>
</table>

**Comments:**

- Corrective Lenses: None
- Glasses: Yes
- Contacts: No

**Pupil Size:**

- Right Eye: Equal
- Left Eye: Equal

**Convergence:**

- Right eye: Present
- Left eye: Present

**Dazed, Confused**

**Speech:**

- Drowsy

**Face:**

- Sweaty

**Coordination:**

- Slow, rigid movements

**Internal Clock:**

- 52 seconds

**Modified Romberg Balance Test:**

- Walk and Turn Test: 2' 2" 3' 3"

- Subject stopped after 9 steps. Did not continue as directed. Rigid movements.

- Left Count: 38
  - One Leg Stand: 24

**Finger to Nose**

- Draw lines to spots touched

**Breath Results:**

- Results: Refused

**HGN:**

- Lack of Smooth Pursuit: Present
- Maximum Deviation: Present

**Vertical Nystagmus:**

- Left Eye: Present
- Right Eye: Present

**Convergence:**

- Right eye: Present
- Left eye: Present

**Reaction to Light:**

- Able to follow stimulus

**Room Light:**

- Left Eye: 4.5
- Right Eye: 4.5

**Darkness:**

- Left Eye: 6.5
- Right Eye: 6.5

**Direct:**

- Left Eye: 3.5
- Right Eye: 3.5

**Blood pressure:**

- 122/102

**Temperature:**

- 100°

**Blood Alcohol:**

- 0.00

**Type of Footwear:**

- N/A

**Nasal area:**

- Clear

**Oral cavity:**

- Clear

**Reaction to Light:**

- Normal

**NIA:**

- 0

**DRE#:**

- 0

**Location:**

- Charlie

**Comments:**

- Nothing observed.

**Where were the drugs used? (Location):**

- N/A

**What drugs or medications have you been using?**

- "Drugs? ... Nothing man"

**How much?**

- N/A

**Time of use?**

- N/A

**Where were the drugs used? (Location):**

- N/A

**Date / Time of arrest:**

- Time DRE was notified: N/A

**Evaluation start time:**

- Evaluation completion time: N/A

**Predicted Station:**

- N/A

**Officer’s Signature:**

- N/A

**Opinion of Evaluator:**

- Not Impaired
- Medical
- Alcohol
- Narcotic Analgesic
- Cannabis

**Internal Clock:**

- Estimated as 30 seconds
**DRUG INFLUENCE EVALUATION**

Evaluators: [Name]

**DATETIME EXAMINED**

Date: [Date]

Time: [Time]

Location: [Location]

**MIRANDA WARNING GIVEN**

[Optional: Yes/No]

[Template: "If/when a Miranda warning is given, write the name and ID of the officer who administered it."]

**DATE OF BIRTH**

[Date]

**SEX**

[Male/Female]

**RACE**

[Optional: Asian/Black/Hispanic/Other]

**ARRAYED**

[Optional: Arrested/Unarmed/Disabled]

**DATE AND TIME OF ARREST**

[Date and Time]

**OFFICER'S SIGNATURE**

[Signature]

**OPINION OF EVALUATOR**

[Impaired/Not Impaired/Under the influence of] [Selected Options]

**FINGER TO NOSE**

[Template: "Draw lines to spots touched."]

**PUPIL SIZE**

[Room Light/1.5-2.0 to 5.0-8.5/2.0-4.5]

**LEFT ARM**

[Horizontal Nystagmus/Able to follow stimulus/Normal (Continually rugging face)/Extreme Latent/Reaction to light/Little to None]

**LEFT EYE**

[Response to Light/None/Normal (Continually rugging face)/Extreme Latent/Reaction to light/Little to None]

**BREATH ODOR**

[Optional: None/Normal/Persistent (Continually rugging face)/Disgusting/Reddened/Normal/Persistent (Continually rugging face)/Disgusting]

**MODIFIED ROMBERG BALANCE**

[Template: "Starts too soon/Cannot keep balance/Uses arms to balance/Puts foot down/Counted slowly." ]

**WALK AND TURN TEST**

[Template: "Starts too soon/Uses arms to balance/Puts foot down/Counted slowly." ]

**BLOOD PRESSURE**

[Optional: Normal/High/Low]

**TEMPERATURE**

[Optional: Normal/High/Low]

**MUSCLE TONE**

[Optional: Normal/Flaccid/Rigid]

**COMMENTS**

[Optional: Additional notes on condition or behavior.]

**OFFICER'S SIGNATURE**

[Signature]

**REVIEWED/APPROVED BY DATE**

[Signature/Date]

**OPINION OF EVALUATOR**

[Impaired/Not Impaired/Under the influence of] [Selected Options]
DRUG INFLUENCE EVALUATION

Evaluator

ReCOrder

Witness

Arrestees Name (Last, First, Middle)

GOLF

Date Examined / Time / Location

Breath Results: Test Refused: Results: 0.00

Chemical Test: Instrument #

Miranda Warning Given: Yes

Test or tests refused

Do you take insulin? Yes

Do you have any physical defects? No

Are you diabetic or epileptic? No

Are you under the care of a doctor or dentist? No

What have you eaten today? When? Beef jerky & pepperoni 4 pm

What have you been drinking? Red Bull

Time of last drink? 1 can N/A

Actual

Do you take insulin? Yes

When did you last sleep? How long? 2 days ago About 3 hours

Are you sick or injured? Yes

Are you taking any medication or drugs? Yes

Are you under the care of a doctor or dentist? Yes

Am I under arrest? Excited, Animated

Speak:

Talkative, Rapid

Corrective Lenses: None

Glasses: No

Contacts, if so: No

Hard: Yes

Soft: No

Pupil Size: 5

Unequal (explain)

Room Light

Darkness

Direct

Nasal area:

Redness

Oral cavity:

Clear

PUPIL SIZE

Room Light 2.5 - 5.0

Darkness 5.0 - 8.5

Direct 2.0 - 4.5

N/A

Left Eye

6.5

8.5

5.5

Left Eye

Rebound Dilation:

None

Pupillary Unrest

Reaction to Light:

Slow

RIGHT ARM

LEFT ARM

Nothing observed.

Blood pressure

174 / 102

Temperature

99.8°

Nothing observed.

Officer's Signature:

DRE #

Reviewed/approved by / date:

Opinion of Evaluator:

Medical

CNS Stimulation

Dissociative Anesthetic

Inhalant

Opinion of Evaluator:

Not Impaired

Alcohol

CNS Depressant

Hallucinogen

Narcotic Analgesic

Cannabis

What drugs or medications have you been using? N/A

I'm not answering that.

How much? N/A

Time of use? N/A

Where were the drugs used? (Location)

Date / time of arrest:

Time DRE was notified:

Evaluation start time:

Evaluation completion time:

Precinct/Station:

Reviewed/approved by / date:

Opinion of Evaluator:

Not Impaired

Alcohol

CNS Depressant

Hallucinogen

Narcotic Analgesic

Cannabis

What drugs or medications have you been using? N/A

I'm not answering that.

How much? N/A

Time of use? N/A

Where were the drugs used? (Location)

Date / time of arrest:

Time DRE was notified:

Evaluation start time:

Evaluation completion time:

Precinct/Station:

Reviewed/approved by / date:
DRUG INFLUENCE EVALUATION

Recorder I \,'\Jitness

Date Examined I Time I Location

Time now I Actu al

!

No

No

@ Equal

Pupil Size:

if so

l

"I don't remember"

I

....ill.... I
2. ...111_ I
3. --120 I

Internal clock

•

I

2

Present
lmmed

•

-�

•

!5

••

�1111\

Blood pressure

184

I

112

100

No resoonse

Date f Time of arrest:

'

Raises arms

9.0
9.0

Time DRE was notified:

D Not Impaired
OMedical

Evaluation start tlme:

u.c•

0Alcohol
OCNS Depressant

I

I

Right Count

26

I NIA

Time of use?

0CNS Stimulant
OHaHucinogen

Oral cavity:

Green coating. Raised taste buds

== =�
'Reaction to Light:

Normal
LEFT ARM

�

-===
I

NIA

Evaluation completion time:

Reviewed/approved by I date:

.

Clear

Pupilrary Unrest
O Yes
@No

� ==

How much?

Nasal area:

6.5-7.5
6.5-7.5

�

I NIA l

Type of footwear:
Boots

Direct
2.0-4.5

Darkness
5.0-8.5

'

Nothing observed.

�''�
One Leg Stand

Leg tremors. Reminded to count.

11

9

�

0

D Droopy

2nd Nine

Cannot do test (explain)
NIA

7.0

!RI Normal

./

RIGHT ARM

Officer's Signature:
Opinion of Evaluator:

I

D Unequal

l.: R
0 0 Sways while balancing
./ ./ ./ ./ ./ ./ 0 0 Uses arms to balance
DD Hops
./
./
0 0 Puts foot down
./ ./ ./ ./ ./ ./
1st Nine

Actual steps taken

Rebound Dilation:
DNo
0 Yes

Musc!e tone:
Normal
QFlaccid
0Rigid
Comments:
V\/hat drugs or medications have you been using?

D

Steps off line

Right Eye

Temperature

22

Left eye

./

Misses heel-toe

7.0

Had to be reminded to remove his finger each time.

Eyelids

.f

Stops walking

Left Eye

�p

0 Equal

Left Count

Convergence

Right eye

Room Light
2.5-5.0

p
p�0�

Tracking:

Blindness:

(3;J(;c)

PUPIL SIZE

1

Poor, staggering

Able to follow stimulus
0Yes DNo

Starts too soon

(D I CD 1

NIA

No response

Coordination:

Cannot keep balance

I

Descnt>e Turn
Staqqered to the riqh!

\

5

11 5
• I
��

Rigid movements. Did not count steps.

,, A

'

.. '. ..
.

T

;g - I I\)

-� 'a {o' �

I

0 None D Left D Right

Vertical Nystagmus
0Yes D No
Left Eye
Right Eye

Present
Im med

Time of last drink?

0No

D Yes O No

I

Maximum Deviation

I

Are you under the care of a doctor or dentist?

Flushed

Present

},· l7
1\.I,

D Yes
race:

Present

.

How much

Blood O

Are you diabetic or epileptic?

Eyes: LJ Reddened Conjunctiva
O Normal 0 Bloodshot 0 Watery

'1

Unne D
Chemical Test
Test or tests refused D

"Uh ..... some juice, I think."

Chemical-like

Finger to Nose
(Draw lines to spots touched)

f

!

What have you been drinking?

Lack of Smooth Pursuit

I I
r

estimated as 30 seconds

D

Indifferent

\
I
I IOJl(Olf', (b---Y Ill 1 '<t...

JI

1Krrestmg vmcer Agency:

Breath Odor:

DHarctD Soft

I

Race

Attitude:

Modified Romberg Balance Walk and T'j!{' Test

Circular sway. Eyelid tremors

I

Session XXIX - 8

Arresting Officer (Name, ID#)

Are you sick or injured?
D Yes 0No
response
Do you have any physical defects?
D Yes 0 No

Angle of Onset

3" 3" 3" 3"

Gase#

Test Refused
Instrument#

0.00

What have you eaten today? When?

D Unenual Fe= tain'
Pulse and time
HGN

1.

I

Sex

Breath Results:
Results:

Are you taking any medication or drugs?
No response
Oves D No
Speech:
Slow, Deliberate, Incomplete responses
�None
Corrective Lenses:

D Glasses D Contacts

Date of Birth

When did you last sleep? How long?

Do you take insulin?

O Yes 0

·1

I

D Fatal n Injury O Property

HOTEL
DYes
D No

Miranda Warning Given
Given by:

r-mung Log "ff

O None

Crash:

Arrestees Name \Last, Hrst, ,vnuu,e)

52

I

DRE#

evaluator

�

VVhere were the drugs used? (Location)

I

PrecinctJStatlon:

0Dissociative Anesthetic
ONarcotic Analgesic

Olnhalant
ocannabis


[This page is intentionally left blank]
### DRUG INFLUENCE EVALUATION

**Evaluator**: [Evaluator's Name]

**Record/Witness**: [Witness's Name]

**Arrestee's Name**: [Arrestee's Name]

**Date Examined**: [Date]

**Date of Birth**: [Date]

**Race**: [Race]

**Sex**: [Sex]

**Arresting Officer**: [Arresting Officer's Name]

**Arresting Officer Agency**: [Agency]

**Session**: XXIX - 9

---

**Miranda Warning Given**

- **Yes**
- **No**

**Time**: [Time]

**HGN**: [Present/Not Present]

**Convergence**: [Present/Not Present]

**Right Eye**: [Present/Not Present]

**Left Eye**: [Present/Not Present]

**Coordination**: [Present/Not Present]

**Corrective Lenses**: [Yes/No]

**Glasses**: [Yes/No]

**Contacts**: [Yes/No]

**Hard**: [Present/Not Present]

**Soft**: [Present/Not Present]

**Breath Odor**: [Present/Not Present]

**Bloodshot**: [Present/Not Present]

**Watery**: [Present/Not Present]

**Nystagmus**: [Present/Not Present]

**Droopy**: [Present/Not Present]

---

**Blood Pressure**: [148/98]

**Temperature**: [98.8]

---

**Pupil Size**: [Room Light/ Darkness/ Direct]

- **Left Eye**: [2.0 - 4.5/ 5.0 - 8.0/ 7.0 - 11.0]
- **Right Eye**: [2.5 - 5.0/ 5.0 - 8.0/ 4.0 - 6.0]

---

**Reaction to Light**: [Slow]

**Pupillary Responsiveness**: [Present/Not Present]

**Bloodshot**: [Present/Not Present]

**Red**: [Present/Not Present]

**Nasal Area**: [Redness, Running nose]

**Ocular Area**: [Redness, Running nose]

**Oral Cavity**: [Redness, Running nose]

---

**PULPIL SIZE**: [Room Light/ Darkness/ Direct]

- **Left Eye**: [2.5 - 5.0/ 5.0 - 8.5/ 4.0 - 4.5]
- **Right Eye**: [5.0 - 7.0/ 4.0 - 4.5]

---

**NIA**

---

**Type of Footwear**: [Boots]

**Internal Clock**

- **30 seconds**

---

**Date of Arrest**: [Date]

**Precinct/Station**: [Station]

**Evaluation Completion Time**: [Time]

---

**Opinion of Evaluator**: [Impaired/ Not Impaired]

**Drug Influence**: [CNS Stimulant/ Dissociative Anesthetic/ HALLUCINOGEN/ Narcotic Analgesic/ Inhaled]

---

**Finger to Nose**: [Staggered, Lost balance]

**Step Test**: [Misses heel-toe, Starts too soon]

---

**Finger to Nose**: [Staggered, Lost balance]

**PUPIL SIZE**: [Room Light/ Darkness/ Direct]

- **Left Eye**: [2.0 - 4.5/ 5.0 - 8.0/ 4.0 - 4.5]
- **Right Eye**: [5.0 - 7.0/ 4.0 - 4.5]

---

**Blood Pressure**: [148/98]

**Temperature**: [98.8]

**Muscle Tone**: [Normal/ Paralytic/ Rigor]

---

**Date Time of Arrester**: [Date/ Time]

**Time DRE Notified**: [Time]

**Evaluation Start Time**: [Time]

---

**Opinion of Evaluator**: [Impaired/ Not Impaired]

**Drug Influence**: [CNS Stimulant/ Dissociative Anesthetic/ HALLUCINOGEN/ Narcotic Analgesic/ Inhaled]

---

**Finger to Nose**: [Staggered, Lost balance]

**HGN**: [Present/Not Present]

**Coordination**: [Present/Not Present]

**Corrective Lenses**: [Present/Not Present]

**Glasses**: [Present/Not Present]

**Contacts**: [Present/Not Present]

**Hard**: [Present/Not Present]

**Soft**: [Present/Not Present]

**Breath Odor**: [Present/Not Present]

**Bloodshot**: [Present/Not Present]

**Watery**: [Present/Not Present]

**Nystagmus**: [Present/Not Present]

**Droopy**: [Present/Not Present]
**Drug Influence Evaluation**

### Evaluator
- **Date Examined / Time:** [Blank]
- **Location:** [Blank]
- **Date of Birth:** [Blank]
- **Race:** [Blank]

### Breath Results
- **Test Results:** 0.06

### Chemical Test
- **Blood:** [Check]
- **Urine:** [Check]
- **None:** [Check]
- **Test refused:** [Check]

### Chemical Test Results
- **Results:** [Blank]

### Physical Examination
- **Symptoms:** [Blank]
- **Medications:** [Blank]
- **Past Medical History:** [Blank]
- **Allergies:** [Blank]

### Coordination
- **Unsteady:** [Check]
- **Withdrawn:** [Check]
- **Cooperative:** [Check]

### Speech
- **Low, Mumbling:** [Check]
- **Articulation:** [Blank]
- **Breath Odor:** [Blank]

### Blood Pressure and Temperature
- **Blood Pressure:** [Blank]
- **Temperature:** [Blank]

### Pupil Size
- **Left Eye:** [Blank]
- **Right Eye:** [Blank]

### Pupillary Unreactive to Light
- **Left:** [Check]
- **Right:** [Check]

### Pupil Reaction to Light
- **Left:** [Check]
- **Right:** [Check]

### DRE Balance Test
- **Internal Clock:** 36 seconds
- **Modified Romberg Balance:** [Blank]
- **Lack of Smooth Pursuit:** [Check]
- **Maximum Deviation:** [Check]

### Finger to Nose
- **Draw lines to spots touched:** [Blank]

### Type of Footwear
- **Lace-up shoes:** [Check]

### Opinions of Evaluator
- **Not Impaired:** [Check]
- **Alcohol:** [Check]
- **CNS Stimulant:** [Check]
- **Dissociative Anesthetic:** [Check]
- **Inhalant:** [Check]
- **Medical:** [Check]
- **CNS Depressant:** [Check]
- **Hallucinogen:** [Check]
- **Narcotic Analgesic:** [Check]
- **Cannabis:** [Check]

### Drug Influences
- **Finger to Nose:** [Blank]
- **Glasses:** [Blank]
- **Contacts, if so:** [Blank]
- **Hard:** [Blank]
- **Soft:** [Blank]
- **Flaccid:** [Blank]
- **Rigid:** [Blank]

### Comments
- **Nothing observed:** [Blank]

### Date and Time
- **Date:** [Blank]
- **Time of arrest:** [Blank]
- **Time DRE notified:** [Blank]

### Officer's Signature
- **Reviewed/approved by / date:** [Blank]
Evaluator

Arrestee's Name (Last, First, Middle) KILO

Date Examined

Recorder

Officer's Signature:

Given by:

Miranda Warning Given

Comments:

Modified Romberg Balance

Given by: No

Are you taking any medication or drugs?

Attitude:

Are you diabetic or epileptic?

Coordination:

Speech:

Blood pressure 108 / 64

Temperature 97.2°

Muscle tone: Normal

Type of footwear: Slip-on shoes

Finger to Nose (Draw lines to spots touched)

PUPIL SIZE Room Light 2.5 - 5.0 Darkness 5.0 - 8.5 Direct 2.0 - 4.5 Nasal area:

Left Eye

Right Eye

Reaction to Light:

N/A

L

R

Sways while balancing

Uses arms to balance

Hops

Puts foot down

Nearly fell. Stopped for safety reasons.

Internal clock estimated as 30 seconds

Describe Turn

Cannot do test (explain)

Type of footwear: Slip-on shoes

Right Count

One Leg Stand

1st Nine

2nd Nine

Left Eye

Right Eye

Cannot keep balance

SLO MOSS

Muscle tone: Normal

N/A

N/A

Time of last drink?

Time of use?

Where were the drugs used? (Location)

Date / Time of arrest:

Time DRE was notified:

Evalution start time:

Evaluation completion time:

Officer's Signature:

Reviewed/approved by / date:

Opinion of Evaluator: Not Impaired

Alcohol

CNS Stimulant

Dissociative Anesthetic

Inhalant

Medical

CNS Depressant

Hallucinogen

Narcotic Analgesic

Cannabis
DRUG INFLUENCE EVALUATION

Evaluator: LIMA

Date Examined / Time / Location: I

Miranda Warning Given: Yes

Given by: LIMA

Arrestee's Name (Last, First, Middle): ORE#

Date of Birth: o

Arresting Officer (Name, ID#): LIMA

Time / Location:

Blood Results:

Chemical Test: Blood

Test Refused: No

Test or tests refused: No

What have you eaten today? When?: Pizza 7 pm

What have you been drinking?: Beer

How much?: Two

Time of last drink?: 3 hours ago

When did you last sleep? How long?: Yesterday 3 hours

Are you diabetic or epileptic?: No

Do you take insulin?: No

Do you have any physical defects?: No

Are you taking any medication or drugs?: Yes

Attitude: Anxious, Restless

Speech: Normal

Eyes:

Reddened Conjunctiva: No

Corrective Lenses: Glasses

D Contacts if so

0 Hard 0 Soft 0 Normal 0 Bloodshot 0 Watery

Pupil Size:

Equal

Unequal (explain): None

Face:

Nasal area:

Redness. No nasal hair.

Oral cavity:

Clear

PUPIL SIZE

Room Light

2.5 - 5.0

5.0 - 8.5

Direct

2.0 - 4.5

Left Eye

7.5

9.0

6.0

Pupil Reaction to Light:

Slight

Pupillary Line:

Yes

No

Reaction to Light:

Sluggish

Type of footwear:

Slip-on boots

Nail area:

Redness. No nasal hair.

Oral cavity:

Clear

PUPIL SIZE

Room Light

2.5 - 5.0

5.0 - 8.5

Direct

2.0 - 4.5

Left Eye

Right Eye

7.5

9.0

6.0

Blood pressure

170 / 100

Temperature

99.8 °

Muscle tone:

Normal

Flaccid

Rigid

Comments:

What drugs or medications have you been using? (Location)

Nothing. Just a couple of beers man.

How much?: N/A

Time of use?: N/A

Where were the drugs used?: N/A

Date / Time of arrest:

Time DRE was notified: I

Evaluation start time: I

Evaluation completion time: I

Precinct/Station:

Officer's Signature:

DRE #:

Reviewed / Approved by / date:

Opinion of Evaluator:

Not Impaired

Alcohol

Medical

CNS Stimulant

Dissociative Anesthetic

Hallucinogen

Narcotic Analgesic

Cannabis
Participant Manual

Drug Recognition Expert Course

Session 30

Transition to the Certification Phase of Training
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Learning Objectives

- Demonstrate mastery of the knowledge and skills the course was intended to help develop
- Summarize the key topics covered
- Offer comments and suggestions for improving the course
- Receive their assignments for Field Certification Training

Upon successfully completing this session the participant will be able to:

- Demonstrate mastery of the knowledge and skills the course was intended to help develop.
- Summarize the key topics covered.
- Offer comments and suggestions for improving the course.
- Receive assignments for Field Certification Training.
- Understand the steps involved in the DRE certification process.

CONTENT SEGMENTS ..................................................................................... LEARNING ACTIVITIES

A. Summary ......................................................................................Participant-Led Presentations
B. Post Test ........................................................................................Participants’ Anonymous Critique of Course
C. Session Wrap-Up ................................................................................. Knowledge Examination
D. Certification Process, Training Assignments and Schedule ..........Instructor-Led Presentation
E. Closing Remarks
A. Summary

The Seven Categories of Drugs

- CNS Depressants
- CNS Stimulants
- Hallucinogens
- Dissociative Anesthetics
- Narcotic Analgesics
- Inhalants
- Cannabis
The Drug Evaluation and Classification Procedure

- Breath Alcohol Test
- Interview of Arresting Officer
- Preliminary Examination
- Examinations of Eyes
- Divided Attention Tests
- Vital Signs Examinations
- Check for Muscle Tone
- Inspection for Injection Sites
- Statements and Observations
- Opinion of the Evaluator
- Toxicological Examination
Major Signs and Symptoms

- CNS Depressants
- CNS Stimulants
- Hallucinogens
- Dissociative Anesthetics
- Narcotic Analgesics
- Inhalants
- Cannabis
B. Post-Test

Knowledge Examination

C. Session Wrap-Up

Critique
The Three-Phases of Training for the DEC Program

Certification involves three-phase training process:
1. Phase I- Two-day (16-hour) Pre-school
2. Phase II- Seven-day (56-hour) DRE School
3. Phase III- Field Certifications (usually within 60 to 90 days, but not longer than six months following the completion of the classroom training)

D. Certification Training Assignments and Schedule

1. Phase I – Pre-School
2. Phase II – DRE School
3. Phase III – Field Certifications
Field Evaluations Requirements

- 12 evaluations (minimum)
- 9 toxicology samples collected
- 7 positive (confirmed) toxicology samples from the lab
- 6 of the 12 evaluations conducted - YOU must be the evaluator
- 3 of the 7 drug categories must be encountered
- Evaluations must be witnessed and supervised by a DRE Instructor

IACP Standard 1.10 requires that the candidate DRE satisfactorily complete a minimum of twelve (12) evaluations, identifying subjects under the influence of at least three of the drug categories. All three must be supported by toxicology.

The candidate DRE must also act as the evaluator for at least six evaluations.

All evaluations, either administered or observed must be documented on the candidate’s rolling log.

Candidate DREs need to have toxicology samples from at least nine (9) subjects evaluated during the certification process.

The candidate DRE cannot be certified unless the opinion concerning the drug category(s) is supported by toxicology 75 percent of the time or in at least seven (7) of the nine samples submitted for certification.

Field certification evaluations must be observed and supervised by a DRE instructor to count towards minimum certification requirements. The evaluation must be observed in its entirety and the instructor who observed the entire evaluation must sign-off on the observed evaluation.
Field Certifications

What’s needed for the Field Certification nights?

- DRE kits
- Certification Progress Log
- Your Participant Manual
- Your Rolling Log
- A “prepared mind”
• Standard 1.12... Prior to concluding field certification training, the candidate shall satisfactorily complete an approved “Certification Knowledge Examination”

• ...The examination shall only be administered after the candidate has completed not less than three drug evaluations

**Final Certification Knowledge Examination**

• Prior to concluding the certification process, the candidate DRE must satisfactorily complete an IACP approved Final Certification Knowledge Examination.

• The Final Certification Knowledge Examination is a multi-part comprehensive examination where the participant cannot make significant errors or omissions.

• Examination consists of five parts which tests the candidate DRE’s knowledge of the drug symptomatology matrix, drug effects, drug combinations, and report writing skills.
After each component required for certification is completed, a DRE Instructor must sign off on the DRE candidate’s log.

The candidate DRE must be recommended for certification by two DRE instructors.
**DRE Certification**

DRE certification is for a period of two years. DRE’s shall be required to renew their certificate of continuing proficiency every two years. Once certified, DREs shall be required to renew their certificates of continuing proficiency every two years.

Continuing proficiency requires:

- Performing a minimum of four (4) acceptable drug evaluations since the last date of certification;
- Completing a minimum of eight (8) hours of approved re-certification training; and
- Presenting an updated Curriculum Vitae and Rolling Log to the appropriate coordinator for review.
QUESTIONS?

E. Closing Remarks
<table>
<thead>
<tr>
<th>CONTROL NUMBER</th>
<th>SUSPECT’S NAME</th>
<th>WITNESS</th>
<th>DATE</th>
<th>OPINION OF DRE</th>
<th>TOXICOLOGICAL RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Drugs and Human Performance Fact Sheets

A panel of international experts on drug-impaired driving met in Seattle during August 2000 to review developments in the field of drugs and human performance over the last 10 years; to identify the specific effects that both illicit and prescription drugs have on driving; and to develop guidance for others when dealing with drug-impaired driving problems. Delegates represented the fields of psychopharmacology, behavioral psychology, drug chemistry, forensic toxicology, medicine, and law enforcement experts trained in the recognition of drug effects on drivers in the field.

These Fact Sheets represent the conclusions of the Panel and include the state of current scientific knowledge in the area of drugs and human performance for the 16 drugs selected for evaluation. The selected drugs include over-the-counter medications such as dextromethorphan and diphenhydramine; prescription medications such as carisoprodol, diazepam and zolpidem; and abused and/or illegal drugs such as cocaine, GHB, ketamine, LSD, marijuana, methadone, methamphetamine, MDMA, morphine, PCP and toluene.

Keyword continuation: illicit and licit drugs and traffic safety, drugs and driving, drug-impaired driving.

17. Key Words
Carisoprodol, cocaine, dextromethorphan, diazepam, diphenhydramine, GHB, ketamine, LSD, marijuana, methadone, methamphetamine, MDMA, morphine, PCP, toluene, zolpidem,

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# Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduction</td>
<td>3</td>
</tr>
<tr>
<td>Cannabis/Marijuana</td>
<td>7</td>
</tr>
<tr>
<td>Carisoprodol (and Meprobamate)</td>
<td>13</td>
</tr>
<tr>
<td>Cocaine</td>
<td>19</td>
</tr>
<tr>
<td>Dextromethorphan</td>
<td>25</td>
</tr>
<tr>
<td>Diazepam</td>
<td>29</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>35</td>
</tr>
<tr>
<td>Gamma-Hydroxybutyrate (GHB, GBL, and 1,4-BD)</td>
<td>39</td>
</tr>
<tr>
<td>Ketamine</td>
<td>45</td>
</tr>
<tr>
<td>Lysergic acid diethylamide (LSD)</td>
<td>51</td>
</tr>
<tr>
<td>Methadone</td>
<td>55</td>
</tr>
<tr>
<td>Methamphetamine (and Amphetamine)</td>
<td>61</td>
</tr>
<tr>
<td>Methylenedioxyamphetamine (MDMA, Ecstasy)</td>
<td>67</td>
</tr>
<tr>
<td>Morphine (and Heroin)</td>
<td>73</td>
</tr>
<tr>
<td>Phencyclidine (PCP)</td>
<td>79</td>
</tr>
<tr>
<td>Toluene</td>
<td>85</td>
</tr>
<tr>
<td>Zolpidem (and Zaleplon, Zopiclone)</td>
<td>91</td>
</tr>
<tr>
<td>Biographical Sketches of Lead Authors and Main Contributors</td>
<td>97</td>
</tr>
</tbody>
</table>
Introduction

The use of psychoactive drugs followed by driving has been an issue of continual concern to law enforcement officers, physicians, attorneys, forensic toxicologists and traffic safety professionals in the U.S. and throughout the world. At issue are methods for identifying the impaired driver on the road, the assessment and documentation of the impairment they display, the availability of appropriate chemical tests, and the interpretation of the subsequent results. A panel of international experts on drug-related driving issues met to review developments in the field of drugs and human performance over the last 10 years; to identify the specific effects that both illicit and prescription drugs have on driving; and to develop guidance for others when dealing with drug-impaired driving problems.

This publication is based on the deliberations of the International Consultative Panel on Drugs and Driving Impairment held in Seattle, WA in August 2000. This meeting was sponsored by the National Safety Council, Committee on Alcohol and other Drugs; the State of Washington Traffic Safety Commission; and the National Highway Traffic Safety Administration. Delegates represented the fields of psychopharmacology, behavioral psychology, drug chemistry, forensic toxicology, medicine, and law enforcement experts trained in the recognition of drug effects on drivers in the field. The Fact Sheets reflect the conclusions of the Panel and have been designed to provide practical guidance to toxicologists, pharmacologists, law enforcement officers, attorneys and the general public on issues related to drug impaired driving.

Sixteen drugs were selected for review and include over-the-counter medications, prescription drugs, and illicit and/or abused drugs. The selected drugs are cannabis/marijuana, carisoprodol, cocaine, dextromethorphan, diazepam, diphenhydramine, gamma-hydroxybutyrate, ketamine, lysergic acid diethylamide, methadone, methamphetamine/amphetamine, methylenedioxymethamphetamine, morphine/heroin, phencyclidine, toluene, and zolpidem.

The Fact Sheets are based on the state of current scientific knowledge and represent the conclusions of the panel. They have been designed to provide practical guidance to toxicologists, pharmacologists, law enforcement officers, attorneys and the general public to use in the evaluation of future cases. Each individual drug Fact Sheet covers information regarding drug chemistry, usage and dosage information, pharmacology, drug effects, effects on driving, drug evaluation and classification (DEC), and the panel’s assessment of driving risks. A list of key references and recommended reading is also provided for each drug. Readers are encouraged to use the Fact Sheets in connection with the other cited impaired driving-related texts.

The information provided is uniform for all the Fact Sheets and provides details on the physical description of the drug, synonyms, and pharmaceutical or illicit sources; medical and recreational uses, recommended and abused doses, typical routes of administration, and potency and purity; mechanism of drug action and major receptor sites; drug absorption, distribution, metabolism and elimination data; blood and urine concentrations; psychological and physiological effects, and drug interactions; drug
effects on psychomotor performance; driving simulator and epidemiology studies; and drug recognition evaluation profiles. Each Fact Sheet concludes with general statements about the drugs’ ability to impair driving performance. The authors strongly believe that all the above information needs to be taken into account when evaluating a drug.

Case interpretation can be complicated by a number of factors and one of the main limitations of the Fact Sheets is that they primarily relate to single drug use. Other factors which influence the risk of effects on driving for any drug include the dose, the dosage frequency, acute and residual effects, chronic administration, route of administration, the concentration of the drug at the site of action, idiosyncrasies of metabolism, drug tolerance or hypersensitivity, and the combined effects of the drug with other drugs or alcohol, to name but a few.

**Individual Fact Sheets**

Cannabis/Marijuana  
Carisoprodol (and Meprobamate)  
Cocaine  
Dextromethorphan  
Diazepam  
Diphenhydramine  
Gamma-Hydroxybutyrate (GHB, GBL, and 1,4-BD)  
Ketamine  
Lysergic acid diethylamide (LSD)  
Methadone  
Methamphetamine (and Amphetamine)  
Methylenedioxymethamphetamine (MDMA, Ecstasy)  
Morphine (and Heroin)  
Phencyclidine (PCP)  
Toluene  
Zolpidem (and Zaleplon, Zopiclone)

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Disclaimer

The information contained in the Drugs and Human Performance Fact Sheets represents the views of the contributors and not necessarily those of their place of employment or the National Highway Traffic Safety Administration.
Cannabis / Marijuana (Δ⁹-Tetrahydrocannabinol, THC)

Marijuana is a green or gray mixture of dried shredded flowers and leaves of the hemp plant Cannabis sativa. Hashish consists of resinous secretions of the cannabis plant. Dronabinol (synthetic THC) is a light yellow resinous oil.

**Synonyms:** Cannabis, marijuana, pot, reefer, buds, grass, weed, dope, ganja, herb, boom, gangster, Mary Jane, sinsemilla, shit, joint, hash, hash oil, blow, blunt, green, kilobricks, Thai sticks; Marinol®

**Source:** Cannabis contains chemicals called cannabinoids, including cannabinol, cannabidiol, cannabinoic acids, cannabigerol, cannabichromene, and several isomers of tetrahydrocannabinol (THC). One of these isomers, Δ⁹-THC, is believed to be responsible for most of the characteristic psychoactive effects of cannabis. Marijuana refers to the leaves and flowering tops of the cannabis plant; the buds are often preferred because of their higher THC content. Hashish consists of the THC-rich resinous secretions of the plant, which are collected, dried, compressed and smoked. Hashish oil is produced by extracting the cannabinoids from plant material with a solvent. In the U. S., marijuana, hashish and hashish oil are Schedule I controlled substances. Dronabinol (Marinol®) is a Schedule III controlled substance and is available in strengths of 2.5, 5 or 10 mg in round, soft gelatin capsules.

**Drug Class:** Cannabis/Marijuana: spectrum of behavioral effects is unique, preventing classification of the drug as a stimulant, sedative, tranquilizer, or hallucinogen. Dronabinol: appetite stimulant, antiemetic.

**Medical and Recreational Uses:** Medicinal: Dronabinol is indicated for the treatment of anorexia associated with weight loss in patients with AIDS, and to treat mild to moderate nausea and vomiting associated with cancer chemotherapy. Recreational: Marijuana is used for its mood altering effects, euphoria, and relaxation. Marijuana is the most commonly used illicit drug throughout the world.*

**Potency, Purity and Dose:** THC is the major psychoactive constituent of cannabis. Potency is dependent on THC concentration and is usually expressed as %THC per dry weight of material. Average THC concentration in marijuana is 1-5%, hashish 5-15%, and hashish oil ≥ 20%. The form of marijuana known as sinsemilla is derived from the unpollinated female cannabis plant and is preferred for its high THC content (up to 17% THC). Recreational doses are highly variable and users often titer their own dose. A single intake of smoke from a pipe or joint is called a hit (approximately 1/20th of a gram). The lower the potency or THC content the more hits are needed to achieve the desired effects; 1-3 hits of high potency sinsemilla is typically enough to produce the desired effects. In terms of its psychoactive effect, a drop or two of hash oil on a cigarette is equal to a single “joint” of marijuana. Medicinally, the initial starting dose of Marinol® is 2.5 mg, twice daily.

**Route of Administration:** Marijuana is usually smoked as a cigarette (‘joint’) or in a pipe or bong. Hollowed out cigars packed with marijuana are also common and are called

* Updated April 2014
"blunts." Joints and blunts are often laced with adulterants including PCP or crack cocaine. Joints can also be dipped in liquid PCP or in codeine cough syrup. Marijuana is also orally ingested.

**Pharmacodynamics:** THC binds to cannabinoid receptors and interferes with important endogenous cannabinoid neurotransmitter systems. Receptor distribution correlates with brain areas involved in physiological, psychomotor and cognitive effects. Correspondingly, THC produces alterations in motor behavior, perception, cognition, memory, learning, endocrine function, food intake, and regulation of body temperature.

**Pharmacokinetics:** Absorption is slower following the oral route of administration with lower, more delayed peak THC levels. Bioavailability is reduced following oral ingestion due to extensive first pass metabolism. Smoking marijuana results in rapid absorption with peak THC plasma concentrations occurring prior to the end of smoking. Concentrations vary depending on the potency of marijuana and the manner in which the drug is smoked, however, peak plasma concentrations of 100-200 ng/mL are routinely encountered. Plasma THC concentrations generally fall below 5 ng/mL less than 3 hours after smoking. THC is highly lipid soluble, and plasma and urinary elimination half-lives are best estimated at 3-4 days, where the rate-limiting step is the slow redistribution to plasma of THC sequestered in the tissues. Shorter half-lives are generally reported due to limited collection intervals and less sensitive analytical methods. Plasma THC concentrations in occasional users rapidly fall below limits of quantitation within 8 to 12 h. THC is rapidly and extensively metabolized with very little THC being excreted unchanged from the body. THC is primarily metabolized to 11-hydroxy-THC which has equipotent psychoactivity. The 11-hydroxy-THC is then rapidly metabolized to the 11-nor-9-carboxy-THC (THC-COOH) which is not psychoactive. A majority of THC is excreted via the feces (~65%) with approximately 30% of the THC being eliminated in the urine as conjugated glucuronic acids and free THC hydroxylated metabolites.

**Molecular Interactions / Receptor Chemistry:** THC is metabolized via cytochrome P450 2C9, 2C11, and 3A isoenzymes. Potential inhibitors of these isoenzymes could decrease the rate of THC elimination if administered concurrently, while potential inducers could increase the rate of elimination.

**Blood to Plasma Concentration Ratio:** 0.55

**Interpretation of Blood Concentrations:** It is difficult to establish a relationship between a person's THC blood or plasma concentration and performance impairing effects. Concentrations of parent drug and metabolite are very dependent on pattern of use as well as dose. THC concentrations typically peak during the act of smoking, while peak 11-OH THC concentrations occur approximately 9-23 minutes after the start of smoking. Concentrations of both analytes decline rapidly and are often < 5 ng/mL at 3 hours. Significant THC concentrations (7 to 18 ng/mL) are noted following even a single puff or hit of a marijuana cigarette. Peak plasma THC concentrations ranged from 46-188 ng/mL in 6 subjects after they smoked 8.8 mg THC over 10 minutes. Chronic users can have mean plasma levels of THC-COOH of 45 ng/mL, 12 hours after use; corresponding
THC levels are, however, less than 1 ng/mL. Following oral administration, THC concentrations peak at 1-3 hours and are lower than after smoking. Dronabinol and THC-COOH are present in equal concentrations in plasma and concentrations peak at approximately 2-4 hours after dosing.

It is inadvisable to try and predict effects based on blood THC concentrations alone, and currently impossible to predict specific effects based on THC-COOH concentrations. It is possible for a person to be affected by marijuana use with concentrations of THC in their blood below the limit of detection of the method. Mathematical models have been developed to estimate the time of marijuana exposure within a 95% confidence interval. Knowing the elapsed time from marijuana exposure can then be used to predict impairment in concurrent cognitive and psychomotor effects based on data in the published literature.

**Interpretation of Urine Test Results:** Detection of total THC metabolites in urine, primarily THC-COOH-glucuronide, only indicates prior THC exposure. Detection time is well past the window of intoxication and impairment. Published excretion data from controlled clinical studies may provide a reference for evaluating urine cannabinoid concentrations; however, these data are generally reflective of occasional marijuana use rather than heavy, chronic marijuana exposure. It can take as long as 4 hours for THC-COOH to appear in the urine at concentrations sufficient to trigger an immunoassay (at 50ng/mL) following smoking. Positive test results generally indicate use within 1-3 days; however, the detection window could be significantly longer following heavy, chronic, use. Following single doses of Marinol®, low levels of dronabinol metabolites have been detected for more than 5 weeks in urine. Low concentrations of THC have also been measured in over-the-counter hemp oil products – consumption of these products may produce positive urine cannabinoid test results.

**Effects:** Pharmacological effects of marijuana vary with dose, route of administration, experience of user, vulnerability to psychoactive effects, and setting of use.

*Psychological:* At recreational doses, effects include relaxation, euphoria, relaxed inhibitions, sense of well-being, disorientation, altered time and space perception, lack of concentration, impaired learning and memory, alterations in thought formation and expression, drowsiness, sedation, mood changes such as panic reactions and paranoia, and a more vivid sense of taste, sight, smell, and hearing. Stronger doses intensify reactions and may cause fluctuating emotions, flights of fragmentary thoughts with disturbed associations, a dulling of attention despite an illusion of heightened insight, image distortion, and psychosis.

*Physiological:* The most frequent effects include increased heart rate, reddening of the eyes, dry mouth and throat, increased appetite, and vasodilatation.

**Side Effect Profile:** Fatigue, paranoia, possible psychosis, memory problems, depersonalization, mood alterations, urinary retention, constipation, decreased motor coordination, lethargy, slurred speech, and dizziness. Impaired health including lung damage, behavioral changes, and reproductive, cardiovascular and immunological effects have been associated with regular marijuana use. Regular and chronic marijuana smokers may have many of the same respiratory problems that tobacco smokers have (daily cough
and phlegm, symptoms of chronic bronchitis), as the amount of tar inhaled and the level of carbon monoxide absorbed by marijuana smokers is 3 to 5 times greater than among tobacco smokers. Smoking marijuana while shooting up cocaine has the potential to cause severe increases in heart rate and blood pressure.

**Duration of Effects:** Effects from smoking cannabis products are felt within minutes and reach their peak in 10-30 minutes. Typical marijuana smokers experience a high that lasts approximately 2 hours. Most behavioral and physiological effects return to baseline levels within 3-5 hours after drug use, although some investigators have demonstrated residual effects in specific behaviors up to 24 hours, such as complex divided attention tasks. Psychomotor impairment can persist after the perceived high has dissipated. In long term users, even after periods of abstinence, selective attention (ability to filter out irrelevant information) has been shown to be adversely affected with increasing duration of use, and speed of information processing has been shown to be impaired with increasing frequency of use. Dronabinol has an onset of 30-60 minutes, peak effects occur at 2-4 hours, and it can stimulate the appetite for up to 24 hours.

**Tolerance, Dependence and Withdrawal Effect:** Tolerance may develop to some pharmacological effects of dronabinol. Tolerance to many of the effects of marijuana may develop rapidly after only a few doses, but also disappears rapidly. Marijuana is addicting as it causes compulsive drug craving, seeking, and use, even in the face of negative health and social consequences. Additionally, animal studies suggests marijuana causes physical dependence. A withdrawal syndrome is commonly seen in chronic marijuana users following abrupt discontinuation. Symptoms include restlessness, irritability, mild agitation, hyperactivity, insomnia, nausea, cramping, decreased appetite, sweating, and increased dreaming.

**Drug Interactions:** Cocaine and amphetamines may lead to increased hypertension, tachycardia and possible cardiotoxicity. Benzodiazepines, barbiturates, ethanol, opioids, antihistamines, muscle relaxants and other CNS depressants increase drowsiness and CNS depression. When taken concurrently with alcohol, marijuana is more likely to be a traffic safety risk factor than when consumed alone.

**Performance Effects:** The short term effects of marijuana use include problems with memory and learning, distorted perception, difficulty in thinking and problem-solving, and loss of coordination. Heavy users may have increased difficulty sustaining attention, shifting attention to meet the demands of changes in the environment, and in registering, processing and using information. In general, laboratory performance studies indicate that sensory functions are not highly impaired, but perceptual functions are significantly affected. The ability to concentrate and maintain attention are decreased during marijuana use, and impairment of hand-eye coordination is dose-related over a wide range of dosages. Impairment in retention time and tracking, subjective sleepiness, distortion of time and distance, vigilance, and loss of coordination in divided attention tasks have been reported. Note however, that subjects can often “pull themselves together” to concentrate on simple tasks for brief periods of time. Significant performance impairments are
usually observed for at least 1-2 hours following marijuana use, and residual effects have been reported up to 24 hours.

**Effects on Driving:** The drug manufacturer suggests that patients receiving treatment with Marinol® should be specifically warned not to drive until it is established that they are able to tolerate the drug and perform such tasks safely. Epidemiology data from road traffic arrests and fatalities indicate that after alcohol, marijuana is the most frequently detected psychoactive substance among driving populations. Marijuana has been shown to impair performance on driving simulator tasks and on open and closed driving courses for up to approximately 3 hours. Decreased car handling performance, increased reaction times, impaired time and distance estimation, inability to maintain headway, lateral travel, subjective sleepiness, motor incoordination, and impaired sustained vigilance have all been reported. Some drivers may actually be able to improve performance for brief periods by overcompensating for self-perceived impairment. The greater the demands placed on the driver, however, the more critical the likely impairment. Marijuana may particularly impair monotonous and prolonged driving. Decision times to evaluate situations and determine appropriate responses increase. Mixing alcohol and marijuana may dramatically produce effects greater than either drug on its own.

**DEC Category:** Cannabis

**DEC Profile:** Horizontal gaze nystagmus not present; vertical gaze nystagmus not present; lack of convergence present; pupil size normal to dilated; reaction to light normal to slow; pulse rate elevated; blood pressure elevated; body temperature normal to elevated. Other characteristic indicators may include odor of marijuana in car or on subject’s breath, marijuana debris in mouth, green coating of tongue, bloodshot eyes, body and eyelid tremors, relaxed inhibitions, incomplete thought process, and poor performance on field sobriety tests.

**Panel's Assessment of Driving Risks:** Low doses of THC moderately impair cognitive and psychomotor tasks associated with driving, while severe driving impairment is observed with high doses, chronic use and in combination with low doses of alcohol. The more difficult and unpredictable the task, the more likely marijuana will impair performance.

**References and Recommended Reading:**


Carisoprodol (and Meprobamate)
Carisoprodol is a white, crystalline powder. Meprobamate is a white powder. Both are available in tablet form.


Source: Carisoprodol and meprobamate are available by prescription only. Carisoprodol itself is not a federally scheduled compound, while meprobamate is a Schedule IV drug. Soma® is available as a 350 mg strength round, white tablet; Soma® Compound is a 250 mg strength two-layered, white and light orange round tablet (also contains aspirin); and Soma® Compound with Codeine is a 250 mg strength two-layered, white and yellow oval tablet (also contains aspirin and codeine phosphate) and is a schedule III controlled substance. Miltown® is available as a 200 mg and 400 mg strength white tablet; Equanil® is a 200 mg and 400 mg strength tablet; and Equagesic® is a 200 mg strength two-layered, pink and yellow, round tablet (also contains aspirin).

Drug Class: Carisoprodol: muscle relaxant, CNS depressant; Meprobamate: antianxiety, CNS depressant.

Medicinal and Recreational Uses: Carisoprodol is a centrally acting skeletal muscle relaxant prescribed for the treatment of acute, musculoskeletal pain. Meprobamate is a major metabolite of carisoprodol, and is a CNS depressant in its own right, indicated for the management of anxiety disorders or for short-term treatment of anxiety symptoms. Use of these drugs begins with prescription for muscular pain or anxiety, and abuse develops for their sedative-hypnotic effects, resulting in increased dosage without medical advice, or continued use after pain or anxiety has subsided.

Potency, Purity and Dose: Carisoprodol is present as a racemic mixture. During treatment, the recommended dose of carisoprodol is for one 350 mg tablet taken three times daily and at bedtime (1400 mg/day). The usual dose for meprobamate is one 400 mg taken four times daily, or daily divided doses of up to 2400 mg. To control chronic pain, carisoprodol is often taken concurrently with other drugs, particularly opiates, benzodiazepines, barbiturates, and other muscle relaxants.

Route of Administration: Oral.

Pharmacodynamics: The pharmacological effects of carisoprodol appear to be due to the combination of the effects of carisoprodol and its active metabolite, meprobamate. Meprobamate is equipotent to carisoprodol. There is some evidence suggesting carisoprodol is a GABA<sub>Α</sub> receptor indirect agonist with CNS chloride ion channel conductance effects. In animals, carisoprodol produces muscle relaxation by blocking interneuronal activity and depressing transmission of polysynaptic neurons in the descending reticular formation and spinal cord. It is unknown if this mechanism of action is also present in humans. In addition to the desired skeletal muscle relaxing effects,
Carisoprodol and meprobamate produce weak anticholinergic, antipyretic and analgesic properties.

**Pharmacokinetics:** Carisoprodol is rapidly absorbed from the gastrointestinal tract and rapidly distributed throughout the CNS. Protein binding is approximately 60%. Carisoprodol is predominantly dealkylated to meprobamate in the liver, and to a lesser extent hydroxylated to hydroxycarisoprodol and hydroxymeprobamate, followed by conjugation and excretion. The half-life of carisoprodol is approximately 100 minutes. Some individuals have impaired metabolism of carisoprodol, and exhibit a half life of 2-3 times that in normal subjects. The half-life of meprobamate is many times longer, between 6 and 17 hours. As a result of the significantly longer half-life of meprobamate relative to carisoprodol, accumulation of meprobamate during chronic therapy may occur.

**Molecular Interactions / Receptor Chemistry:** The cytochrome P450 2C19 isoenzyme is responsible for the conversion of carisoprodol to meprobamate. Potential inhibitors of the 2C19 isoenzyme could decrease the rate of drug elimination if administered concurrently, while potential inducers of the 2C19 isoenzyme could increase the rate of elimination.

**Blood to Plasma Concentration Ratio:** Data not available for carisoprodol; 3.3 to 5.0 for meprobamate.

**Interpretation of Blood Concentrations:** Following therapeutic doses of carisoprodol, blood concentrations are typically between 1 and 5 mg/L for carisoprodol, and between 2 and 6 mg/L for meprobamate. A single oral dose of 350 mg carisoprodol produced average peak plasma concentrations of 2.1 mg/L carisoprodol at one hour, declining to 0.24 mg/L at 6 hours. Following a single oral dose of 700 mg, average peak plasma concentrations of carisoprodol were 3.5 mg/L at 45 minutes, and meprobamate concentrations of 4.0 mg/L were obtained in 220 minutes. A single oral dose of 700 mg carisoprodol has also produced peak plasma concentrations of 4.8 mg/L carisoprodol. Following administration of meprobamate in the treatment of anxiety, concentrations are typically around 10 mg/L, but can range between 3 and 26 mg/L. A single oral dose of 1200 mg meprobamate produced concentrations of 15.6 mg/L at 4 hours. Plasma meprobamate concentrations of greater than 100 mg/L have been associated with deep coma; light coma between 60 and 120 mg/L; and patients with levels below 50 mg/L are invariably conscious.

**Interpretation of Urine Test Results:** Both drugs are excreted into the urine and are likely be detectable for several days following cessation of use. Less than 1% of a single oral dose of carisoprodol is excreted unchanged in the 24 hour urine, with meprobamate accounting for 4.7% of the dose. Following administration of meprobamate, up to 11% of a single dose is excreted in the urine in 24 hours.

**Effects:**
*Psychological:* Dizziness, drowsiness, sedation, confusion, disorientation, slowed thinking, lack of comprehension, drunken behavior, obtunded, coma.
Physiological: CNS depression, nystagmus (becoming more evident as concentrations increase), loss of balance and coordination, sluggish movements, slurred speech, bloodshot eyes, ataxia, tremor, sleep disturbances.

Side Effect Profile: Agitation, tremor, paresthesia, irritability, depression, facial flushing, headache, vertigo, postural hypotension, fainting, weakness, loss of balance and coordination, impairment of visual accommodation, tachycardia, nausea, vomiting, and stomach upset. In abuse or overdose, subjects are consistently sedated and obtunded, frequently becoming comatose. Overdose symptoms may include shallow breathing, clammy skin, dilated pupils, weak and rapid pulse, paradoxical excitement and insomnia, convulsions, and possible death. Meprobamate overdose can produce drowsiness, ataxia, severe respiratory depression, severe hypotension, shock, heart failure, and death.

Duration of Effects: The effects of carisoprodol begin within 30 minutes of oral administration, and last for up to 4-6 hours. In overdose, coma may last from several hours to a day or more. Meprobamate has a much longer duration of effect than carisoprodol due to a much longer half-life.

Tolerance, Dependence and Withdrawal: Development of abuse and moderate physical and psychological dependence can occur with chronic use of both carisoprodol and meprobamate. Abrupt discontinuation of long-term use can be followed by mild withdrawal symptoms such as anxiety, abdominal cramps, insomnia, headache, nausea, vomiting, ataxia, tremor, muscle twitching, confusion, and occasionally chills, convulsions and hallucinations. Onset of withdrawal from meprobamate occurs within 12-48 hours following cessation of use, and can last a further 12-48 hours. Carisoprodol has been shown to produce cross-tolerance to barbiturates.

Drug Interactions: Alcohol enhances the impairment of physical abilities produced by carisoprodol, and increased sedation, extreme weakness, dizziness, agitation, euphoria and confusion may be observed. Alcohol also inhibits the metabolism of meprobamate and produces an additive depressant effect on the CNS that includes sleepiness, disorientation, incoherence and confusion. The concurrent administration of other centrally acting drugs such as opiates, benzodiazepines, barbiturates, and other muscle relaxants can contribute to impairment. Meprobamate may enhance the analgesic effects of other drugs.

Performance Effects: Very limited studies are available for carisoprodol, however, single oral doses of 700 mg have not been shown to affect psychomotor and cognitive tests within 3 hours of dosing, to a significant degree. In contrast, single doses of meprobamate are capable of causing significant performance impairment. Performance effects include impaired divided attention, impaired coordination and balance, slowed reflexes and increased reaction time. With chronic dosing of either drug, it is likely that decrements in psychomotor performance would be even more pronounced.

Effects on Driving: The drug manufacturer suggests patients should be warned that carisoprodol and meprobamate may impair the mental and/or physical abilities required
for the performance of potentially hazardous tasks, such as driving a motor vehicle. Reported signs of psychomotor and cognitive impairment in subjects found to be driving under the influence of carisoprodol/meprobamate include poor perception, impaired reaction time, slow driving, confusion, disorientation, inattentiveness, slurred or thick speech, slow responses, somnolence, lack of balance and coordination, unsteadiness, and difficulty standing, walking or exiting vehicles.

Logan et al., 2000 describes 21 driving under the influence cases where carisoprodol and/or meprobamate were the only drugs detected. The mean carisoprodol and meprobamate concentrations were 4.6 mg/L (range 0-15 mg/L) and 14.5 mg/L (range 1-36 mg/L), respectively. Signs of impairment were noted at blood concentrations as low as 1 mg/L of meprobamate, however, the most severe driving impairment and the most overt symptoms of intoxication occurred in drivers whose combined carisoprodol and meprobamate blood concentrations were greater than 10 mg/L. Signs consistent with CNS depression were typically observed, including poor balance and coordination, horizontal gaze nystagmus, slurred speech, dazed or groggy appearance, depressed reflexes, slow movements, disorientation to place and time, and a tendency to dose off or fall asleep. Many subjects were involved in accidents, and other observed driving behaviors included extreme lane travel and weaving, striking other vehicles and fixed objects, slow speed, and hit and run accidents where the subject appeared unaware they had hit another vehicle.

**DEC Category:** CNS depressant

**DEC Profile:** Horizontal gaze nystagmus present; vertical gaze nystagmus may be present in high doses; lack of convergence present; pupil size normal to dilated; reaction to light slow; pulse rate normal to down; blood pressure normal to down; body temperature normal to down. Other characteristic indicators may include slurred speech, drowsiness, disorientation, drunken behavior without the odor of alcohol, and poor performance on field sobriety tests.

**Panel’s Assessment of Driving Risks:** A single therapeutic dose of carisoprodol is unlikely to cause significant performance impairment. However, single therapeutic doses of meprobamate and chronic doses of carisoprodol may produce moderate to severe impairment of psychomotor skills associated with safe driving.

**References and Recommended Reading:**


Cocaine
Cocaine hydrochloride is a white to light brown crystalline powder, shiny rather than dull in appearance. Cocaine base is white to beige in color; waxy/soapy to flaky solid chunks.

**Synonyms:** Methylbenzylecgonine. *Cocaine hydrochloride:* coke, snow, flake, blow, cane, dust, shake, toot, nose candy, white lady. *Cocaine base:* crack, rock, free-base.

**Source:** Naturally derived CNS stimulant extracted and refined from the leaves of the coca plant (*Erythroxylon coca*), grown primarily in the Andean region of South America and to a lesser extent in India, Africa and Indonesia. The picked coca leaves are dried in the open air and then “stomped” as part of the process to extract the alkaloid, resulting in coca paste and eventually cocaine hydrochloride. It is illegal to possess and sell cocaine in the U.S. and cocaine is a Schedule II controlled substance. “Crack” is the street name given to cocaine that has been processed from cocaine hydrochloride. It is prepared by adding baking soda to aqueous cocaine hydrochloride and heating it until the free-base cocaine precipitates into small pellets. The mixture is cooled and filtered, and then the “rocks” are smoked in a crack pipe.

**Drug Class:** CNS stimulant, local anesthetic.

**Medical and Recreational Uses:** Minor use as a topical local anesthetic for ear, nose and throat surgery. Traditionally, the coca leaves are chewed or brewed into a tea for refreshment and to relieve fatigue. Recreationally, cocaine is used to increase alertness, relieve fatigue, feel stronger and more decisive, and is abused for its intense euphoric effects.

**Potency, Purity and Dose:** In ear, nose and throat surgery cocaine is commercially supplied as the hydrochloride salt in a 40 or 100 mg/mL solution. Depending on the demographic region, street purity of cocaine hydrochloride can range from 20-95%, while that of crack cocaine is 20-80%. The hydrochloride powder is often diluted with a variety of substances such as sugars for bulk (lactose, sucrose, inositol, mannitol), other CNS stimulants (caffeine, ephedrine, phenylpropanolamine), or other local anesthetics (lidocaine, procaine, benzocaine). Commonly abused doses are 10-120 mg. Repeated doses are frequently taken to avoid the dysphoric crash that often follows the initial intense euphoric effects. Cocaine is frequently used in combination with other drugs; injected with heroin (“speedball”) or taken with alcohol to reduce irritability; smoked with phencyclidine (“tick”); and smoked in marijuana blunts (“turbo”).

**Route of Administration:** Topically applied for use as a local anesthetic. Recreationally, coca leaves can be chewed, however, cocaine abusers typically smoke “crack” in a glass pipe or inject the hydrochloride salt intravenously. Cocaine hydrochloride can be smoked to some effect but this is very inefficient as the powder tends to burn rather than vaporize. Snorting (insufflation/intransanal) is also popular. Subcutaneous injection (skin-popping) is rarely used.
**Pharmacodynamics:** Cocaine is a strong CNS stimulant that interferes with the reabsorption process of catecholamines, particularly dopamine, a chemical messenger associated with pleasure and movement. Cocaine prevents the reuptake of dopamine by blocking the dopamine transporter which leads to increased extracellular dopamine, resulting in chronic stimulation of postsynaptic dopamine receptors. This results in the euphoric ‘rush’. When dopamine levels subsequently fall, users experience a dysphoric ‘crash’. Similarly, cocaine interferes with the uptake of norepinephrine and serotonin (5-HT), leading to accumulation of these neurotransmitters at postsynaptic receptors. As a local anesthetic, cocaine reversibly blocks the initiation and conduction of the nerve impulse. Cocaine additionally produces vasoconstriction and dilated pupils.

**Pharmacokinetics:** Cocaine is rapidly absorbed following smoking, snorting and intravenous administration. Bioavailability is 57% following snorting and ~70% following smoking. Cocaine is 91% bound in plasma. Cocaine is extensively metabolized to a variety of compounds: benzoylecgonine, ecgonine, and ecgonine methyl ester are the major metabolites and are centrally inactive. Benzoylecgonine is produced upon loss of the methyl group and is the major urinary metabolite. Norcocaine is a very minor metabolite, but is active and neurotoxic. Cocaethylene, formed following concurrent ingestion of cocaine and alcohol, is also active and is equipotent to cocaine in blocking dopamine reuptake. The apparent half-life for cocaine is short, approximately 0.8 ± 0.2 hours, while the half-life of benzoylecgonine is 6 hours.

**Molecular Interactions / Receptor Chemistry:** The cytochrome P450 3A4 isoenzyme is responsible for the N-demethylation of cocaine to norcocaine. Potential inhibitors of the 3A4 isoenzyme could decrease the rate of drug elimination if administered concurrently, while potential inducers could increase the rate of drug elimination. Cocaine itself is an inhibitor of the CYP2D6 isoform.

**Blood to Plasma Concentration Ratio:** averages ~ 1.0

**Interpretation of Blood Concentrations:** The presence of cocaine at a given blood concentration cannot usually be associated with a degree of impairment or a specific effect for a given individual without additional information. This is due to many factors, including individual levels of tolerance to the drug and artifactual changes in cocaine concentrations on storage. There is a large overlap between therapeutic, toxic and lethal cocaine concentrations and adverse reactions have been reported after prolonged use even with no measurable parent drug in the blood. Typical concentrations in abuse range from 0-1mg/L, however, concentrations up to 5mg/L and higher are survivable in tolerant individuals. After single doses of cocaine, plasma concentration typically average 0.2-0.4 mg/L. Repeated doses of cocaine may result in concentrations greater than 0.75 mg/L.

Following intranasal administration of 106 mg, peak plasma concentrations of cocaine averaged 0.22 mg/L at 30 minutes, while benzoylecgonine concentrations averaged 0.61 mg/L at 3 hours. Oral administration of 140 mg/70 kg cocaine resulted in peak plasma concentrations averaging 0.21 mg/L of cocaine at 1 hour. Single 32 mg intravenous doses of cocaine produced an average peak plasma concentration of 0.31 mg/L of cocaine within 5 minutes. Smoking 50 mg of cocaine base resulted in peak
plasma cocaine concentrations averaging 0.23 mg/L at ~ 45 minutes and 0.15 mg/L of benzoylecgonine at 1.5 hours.

**Interpretation of Urine Test Results:** Urinary excretion is less than 2% for unchanged cocaine, 26-39% for benzoylecgonine, and 18-22% for ecgonine methyl ester. 64-69% of the initial dose is recovered after 3 days. Very low concentrations of cocaine may be detected in urine during the initial few hours, however, benzoylecgonine persists in urine at detectable concentrations from 2-4 days. Chronic, heavy use of cocaine can result in detectable amounts of benzoylecgonine in urine for up to 10 days following a binge.

**Effects:**

**Early phase – Psychological:** Euphoria, excitation, feelings of well-being, general arousal, increased sexual excitement, dizziness, self-absorbed, increased focus and alertness, mental clarity, increased talkativeness, motor restlessness, offsets fatigue, improved performance in some simple tasks, and loss of appetite. Higher doses may exhibit a pattern of psychosis with confused and disoriented behavior, delusions, hallucinations, irritability, fear, paranoia, antisocial behavior, and aggressiveness.

**Physiological:** Increased heart rate and blood pressure, increased body temperature, dilated pupils, increased light sensitivity, constriction of peripheral blood vessels, rapid speech, dyskinesia, nausea, and vomiting.

**Late phase - Psychological:** Dysphoria, depression, agitation, nervousness, drug craving, general CNS depression, fatigue, insomnia.

**Physiological:** Itching/picking/scratching, normal heart rate, normal pupils.

**Side Effect Profile:** Nervousness, restlessness, tremors, anxiety, and irritability. Chronic use may lead to personality changes, hyperactivity, psychosis, paranoia, and fear. Cocaine overdose can be characterized by agitation, enhanced reflexes, hostility, headache, tachycardia, irregular respiration, chills, nausea, vomiting, abdominal pain, rise in body temperature, hallucinations, convulsions, delirium, unconsciousness, seizures, stroke, cerebral hemorrhage, heart failure, and death from respiratory failure. Cocaine excited delirium is a syndrome often caused by excessive cocaine use, and is associated with a dissociative state, violence to persons and property, exaggerated strength, hyperthermia, cardiorespiratory arrest and sudden death.

Burnt lips and fingers from crack pipes are frequently seen, as are rashes and skin reddening from scratching. Smokers may suffer from acute respiratory problems including cough, shortness of breath, and severe chest pains with lung trauma and bleeding. Prolonged cocaine snorting can result in ulceration of the mucous membrane of the nose. The injecting drug user is at risk for transmitting or acquiring HIV infection/AIDS if needles or other injection equipment are shared.

**Duration of Effects:** The faster the absorption the more intense and rapid the high, but the shorter the duration of action. Injecting cocaine produces an effect within 15-30 seconds. A hit of smoked crack produces an almost immediate intense experience and will typically produce effects lasting 5-15 minutes. Similarly, snorting cocaine produces effects almost immediately and the resulting high may last 15-30 minutes. The effects
onset more slowly after oral ingestion (~1 hour). General effects will persist for 1-2 hours depending on the dose and late phase effects following binge use may last several days.

**Tolerance, Dependence and Withdrawal Effects:** Cocaine is a powerfully addictive drug of abuse and an appreciable initial tolerance to the euphoric high may develop. Cocaine is psychologically addicting, particularly with heavy or frequent use, and possibly physically addicting as well. The short duration of effects is one reason leading to probability of addition. As effects wear off, more drug is frequently administered and a pattern of repeated use occurs. Following binge use of cocaine, the “crash” can last from 9 hours to 4 days and may consist of agitation, depressed moods, insomnia to hypersomnolence, and initial drug craving. Withdrawal symptoms can typically last from 1-3 weeks and may consist of alternating low and high drug craving, low to high anxiety, paranoia, dysphoria, depression, apathy, irritability, disorientation, hunger, fatigue, bradycardia, and long periods of sleep.

**Drug Interactions:** The combined use of cocaine and ethanol forms cocaethylene in the body, a substance which intensifies cocaine’s euphoric effects while possibly increasing the risk of sudden death. In laboratory studies, cocaine has been shown to partially reverse some of the adverse effects of alcohol, but may contribute to the detrimental effects of marijuana.

**Performance Effects:** Most laboratory-based studies have been limited by the low doses of cocaine that were allowed. At these single low doses, studies have shown performance enhancement in attentional abilities and increased behavioral and cortical arousal, but have no enhancement of effects on learning, memory, and other cognitive processes. Faster reaction times and diminished effects of fatigue have been observed. Improvements were greatest in behaviorally impaired subjects (e.g. sleep deprived, fatigued, or concurrent use of ethanol) and least improvements were observed in well-rested, healthy subjects. More deleterious effects are expected after higher doses, chronic ingestion and during drug withdrawal, and include agitation, anxiety, distress, inability to focus on divided attention tasks, inability to follow directions, confusion, hostility, time distortion, and poor balance and coordination. Laboratory studies have also demonstrated increased risk taking (rapid braking or steering) and deleterious effects on vision related to mydriasis. Self-reported increases in sensitivity to light, seeing halos around bright objects, flashes or movement of light in peripheral field, difficulty focusing, blurred vision, and glare recovery problems have been reported.

**Effects on Driving:** Observed signs of impairment in driving performance have included subjects speeding, losing control of their vehicle, causing collisions, turning in front of other vehicles, high-risk behavior, inattentive driving, and poor impulse control. As the effects of cocaine wear off subjects may suffer from fatigue, depression, sleepiness, and inattention. In epidemiology studies of driving under the influence cases, accidents, and fatally injured drivers, between 8-23% of subjects have had cocaine and/or metabolites detected in their blood. An examination of 253 fatally injured drivers in Wayne County, Michigan between 1996-1998, found that 10% of cases were positive for blood cocaine and/or metabolites. On review of accident and witness reports, aggressive
driving (high speed and loss of vehicle control) was revealed as the most common finding. Ethanol was detected in 56% of these cases, and all of these drivers lost control of their vehicles. In Memphis, Tennessee in 1993, 13% of 150 drivers stopped for reckless driving were determined to be driving under the influence of cocaine based on observations of behavior and appearance, performance on field sobriety tests, and positive urine cocaine tests.

A 25 year-old male driver, who made an improper turn against oncoming traffic, had a blood cocaine concentration of 0.04 mg/L and 0.06 mg/L of benzoylecgonine, 2 hours after the collision. A 30 year-old female caused an accident after failing to stop at a traffic light; the driver admitted to ingesting a large amount of cocaine ~ 2.5 hours prior to the collision, and 0.32 mg/L cocaine was detected in her blood 1 hour post accident.

**DEC Category:** CNS stimulant.

**DEC Profile:** Horizontal gaze nystagmus not present; vertical gaze nystagmus not present; lack of convergence not present; pupil size dilated; reaction to light slow; pulse rate elevated; blood pressure elevated; body temperature elevated. Other characteristic indicators may include excessive activity, increased alertness, talkativeness, irritability, argumentativeness, nervousness, body tremors, anxiety, redness to nasal area and runny nose.

**Panel's Assessment of Driving Risks:** Single low doses of cocaine may improve mental and motor performance in persons who are fatigued or sleep deprived, however, cocaine does not necessarily enhance the performance of otherwise normal individuals. Cocaine may enhance performance of simple tasks but not complex, divided-attention tasks such as driving. Most laboratory studies have been limited by the low single doses of cocaine administered to subjects. At these low doses, most studies showed performance enhancement in attentional abilities but no effect on cognitive abilities. Significant deleterious effects are expected after higher doses, chronic ingestion, and during the crash or withdrawal phase.

**References and Recommended Reading:**


*Physicians’ Desk Reference*, Medical Economics Company, Montvale, NJ, 2002


Dextromethorphan
Dextromethorphan is a white powder. Available primarily in tablet, capsule and liquid form.

**Synonyms:** 3-methoxy-17-methyl-9α, 13α, 14 α-morphinan hydrobromide monohydrate; dextromethorphan hydrobromide, DXM, “robbo tripping”; Anaplex-DM®, Diabe-Tuss DM™, Benylin®, Pertussin®, Delsym®, Sucrets®, Bromfed-DM®, Robitussin®, Vicks Formula 44, etc.

**Source:** Synthetic analog of codeine and d-isomer of 3-methoxy-N-methylmorphinan. Available as lozenges, capsules, tablets, and cough syrups, in a variety of prescription medications and over-the-counter cough and cold remedies. Products contain dextromethorphan alone or in combination with guaifenesin, brompheniramine, pseudoephedrine, phenylephrine, promethazine, codeine, acetaminophen, and/or chlorpheniramine. For example, Diabe-Tuss DM™ syrup contains 15 mg dextromethorphan; Benylin® Adult and Pediatric contain 15 mg and 7.5 mg dextromethorphan, respectively; and Anaplex-DM® contains 30 mg dextromethorphan, 4 mg brompheniramine and 60 mg pseudoephedrine.

**Drug Class:** Non-opioid antitussive, cough suppressant, CNS depressant (in high doses).

**Medical and Recreational Uses:** Used as an antitussive for temporary relief of coughs caused by minor throat and bronchial irritation. Recreationally used for effects ranging from mild stimulation and intoxication, to dissociation.

**Potency, Purity and Dose:** As an antitussive, the recommended dosage for adults and children aged 12 years and older is 60-120 mg daily in divided doses; for children aged 6-12 years, 30-60 mg daily in divided doses; and for children aged 2-6 years, 15-30 mg daily in divided doses. Each brand contains different quantities of dextromethorphan, generally 20-30 mg per dose, and the majority contain other drugs as previously mentioned. Approximate recreational doses are: threshold dose 80-90 mg; light 100-200 mg; common 200-400 mg; strong 400-600; and heavy dose 600-1500 mg.

**Route of Administration:** Oral.

**Pharmacodynamics:** Dextromethorphan acts centrally to elevate the threshold for coughing, and has no significant analgesic or sedative properties at antitussive doses. It is proposed that dextromethorphan is a glutamate and NMDA antagonist, and blocks the dopamine reuptake site. It may also increase 5HT1A activity possibly via NMDA antagonism.

**Pharmacokinetics:** Dextromethorphan is rapidly absorbed from the gastrointestinal tract and peak plasma concentrations are reached in approximately 2.5 hours. Dextromethorphan is widely distributed, and is rapidly and extensively metabolized by the liver. Dextromethorphan is demethylated to dextrorphan, an active metabolite, and to
3-methoxymorphinan and 3-hydroxymorphinan. It is primarily excreted as unchanged parent drug and dextrorphan.

**Molecular Interactions / Receptor Chemistry:** The cytochrome P450 2D6 isoenzyme is responsible for the conversion of dextromethorphan to dextrorphan; and P450 3A4 and 3A5 isoenzymes are responsible for converting dextromethorphan to 3-methoxymorphinan and 3-hydroxymorphinan. Potential inhibitors of these isoenzymes could decrease the rate of dextromethorphan elimination if administered concurrently, while potential inducers could increase the rate of elimination.

**Blood to Plasma Concentration Ratio:** Data not available.

**Interpretation of Blood Concentrations:** A single 20 mg oral dose of dextromethorphan produced peak concentrations of 1.8 ng/mL in serum after 2.5 hours. Chronic oral dosing of 120 mg daily, in divided doses, resulted in peak plasma dextromethorphan concentrations of 0.5–5.9 ng/mL (mean 2.4 ng/mL) in extensive metabolizers, and 182–231 ng/mL (mean 207 ng/mL) in poor metabolizers.

**Interpretation of Urine Test Results:** In a 24 hour period, less than 2.5% of a dose is excreted unchanged in the urine, while up to 30% of the conjugated dextrorphan is excreted.

**Effects:** At recommended doses, dextromethorphan produces little or no CNS depression. At recreational doses, positive effects may include acute euphoria, elevated mood, dissociation of mind from body, creative dream-like experiences, and increased perceptual awareness. Other effects include disorientation, confusion, pupillary dilation, and altered time perception, visual and auditory hallucinations, and decreased sexual functioning. Recreational doses of approximately 100-200 mg have a mild, stimulant effect (likened to MDA); doses of 200-500 mg produce a more intoxicating effect (likened to being ‘drunk and stoned’); 500-1000 mg may result in mild hallucinations and a mild dissociate effect (likened to a low dose of ketamine) and an overall disturbance in thinking, senses and memory; while doses over 1000 mg may produce a fully dissociative effect (likened to a high dose of ketamine). Recreationally abused doses are capable of impairing judgment, memory, language, and other mental performances.

**Side Effect Profile:** Adverse effects with recommended antitussive doses are rare. However, nausea, other gastrointestinal disturbances, slight drowsiness and dizziness can occur. Following acute doses of between 250-1500 mg, the following clinical and overdose symptoms have been reported: excitation, nausea, vomiting, drowsiness, dizziness, blurred vision, nystagmus, dilated pupils, body itching, rash, ataxia, sweating, hot/cold flashes, fever, hypertension, shallow respiration, urinary retention, diarrhea, opisthotonos (spasm where head and heels are bent back, and torso is bent forward), toxic psychosis (hyperactivity, marked visual and auditory hallucinations), coma, and an increase in heart rate, blood pressure and body temperature. Side effects can be serious if very large doses of the combined preparations are ingested; for example, guaifenesin and
Dextromethorphan can cause severe nausea and vomiting; chlorpheniramine and dextromethorphan can cause seizure, loss of consciousness and bleeding.

**Duration of Effects:** Dextromethorphan exerts its antitussive effects within 15-30 minutes of oral administration. The duration of action is approximately 3-6 hours with conventional dosage forms.

**Tolerance, Dependence and Withdrawal Effects:** At recommended antitussive doses, addiction does not occur. Mild psychological dependence and depression may occur with regular use of increased doses. Abrupt discontinuation of higher doses may produce insomnia, dysphoria and depression. Poor metabolizers of dextromethorphan have been shown to tolerate lower doses of the drug compared to extensive metabolizers, and report greater sedation, dysphoria and psychomotor impairment. Preliminary evidence also suggests that extensive metabolizers may report a greater dextromethorphan abuse potential due to the increased rate of metabolism to the active metabolite dextrorphan.

**Drug Interactions:** Should not be taken with Monoamine Oxide Inhibitors (MAOIs) and Selective Serotonin Reuptake Inhibitors (SSRIs) because of an apparent serotonin syndrome (fever, hypertension, arrhythmias). Should be used with caution in atopic children due to histamine release. Additive CNS depressant effects when co-administered with alcohol, antihistamines, psychotropics, and other CNS depressant drugs.

**Performance Effects:** Minimal at therapeutic levels, however, with high doses one can expect gross cognitive and psychomotor impairment.

**Effects on Driving:** Little to no effect at therapeutic levels, however with high doses one could expect significant impairment. The drug manufacturer states that the combined preparation of promethazine and dextromethorphan may cause marked drowsiness or impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a vehicle. Patients should be told to avoid engaging in such activities until it is known that they do not become drowsy or dizzy. Similar effects could be seen with other combined dextromethorphan preparations.

**DEC Category:** CNS depressant

**DEC Profile:** Data not available; however, the profile for a CNS depressant is: horizontal gaze nystagmus present; vertical gaze nystagmus present at high doses; lack of convergence present; pupil size normal to dilated; reaction to light slow; pulse rate down; blood pressure down; body temperature normal. Such effects are more likely to be seen following recreational doses of dextromethorphan.

**Panel’s Assessment of Driving Risks:** Minimal to no risk at therapeutic levels. Potentially mild to moderate driving risk with higher recreational use.

**References and Recommended Reading:**


**Diazepam**

Diazepam is a colorless, crystalline compound. Available primarily in tablet or liquid form.

*Synonyms:* 7-chloro-1,3-dihydro-1-methyl-5-phenyl-2H-1,4-benzodiazepin-2-one; Valium®, Valrelease®, Vazepam®, Diaz Intensol®, Diastat®, Dizac®.

*Sources:* Diazepam is a Schedule IV controlled substance and is available by prescription in tablet, gel and injectable form. Valium® tablets are white (2 mg), yellow (5 mg) or blue (10 mg) round tabs with a cut out “V” design. Valium® Injectable is available in 5 mg/mL strength liquid.

*Drug Class:* Tranquilizer, sedative, CNS depressant.

*Medical and Recreational Uses:* Used medicinally in the management of anxiety disorders, as an adjunct for the relief of skeletal muscle spasm and for convulsive disorders/status epilepticus, and as a minor tranquilizer or sedative. Also used to suppress or dampen acute alcohol withdrawal, and anxiety-related gastrointestinal disorders such as stress ulcers. Diazepam is used recreationally as a sedative or to enhance the effects of alcohol or opioids. For example, administration of diazepam 30 minutes after a dose of oral methadone reportedly produces an augmented high. Diazepam is used by cocaine users to increase seizure threshold and by heroin users to enhance the effects of heroin, and by both of these users to reduce the impact of withdrawal symptoms between doses.

*Potency, Purity and Dose:* Commonly prescribed doses of Valium® are 5-40 mg daily. For anxiety, 2-10 mg is taken twice to four times daily; for alcohol withdrawal symptoms 10 mg is taken three to four times daily. For the injectable form, 2-20 mg is administered intramuscularly or intravenously. Street doses may consist of several tablets administered at once.

*Route of Administration:* Usually oral, but intravenous injection is possible after preparing a solution from crushed tablets. Commercially available liquid Valium® can be injected, and gel forms can be rectally administered.

*Pharmacodynamics:* Diazepam is a 1,4-benzodiazepine, which binds with high affinity to the GABA<sub>A</sub> receptor in the brain to reduce arousal and to affect emotions. Diazepam’s action causes an increase in affinity of the major inhibitory neurotransmitter, GABA. GABA binds mainly to the α subunit while diazepam binds to the β subunit. The γ subunit is also essential for modulation of chloride transport by benzodiazepines. Diazepam increases chloride transport through ion-channels and ultimately reduces the arousal of the cortical and limbic systems in the CNS. Diazepam depresses the electrical after-discharge in the amygdala and hippocampus regions of the limbic system that affect emotions.

*Pharmacokinetics:* Diazepam is rapidly absorbed. Oral bioavailability is approximately 100%, and close to 99% is bound in plasma. The half-life of diazepam is 43±13 hours,
but ranges from 40-100 hours if the contribution from active metabolites is included. Diazepam is metabolized to nordiazepam which is an active metabolite with a half-life of 40-99 hours. Temazepam and oxazepam are minor active metabolites of diazepam. Diazepam is excreted in urine mainly as oxazepam conjugate (~33 %), and temazepam conjugate, with only traces of diazepam and nordiazepam.

**Molecular Interactions / Receptor Chemistry:** Diazepam is demethylated to nordiazepam via P450 2C19 and 3A4; and 3-hydroxylation to temazepam and oxazepam occurs via P450 3A4. Potential inhibitors of 2C19 and 3A4 could decrease the rate of diazepam elimination if administered concurrently, while potential inducers of these isoenzymes could increase the rate of elimination.

**Blood to Plasma Concentration Ratio:** 0.55 and 0.70 reported; 0.59 for nordiazepam.

**Interpretation of Blood Concentrations:** Simple interpretation of blood concentrations without any knowledge of drug-taking history is ill advised. Given changing responses with repeated use and variability in response, blood concentrations will not provide a good indication of likely behavioral effects. Additionally, the long half-life of diazepam may cause accumulation to occur with repeated use. Blood concentrations may be several-fold higher after chronic use compared to single use, and there are significant increases in blood levels in the elderly.

Therapeutic blood concentrations typically range from 0.1-1.0 mg/L. Single oral doses of 10 mg result in diazepam concentrations of 0.2-0.6 mg/L at 0.5-2 hours, while chronic doses of 30 mg produce steady state diazepam concentrations of 0.7-1.5 mg/L and nordiazepam concentrations of 0.35-0.53 mg/L. Plasma concentrations of 0.3-0.4 mg/L are recommended for anxiolytic effects, and > 0.6 mg/L for control of seizures. Higher concentrations might suggest misuse or abuse.

**Interpretation of Urine Test Results:** Urine concentrations of metabolites are detectable for several days to weeks after last use. Urinary excretion of unchanged drug is less than 1%.

**Effects:** At low doses, diazepam is a moderate tranquilizer, causing sleepiness, drowsiness, confusion, and some loss of anterograde memory. At high doses, excitement, disinhibition, severe sedation, and effects on respiration occur, particularly if respiration is impaired by other drugs or by disease. Diazepam can produce a state of intoxication similar to that of alcohol, including slurred speech, disorientation, and drunken behavior.

**Side Effect Profile:** Side effects may include dry mouth, blurred or double vision, headache, vertigo, urinary retention, excessive perspiration, nausea and vomiting, ataxia, tremor, depression, hypotension and diminished reflexes. The elderly are more likely to develop significant adverse CNS effects from the use of diazepam. In overdose, paradoxical reactions of anxiety, insomnia, stimulation, hallucination, and acute hyperexcited state may occur. Shallow breathing, clammy skin, dilated pupils, weak and rapid pulse, coma, and death are possible.
**Duration of Effects:** Dose-dependent, however, with therapeutic doses onset of effects occurs within 30 minutes and significant effects can last for 12-24 hours.

**Tolerance, Dependence and Withdrawal Effects:** Regular use will produce tolerance to most of the sedative and adverse effects, but tolerance may not occur for the anxiolytic benefits of diazepam. Tolerance may take several weeks or months to develop depending on dose and frequency of administration. Diazepam is capable of causing mild physical and psychological dependence and is regarded as having a significant abuse potential. Abstinence or abrupt withdrawal may produce excitement, restlessness, dysphoria, anxiety, apprehension, fearfulness, dizziness, headache, muscle stiffness, tremors, insomnia, and sensitivity to light and sound. More severe symptoms may include intense rebound nausea, vomiting, abdominal cramps, delirium, hallucinations, hyperthermia, sweating, panic attacks, confusional or paranoid psychoses, tachycardia, increased blood pressure, and occasionally seizures or convulsions.

**Drug Interactions:** Other benzodiazepines, alcohol, phenothiazines, narcotic analgesics, barbiturates, MAOI’s, and other CNS depressants may potentiate action of diazepam. Alcohol enhances such effects as drowsiness, sedation, and decreased motor skills, and can also exacerbate the memory impairing effects of diazepam. Cimetidine delays clearance of diazepam. Valproate may potentiate the CNS depressant effects. Theophylline has an antagonistic action to some of the deleterious effects of diazepam.

**Performance Effects:** Laboratory studies have shown that single doses of diazepam (5-20 mg) are capable of causing significant performance decrements, with maximal effect occurring at approximately 2 hour post dose, and lasting up to at least 3-4 hours. Decreases in divided attention, increases in lane travel, slowed reaction time (auditory and visual), increased braking time, decreased eye-hand coordination, and impairment of tracking, vigilance, information retrieval, psychomotor and cognitive skills have been recorded. Lengthened reaction times have been observed up to 9.5 hours post dose. Lethargy and fatigue are common, and diazepam increases subjective perceptions of sedation. Such performance effects are likely to be exacerbated in the elderly. In drug users, diazepam has greater behavioral changes, including subjects’ rating of liking and decrements in psychomotor and cognitive performance. Reduced concentration, impaired speech patterns and content, and amnesia can also be produced, and diazepam may produce some effects that may last for days. Laboratory studies testing the effect of ethanol on subjects already using benzodiazepines demonstrate further increases in impairment of psychomotor and other driving skills, compared to either drug alone.

**Effects on Driving:** The drug manufacturer suggests patients treated with diazepam be cautioned against engaging in hazardous occupations requiring complete mental alertness such as driving a motor vehicle. Simulator and driving studies have shown that diazepam produces significant driving impairment over multiple doses. Single doses of diazepam can increase lateral deviation of lane control, reduce reaction times, reduce ability to perform multiple tasks, decrease attention, adversely affect memory and cognition, and increase the effects of fatigue. Significant impairment is further increased when diazepam is combined with low concentrations of alcohol (0.05 g/100 mL). A number of
epidemiological studies have been conducted to evaluate the risk of crashes associated with the use of diazepam and other benzodiazepines. These show a range of relative risk, but most demonstrate increases in risk compared to drug free drivers. These increases have been twice to several fold. The elderly may have an increased risk of a motor vehicle crash.

**DEC Category:** CNS depressant

**DEC Profile:** Horizontal gaze nystagmus present; vertical gaze nystagmus present in high doses; lack of convergence present; pupil size normal; reaction to light slow; pulse rate down; blood pressure down; body temperature normal. Other characteristic indicators may include behavior similar to alcohol intoxication without the odor of alcohol, staggering and stumbling, lack of balance and coordination, slurred speech, disorientation, and poor performance on field sobriety tests.

**Panel’s Assessment of Driving Risks:** The incidences of diazepam in drivers involved in road crashes and in drivers suspected of being under the influence, suggest an adverse effect of diazepam on road safety. Data are available to demonstrate that single therapeutic doses of diazepam can significantly impair psychomotor skills associated with safe driving, with some effects still observable the morning after a nighttime dose.

**References and Recommended Reading:**

- 32 -


Diphenhydramine
Diphenhydramine is a white, crystalline powder. Available primarily in tablet, capsule and liquid form.

**Synonyms:** 2-(diphenylmethoxy)-N,N-dimethylethylamine hydrochloride; diphenhydramine hydrochloride; Benadryl®, Unisom® Sleepgels, Dytuss®, Dramamine®.

**Source:** Available in capsules, tablets, chewable tablets, syrups, elixirs, topical, and injectable forms in a variety of prescription and over-the-counter medications. Products contain diphenhydramine alone or in combination with other drugs such as pseudoephedrine and acetaminophen. Diphenhydramine is also an ingredient in several Tylenol® (i.e., acetaminophen) preparations. Dimenhydrinate (Dramamine®) is a combination of diphenhydramine and 8-chlorotheophylline in equal molecular proportions.

**Drug Class:** Antihistamine, antiemetic, sleep aid, sedative, CNS depressant.

**Medical and Recreational Uses:** Used as an antihistamine for the temporary relief of seasonal and perennial allergy symptoms. Diphenhydramine is also used as a sleep aid and a cough suppressant, and has been used as a centrally acting antitussive although the mechanism for this action is unclear. Dramamine is used as a prophylaxis against and for the treatment of motion sickness.

**Potency, Purity and Dose:** As an antihistamine, recommended doses for adults is 25-50 mg diphenhydramine every 6-8 hours, not to exceed 50-100 mg every 4-6 hours. For children, 12.5-25 mg three or four times daily is recommended. As a sleep aid the dose is 50 mg at bedtime. Adults can be given 10-50 mg intravenously or intramuscularly, up to a maximum daily dose of 400 mg.

**Route of Administration:** Oral, injected, and topical applications.

**Pharmacodynamics:** Diphenhydramine is a first generation antihistamine and is a H₁ receptor antagonist. Antagonism is achieved through blocking the effect of histamine more than blocking its production or release. Diphenhydramine inhibits most responses of smooth muscle to histamine and the vasoconstrictor effects of histamine. The antagonism may also produce anticholinergic effects, antiemetic effects, and significant sedative side effects.

**Pharmacokinetics:** Following oral administration diphenhydramine is well absorbed from the gastrointestinal tract, is widely distributed throughout the body, and is able to pass though the blood-brain barrier. The oral availability is 61%, and 78% is bound in plasma. Peak plasma concentrations are reached in 2-3 hours. Diphenhydramine is metabolized to nordiphenhydramine (active metabolite), dinordiphenhydramine, and diphenylmethoxyacetic acid. The plasma half-life is 8.5±3.2 hours; shorter and longer
half-lives have been reported for children and elderly subjects, respectively. Urinary
excretion of unchanged diphenhydramine is 1.9%.

Molecular Interactions / Receptor Chemistry: Diphenhydramine is metabolized via
cytochrome P450 2D6 isoenzyme. Potential inhibitors of P450 2D6 could decrease the
rate of drug elimination if administered concurrently, while potential inducers could
increase the rate of drug elimination.

Blood to Plasma Concentration Ratio: 0.77 and 0.82 reported.

Interpretation of Blood Concentrations: Following a single oral dose of 50 mg,
average peak plasma concentrations of 83 ng/mL diphenhydramine were detected at 3
hours, declining to 9 ng/mL by 24 hours. A single oral 100 mg dose resulted in average
peak plasma concentrations of 112 ng/mL at 2 hours post dose. Effective antihistamine
concentrations are greater than 25 ng/mL, drowsiness can be observed at 30-40 ng/mL,
and mental impairment may be observed with concentrations above 60 ng/mL.

Interpretation of Urine Test Results: Less than 2% of an oral dose is excreted in the 24
hour urine as unchanged parent drug, while approximately 11% is eliminated as its
glucuronide conjugate.

Effects: First generation H1 antagonists can both stimulate and depress the CNS.
Stimulation results in restlessness, nervousness and inability to sleep, while depressive
effects include diminished alertness, slowed reaction time and somnolence.
Diphenhydramine is particularly prone to cause marked sedation. Drowsiness, reduced
wakefulness, altered mood, impaired cognitive and psychomotor performance may also
be observed.

Side Effect Profile: Includes agitation, anticholinergic side effects such as dry mouth,
confusion, dizziness, drowsiness, fatigue, disturbed coordination, irritability, paresthesia,
blurred vision, and depression. In overdose, symptoms may include excitement, ataxia,
tremor, sinus tachycardia, fever, hallucination, athetosis, convulsions or seizures,
hypotension, deep coma, cardiorespiratory collapse, and death. Fixed and dilated pupils
are also observed. Gastrointestinal symptoms are less with diphenhydramine than with
other H1 antagonists.

Duration of Effects: Dose-dependent, however, following oral administration of
therapeutic doses, peak plasma concentrations are reached in 2-3 hours and effects
usually last 4-6 hours.

Tolerance, Dependence and Withdrawal Effects: Some tolerance may develop to the
sedative effects of diphenhydramine with repeated oral dosing. No reported dependence
or withdrawal effects with doses recommended.

Drug Interactions: Effects of diphenhydramine are increased by the presence of
alcohol, MAOI’s, diazepam, hypnotics, sedatives, tranquilizers, and other CNS
depressants. Alcohol enhances such effects as drowsiness, sedation and decreased motor skills. These decrements in effect are more pronounced in the elderly. MAOI’s prolong and intensify the anticholinergic effects of diphenhydramine.

**Performance Effects:** All first generation antihistamines, including diphenhydramine, have been demonstrated to diminish cognitive and psychomotor performance in healthy volunteers. Impairment might even be of greater clinical significance in patients when the allergic disorder per se adversely affects CNS function, as suggested in studies in which a reduction in cognitive functioning in patients was exacerbated by diphenhydramine. Laboratory studies have shown diphenhydramine to decrease alertness, decrease reaction time, induce somnolence, impair concentration, impair time estimation, impair tracking, decrease learning ability, and impair attention and memory within the first 2-3 hours post dose. Significant adverse effects on vigilance, divided attention, working memory, and psychomotor performance have been demonstrated. It is important to note that impairment has been shown to occur even in the absence of self-reported sleepiness or sedation. Concurrent use of diazepam and diphenhydramine caused significant performance decrements at 2 hours, and to some degree up to 4 hours.

**Effects on Driving:** The drug manufacturer states that patients should be warned about engaging in activities requiring mental alertness such as driving a car. Diphenhydramine has repeatedly been shown to severely impair tracking and reaction time performance in actual on-the-road driving tests. Single doses of 50 mg have been shown to cause significant impairment during a 90 km highway test (measuring vehicle following, constant speed and lateral position). In contrast, single 25-100 mg doses caused no significant driving effects during a short 15 minute driving test. Using the Iowa Driving Simulator, Weiler et al, 2000 compared the effects of a single oral dose of 50 mg diphenhydramine to the effects corresponding to a blood alcohol concentration of 0.1 g/100 mL. Diphenhydramine caused significantly less coherence (ability to maintain a constant distance) and impaired lane keeping (steering instability and crossing center line) compared to alcohol. Overall driving performance was the poorest after taking diphenhydramine, and participants were most drowsy after taking diphenhydramine (before and after testing). The authors concluded that diphenhydramine clearly impairs driving performance, and may have an even greater impact than does alcohol on the complex task of operating a motor vehicle.

**DEC Category:** CNS depressant

**DEC Profile:** Data not available; however, the profile for a CNS depressant is: horizontal gaze nystagmus present; vertical gaze nystagmus present at high doses; lack of convergence present; pupil size normal; reaction to light slow; pulse rate normal; blood pressure normal; body temperature normal. Diphenhydramine may produce dilated pupils.

**Panel’s Assessment of Driving Risks:** Single therapeutic doses of diphenhydramine have been shown to significantly impair psychomotor performance during the first 4 hours, and may have a greater impact on driving performance than alcohol.
References and Recommended Reading:
Gamma-Hydroxybutyrate (GHB, GBL, and 1,4-BD)

GHB is a clear liquid, or a white powder with a soap-like texture. Precursor drugs such as gamma-butyrolactone (GBL) and 1,4 butanediol (1,4-BD) are clear liquids.

**Synonyms:**
- **GHB:** Sodium oxybate, Xyrem® oral solution; liquid X, liquid XTC, salt water, scoop, soap, grievous bodily harm, georgia home boy, G, G-caps, easy lay, everclear, vita G, degreaser + lye, smart drug, gamma-OH, Somatomax.
- **GBL:** 2(3)-furanone dihydro; Blue Nitro, G3, Invigorate, Jolt, ReActive, REMForce, RenewTrient, Rest-eze, Revivarant, Verve, V35.
- **1,4-BD:** tetramethylene glycol; Amino Flex, Enliven, FX, GHRE, Inner G, NRG3, Pine Needle Extract, Revitalize, Serenity, SomatoPro, Thunder Nectar, Zen.

**Source:** GHB was first synthesized in 1960 as an experimental GABA analog, and was classified as a food and dietary supplement and sold in health food stores in early 1990. It was available in tablet, capsule and liquid forms. In late 1990, the FDA banned over-the-counter sales of GHB in the U.S. In 1999, the FDA issued warnings on the dangers of its precursor drugs GBL and 1,4-BD. In early 2000, GHB was federally reclassified as a Schedule 1 controlled substance. GBL and 1,4-BD are not scheduled, however, GBL is classified as a list 1 chemical and a controlled substance analog, while 1,4-BD is listed as a controlled substance analog. GHB can be clandestinely made and the ingredients are available in kit form over the internet. GHB is made from GBL and a base (e.g. lye/NaOH), the mixture is heated, and vinegar is added to reduce the pH. Acetone can then be added and the mixture dried, resulting in GHB powder. GBL and 1,4-BD are commercially available as industrial solvents and are used as ingredients in cleaners, solvents, paint removers, and engine degreasers. They are also sold as “natural supplements” over the internet, and in some health food stores and gymnasiums, and are marketed as natural, non-toxic dietary supplements.

**Drug Class:** CNS depressant, sedative, anesthetic.

**Medical and Recreational Uses:** In Europe, GHB is used as an anesthetic adjunct and hypnotic agent, used to treat narcolepsy, and used to suppress symptoms of alcohol-dependence and opiate withdrawal syndrome. In the U.S., medically formulated sodium oxybate (Xyrem®) has been approved as a Schedule III controlled substance for the treatment of cataplexy (sudden loss of muscle tone associated with narcolepsy). Recreationally, GHB is used for its intoxicating effects (euphoria, reduced inhibitions, sedation), and by bodybuilders as an alternative to anabolic steroids. GBL and 1,4-BD rapidly convert to GHB within the human body following oral administration and are taken as GHB substitutes. They are marketed as anti-aging drugs, for weight loss, to treat insomnia, anxiety and depression, and as mood enhancers and energizers.

**Potency, Purity and Dose:** Clinical doses for alcohol withdrawal syndrome are 25-50 mg/kg every 12 hours (1.7-3.5 g/70 kg); sleep induction 20-30 mg/kg (1.5-2.25 g/70 kg); prolonged deep sleep 75-100 mg/kg (5-7 g/70 kg); and anesthetic induction greater than 100 mg/kg (> 7 g/70 kg). Illicit manufacture often introduces impurities and wide
variations in potency. Recreational use of GHB often involves doses well in excess of one teaspoon (~2.5 g, or 35 mg/kg in a 70 kg adult) of the powder dissolved in water/alcohol, or one capful of liquid GHB, GBL, or 1,4-BD; such doses far exceed therapeutic doses. Chronic use can consist of dosing every few hours, around the clock, for months to years. Up to 100 g GHB has been reportedly used by an individual in one day. GHB and its precursor drugs are often used in combination with alcohol, MDMA, marijuana, methamphetamine, and cocaine.

**Route of Administration:** Oral, intravenous.

**Pharmacodynamics:** GHB is a naturally occurring compound present in both mammalian CNS and peripheral tissue. It is also a minor metabolite and precursor of the major inhibitory neurotransmitter GABA. GHB is also the pharmacologically active form of both GBL and 1,4-BD. GHB has weak agonist activity at GABAB receptors and there appears to be a distinct GHB receptor site in the brain. GHB dose-dependently alters dopaminergic activity; at sub-anesthetic doses there is an initial excitation of dopamine neurons producing elevated levels of synaptic dopamine; at anesthetic doses GHB blocks impulse flow from dopamine neurons resulting in a build-up of dopamine in the nerve terminals. GHB mimics natural physiological sleep, enhances REM sleep, and increases stage 3 and 4 of slow-wave sleep. GHB decreases alcohol consumption and intensity of withdrawals. Beyond the CNS effects, GHB has significant cardiovascular pharmacology, causing bradycardia and dysregulation of blood pressure (hyper- and hypotension). Interestingly, GHB causes a detectable increase in growth hormone and prolactin concentrations with doses as small as 3 g, and this is the basis for its use in body building despite there being no evidence of an actual increase in body mass.

**Pharmacokinetics:** Oral doses are rapidly absorbed from the gastrointestinal tract and exhibit first pass metabolism. Absorption is capacity limited (an increase in dose results in increased time to peak concentration). There is an increased rate of absorption of GHB on an empty stomach leading to a decreased time to peak concentration and an increased concentration. Accumulation is not known to occur following repeated doses. GHB readily crosses the blood-brain barrier and placental barrier, and is distributed in the brain, cerebrospinal fluid, vitreous, liver, and kidney. The dose-response curve is steep, and a large between and within subject variability is noted. GHB is rapidly eliminated and has a half-life of 27 minutes (range 20-53 minutes) which appears to increase with higher doses, a sign of zero order or saturation kinetics. GHB is metabolized to succinic semialdehyde (SSA) via GHB-dehydrogenase, then to succinic acid via SSA-dehydrogenase. GBL is metabolized to GHB via lactonase; while 1,4-BD is first metabolized to γ-hydroxybutyraldehyde via alcohol dehydrogenase, then to GHB via aldehyde dehydrogenase.

**Molecular Interactions / Receptor Chemistry:** Metabolism via cytochrome P450 isoenzymes has not been described.

**Blood to Plasma Concentration Ratio:** 1.2 (N=1)
**Interpretation of Blood Concentrations:** Peak plasma concentrations are observed at 20-45 minutes. Due to rapid elimination, GHB is undetectable in plasma or blood after 6-8 hours. Following single oral doses of 25 mg/kg GHB in 10 alcoholic dependant patients, mean peak plasma GHB concentrations were 54 mg/L (24-88 mg/L). Single oral doses of 12.5, 25, and 50 mg/kg in 8 healthy subjects produced mean peak plasma GHB concentrations of 23, 46 and 80 mg/L, respectively. Single oral doses of 26-52 mg/kg in 6 narcoleptic patients resulted in mean peak plasma GHB concentrations of 63 mg/L (30-102 mg/L). The same doses were administered to the same subjects 4 hours later, and the mean peak GHB concentrations obtained were 91 mg/L (47-125 mg/L). An intravenous dose of 50 mg/kg in an adult produced a peak blood GHB concentration of approximately 170 mg/L within 15 minutes. Patients presenting to an emergency department with GHB overdose/intoxication, had blood GHB concentrations ranging from 29-432 mg/L (mean 118 mg/L; N = 54).

Although GHB is naturally present in the human body, endogenous blood GHB concentrations are typically well below 1 mg/L in living subjects. In contrast, endogenous postmortem production of GHB can occur, and concentrations of up to 170 mg/L GHB have been reported in non-GHB using subjects. In postmortem analysis the analysis of multiple specimens such as vitreous and urine is recommended.

**Interpretation of Urine Test Results:** Peak urine concentrations are observed within 4 hours of administration and GHB is undetectable in urine after 10-12 hours. Endogenous concentrations of up to ~7 mg/L GHB have been detected in urine of non-GHB using subjects. It is suggested that a cut-off for urinary GHB be set at 10 mg/L. Similarly, in postmortem urine specimens from non-GHB using subjects, urine concentrations of GHB are typically below 10 mg/L.

**Effects:**
*Psychological:* At low doses, effects are similar to those seen with alcohol. Effects include relaxation, reduced inhibitions, euphoria, confusion, dizziness, drowsiness, sedation, inebriation, agitation, combativeness, and hallucinations.

*Physiological:* Nausea, vomiting, profuse sweating, somnolence, visual disturbances, nystagmus, loss of peripheral vision, short-term amnesia, uncontrolled shaking or seizures, bradycardia, hypothermia, suppression of gag reflex, respiratory depression, and transient or unarousable unconsciousness.

**Side Effect Profile:** Disorientation, sweating, vomiting, incontinence, apnea, severe ataxia, sinus bradycardia, twitching, seizure-like activity and hypothermia. In overdose, symptoms may include severe respiratory depression, mild acute respiratory acidosis, sinus bradycardia or sinus tachycardia, suppression of gag reflex, acute delirium, combativeness, unarousable unconsciousness, coma, and patients often need to be intubated. Deaths have been reported following overdose from GHB, GBL and 1,4-BD alone, and in combination with other drugs.

**Duration of Effects:** Onset of effects occurs within 10-20 minutes, peak plasma concentrations are achieved within 20-45 minutes, and effects generally last 2-5 hours. Complete recovery from GHB overdose can occur within 3-6 hours. Sleep induction time
is shortest with GBL and longest with 1,4-BD, as GBL is more lipophilic and is absorbed faster. There is a longer duration of effect following 1,4-BD ingestion as it metabolizes more slowly to GHB than does GBL.

**Tolerance, Dependence and Withdrawal Effects:** Tolerance can develop to GHB with chronic abuse and even following chronic treatment. Subjects do not become tolerant to all the effects (e.g. tolerance does not develop to the enhanced sleep that GHB produces). Cross-tolerance exists between GHB and ethanol. Severe physical and psychological addiction occurs with chronic abuse. Clinical presentation of withdrawal may include mild clinical anxiety, confusion, agitation, tremor, muscular cramps, insomnia, combativeness, delirium, delusions, paranoia with hallucinations (auditory, tactile and visual), tachycardia, hypotension, and an occasional schizophrenic-like state. The withdrawal syndrome can start as early as 1-2 hours after the last dose in addicted individuals.

**Drug Interactions:** Potential additive effects between GHB and other sedating CNS depressants, including alcohol, antidepressants, antipsychotics, antihistamines and muscle relaxants. In rats, ethanol has significant synergistic effects on the sedative, behavioral and toxic effects of GHB, GBL and 1,4-BD. Ethanol also delays the conversion of 1,4-BD to GHB, because both 1,4-BD and ethanol utilize alcohol-dehydrogenase in their metabolic pathways. Several drugs have been shown to inhibit GHB-dehydrogenase and it is not known clinically what effects these drugs would have if administered concurrently. These drugs include valproate, ethosuximide, salicylate, amobarbital, phenytoin, disulfiram and cyanide.

**Performance Effects:** Oral GHB doses of 1-2 g have been shown not to deteriorate reactive, attentive and co-ordination skills related to driving, nor increase the effects of low dose alcohol. Similarly, oral doses of 12.5-25 mg/kg GHB had no effect on attention, vigilance, alertness, short-term memory or psychomotor coordination; although dizziness or dullness were experienced in 50-66% of subjects. It is important to note, however, that doses used in laboratory studies to date have been well below both recreational and abused doses of GHB.

**Effects on Driving:** Signs of behavioural effects and impaired performance have been reported in several driving case reports. In 13 driving under the influence cases where GHB was detected, the reported symptoms were generally those of a CNS depressant. The subjects were typically stopped because of erratic driving, such as weaving, ignoring road signs, and near-collisions. Common signs of impairment included confusion and disorientation, incoherent speech, short-term memory loss, dilated pupils, lack of balance and unsteady gait, poor coordination, poor performance of field sobriety tests, copious vomiting, unresponsiveness, somnolence, and loss of consciousness. GHB concentrations in blood specimens collected between 1-3.5 hours of the arrest ranged from 26-155 mg/L (median 95 mg/L). In another 11 cases of driving under the influence of GHB, concentrations of GHB in blood and urine specimens ranged from 81-360 mg/L and 780-2380 mg/L, respectively. Circumstances of their arrest, observed driving behavior and signs of impairment were similar to the previous study. Other reported symptoms have
included dizziness, drowsiness, agitation, loss of peripheral vision, slow responses, slow and slurred speech, and transient unconsciousness.

**DEC Category:** CNS depressant

**DEC Profile:** Horizontal gaze nystagmus present; vertical gaze nystagmus present in high doses; lack of convergence present; pupil size generally dilated; reaction to light slow; pulse rate normal; blood pressure normal; body temperature generally down. Other characteristic indicators include vomiting, sweating, slurred speech, somnolence or transient unconsciousness, poor balance and coordination, and poor performance on field sobriety tests. Note that while pulse rate and blood pressure may decrease after GHB ingestion, both parameters may be elevated during drug withdrawal.

**Panel’s Assessment of Driving Risks:** Given the ability of GHB to induce sleep and unconsciousness, recreational use of GHB or its precursor drugs have the potential to produce moderate to severe driving impairment.

**References and Recommended Reading:**
**Ketamine**

Ketamine is a white, crystalline powder or clear liquid.

**Synonyms:** (+/-)-2-(2-chlorophenyl)-2-(methylamino)-cyclohexanone; Ketalar®, Ketaject®, Ketaset®, Vetalar®; K, Special K, Vitamin K, Lady K, Jet, Super Acid, Bump, Special LA Coke, KitKat, Cat Valium.

**Source:** Available by prescription only, and is commercially available as a veterinary anesthetic. It is difficult to synthesize clandestinely and is usually stolen from veterinarian offices or diverted from legitimate pharmaceutical sources in liquid form. Ketamine is currently a schedule III controlled substance in the US.

**Drug Class:** Dissociative anesthetic, hallucinogen, psychotomimetic.

**Medical and Recreational Uses:** Primarily used in veterinary applications as a tranquilizer. Also used as an anesthetic induction agent for diagnostic and surgical procedures in humans, prior to the administration of general anesthetics. Occasionally used as a short-acting general anesthetic for children and elderly patients. Recreationally used as a psychedelic and for its dissociative effects.

**Potency, Purity and Dose:** Ketamine is available as a racemic mixture with the S- (+)- isomer being more potent than the R-(-)- isomer. Commercially supplied as the hydrochloride salt in 0.5 mg/mL and 5 mg/mL ketamine base equivalents. For induction of 5-10 minutes surgical anesthesia, a dose of 1.0-4.5 mg/kg is intravenously administered; 6.5-13 mg/kg is given intramuscularly for 12-25 minutes of surgical anesthesia. The liquid from injectable solutions can be gently heated to evaporate the water, leaving a white powder (ketamine hydrochloride) which can be snorted or orally ingested. Recreational doses are highly variable. Common doses are 25-50 mg intramuscularly, 30-75 mg snorting, and 75-300 mg oral. Snorting a small line (“bump”, 30-50 mg) usually results in a dreamy effect. “K-hole” can be obtained following a dose of 60-125 mg intramuscularly, or by snorting 100-250 mg. Impurities are rarely seen, although ketamine hydrochloride itself can be used as a heroin adulterant.

**Route of Administration:** Injected, snorted, orally ingested, and rectally administered. Similar to phencyclidine (PCP), ketamine can be added to tobacco or marijuana cigarettes and smoked.

**Pharmacodynamics:** Involves analgesia, anesthetic and sympathomimetic effects that are mediated by different sites of action. Non-competitive NMDA receptor antagonism is associated with the analgesic effects; opiate receptors may contribute to analgesia and dysphoric reactions; and sympathomimetic properties may result from enhanced central and peripheral monoaminergic transmission. Ketamine blocks dopamine uptake and therefore elevates synaptic dopamine levels. Inhibition of central and peripheral cholinergic transmission could contribute to induction of the anesthetic state and hallucinations. Ketamine is structurally similar to PCP, but 10-50 times less potent in blocking NMDA effects.
**Pharmacokinetics:** Bioavailability following an intramuscular dose is 93%, intranasal dose 25-50%, and oral dose 20±7%. Ketamine is rapidly distributed into brain and other highly perfused tissues, and is 12% bound in plasma. The plasma half-life is 2.3 ± 0.5 hours. Oral administration produces lower peak concentrations of ketamine, but increased amounts of the metabolites norketamine and dehydronorketamine. Ketamine and its metabolites undergo hydroxylation and conjugation. Norketamine produces effects similar to those of ketamine. There are no significant differences between the pharmacokinetic properties of the S-(+) and R-(-)-isomers.

**Molecular Interaction / Receptor Chemistry:** Cytochrome P450 3A4 is the principal enzyme responsible for ketamine N-demethylation to norketamine, with minor contributions from CYP2B6 and CYP2C9 isoforms. Potential inhibitors of these isoenzymes could decrease the rate of ketamine elimination if administered concurrently, while potential inducers could increase the rate of elimination.

**Blood to Plasma Concentration Ratio:** Data not available.

**Interpretation of Blood Concentrations:** There is no direct correlation between ketamine concentrations and behavior. Drowsiness, perceptual distortions and intoxication may be dose related in a concentration range of 50 to 200 ng/mL, and analgesia begins at plasma concentrations of about 100 ng/mL. During anesthesia, blood ketamine concentrations of 2000-3000 ng/mL are used, and patients may begin to awake from a surgical procedure when concentrations have been naturally reduced to 500-1000 ng/mL.

**Interpretation of Urine Test Results:** Urinary excretion of unchanged drug is 4±3%, and ketamine use can be detected in urine for about 3 days. Concentration ranges for ketamine in urine have been reported as low as 10 ng/mL and up to 25,000 ng/mL.

**Effects:** Users have likened the physical effects of ketamine to those of PCP, and the visual effects to LSD.

**Psychological:** Decreased awareness of general environment, sedation, dream-like state, vivid dreams, feelings of invulnerability, increased distractibility, disorientation, and subjects are generally uncommunicative. Intense hallucinations, impaired thought processes, out-of-body experiences, and changes in perception about body, surroundings, time and sounds. Delirium and hallucinations can be experienced after awakening from anesthesia.

**Physiological:** Anesthesia, cataplexy, immobility, tachycardia, increased blood pressure, nystagmus, hypersalivation, increased urinary output, profound insensitivity to pain, amnesia, slurred speech, and lack of coordination.

**Side Effect Profile:** High incidence of adverse effects, including anxiety, chest pain, palpitations, agitation, rhabdomyolysis, flashbacks, delirium, dystonia, psychosis, schizophrenic-like symptoms, dizziness, vomiting, seizures, and paranoia.
**Duration of Effects:** Onset of effects is within seconds if smoked, 1-5 minutes if injected, 5-10 minutes if snorted and 15-20 minutes if orally administered. Effects generally last 30-45 minutes if injected, 45-60 minutes if snorted, and 1-2 hours following oral ingestion. Ketamine is often readministered due to its relatively short duration of action. Some subjects may experience dreams 24 hours later. Marked dissociative effects, schizotypal symptoms and impaired semantic memory are found in some recreational users days after drug use.

**Tolerance, Dependence and Withdrawal Effects:** In long-term exposure, high tolerance, drug craving, and flashbacks are described. Little evidence of a physiological withdrawal syndrome unless abrupt discontinuation in chronic users.

**Drug Interactions:** Midazolam attenuates altered perception and thought processes. Lorazepam may decrease ketamine-associated emotional distress but does not decrease cognitive or behavioral effects of ketamine. Acute administration of diazepam increases the half-life of ketamine. Lamotrigine significantly decreases ketamine-induced perceptual abnormalities, but increases the mood elevating effects. Haloperidol may decrease impairment by ketamine in executive control functions, but does not affect psychosis, perceptual changes, negative schizophrenic-like symptoms, or euphoria. Alfentanil is additive to ketamine in decreasing pain and increasing cognitive impairment. Physostigmine and 4-aminopyridine can antagonize some pharmacodynamic effects of ketamine.

**Performance Effects:** Broad spectrum of cognitive impairments and marked dissociative effects. Increased distractibility and intensely visual or polysensual hallucinations. Impairment of immediate and delayed recall, and verbal declarative memory. Memory impairment is associated with encoding or retrieval processes, and not accounted for by decreased attention. Impaired language function, failure to form and use memory traces of task relevant information. Overall decreased awareness, increased reaction time, distorted perceptions of space, non-responsiveness, and blurred vision. The S-(+)-isomer impairs psychomotor function 3-5 times more than the R-(-)-isomer.

**Effects on Driving:** The drug manufacturer suggests that patients should be cautioned that driving an automobile should not be undertaken for 24 hours or more following anesthesia. No driving studies have been performed.

**DEC Category:** Phencyclidine.

**DEC Profile:** Horizontal gaze nystagmus present; vertical gaze nystagmus present; lack of convergence present; pupil size normal; reaction to light normal; pulse rate elevated; blood pressure elevated; body temperature elevated. Other characteristic indicators may include rigid muscles, cyclic behavior, and lack of response to painful stimuli.

**Panel’s Assessment of Driving Risks:** The use of ketamine is not conceivably compatible with the skills required for driving due to its moderate to severe psychomotor, cognitive, and residual effects.
References and Recommended Reading:


Lysergic acid diethylamide (LSD)
LSD is a white powder or a clear, colorless liquid.

**Synonyms:**  
d-lysergic acid diethylamide; acid, animal, barrels, beast, blotter, ‘cid, dots, kool aid, LSD-25, lysergide, microdots, panes, sandoz, tabs, trips, white lightning, window panes.

**Source:** LSD is manufactured from lysergic acid which occurs naturally in the ergot fungus that grows on wheat and rye. It is a Schedule I controlled substance, available in liquid, powder, tablet (microdots), and capsule form. The liquid is often applied to blotter paper squares (frequently with colorful designs), stickers, sugar cubes, candy, or soda crackers. LSD is also available in dropper bottles or in the form of gelatin sheets/shapes (window panes).

**Drug Class:** Hallucinogen, psychedelic, psychotomimetic.

**Medical and Recreational Uses:** No medicinal use. Recreationally used as a hallucinogen and for its ability to alter human perception and mood.

**Potency, Purity and Dose:** The strength of illicit LSD nowadays ranges from 20 to 80 µg per dose, which is considerably less than doses reported during the 1960s and early 1970s, of 100-200 µg or higher per unit. Experienced users typically administer 100-200 µg for a “good high”. The potency of liquid LSD in dropper bottles may vary because the liquid is water based.

**Route of Administration:** Primarily oral administration, but can be inhaled, injected, and transdermally applied.

**Pharmacodynamics:** LSD is primarily a non-selective 5-HT agonist. LSD may exert its hallucinogenic effect by interacting with 5-HT$_{2A}$ receptors as a partial agonist and modulating the NMDA receptor-mediated sensory, perceptual, affective and cognitive processes. LSD mimics 5-HT at 5-HT$_{1A}$ receptors, producing a marked slowing of the firing rate of serotonergic neurons.

**Pharmacokinetics:** LSD has a plasma half-life of 2.5-4 hours. Metabolites of LSD include N-desmethyl-LSD, hydroxy-LSD, 2-oxo-LSD, and 2-oxo-3-hydroxy-LSD. These metabolites are all inactive.

**Molecular Interactions / Receptor Chemistry:** Metabolism via cytochrome P450 isoenzymes has not been described.

**Blood to Plasma Concentration Ratio:** Data not available.

**Interpretation of Blood Concentrations:** Threshold toxic dose in humans has been reported with 100-200 µg with associated blood concentrations of 2-30 ng/mL. Intravenous doses of 1-2 µg /kg have been associated with blood concentrations of 1-5
ng/mL LSD. Single oral doses of 160 µg resulted in peak plasma concentrations of up to 9 ng/mL LSD.

*Interpretation of Urine Test Results:* LSD use can typically be detected in urine for periods of 2-5 days. In a reported case of LSD intoxication, a concentration of 11 ng/mL of LSD was detected in the urine. In subjects receiving 200-400 µg of LSD, concentrations in urine ranged from 1-55 ng/mL.

**Effects:** Effects are unpredictable and will depend on the dose ingested, the user’s personality and mood, expectations and the surroundings. *Psychological:* Hallucinations, increased color perception, altered mental state, thought disorders, temporary psychosis, delusions, body image changes, and impaired depth, time and space perceptions. Users may feel several emotions at once or swing rapidly from one emotion to another. “Bad trips” may consist of severe, terrifying thoughts and feelings, fear of losing control, and despair. *Physiological:* Tachycardia, hypertension, dilated pupils, sweating, loss of appetite, sleeplessness, dry mouth, tremors, speech difficulties, and piloerection.

*Side Effect Profile:* Rhabdomyolysis, renal failure, prolonged mania, panic, impairment in color discrimination, and residual visual effects have been described. LSD users may manifest relatively long-lasting psychoses, such as schizophrenia or severe depression.

*Duration of Effects:* Onset of effects is rapid following intravenous administration (10 minutes). Following oral ingestion, onset of the first effects are experienced in 20-30 minutes, peaking at 2-4 hours and gradually diminishing over 6-8 hours. Residual effects may last longer. Flashbacks may occur suddenly, often without warning, and may occur within a few days or more than a year after use.

*Tolerance, Dependence and Withdrawal Effects:* Frequent, repeated doses of LSD are unusual and therefore tolerance is not commonly seen. Tolerance does develop to the behavioral effects after 3-4 daily doses, but no withdrawal syndrome has been described. LSD is not considered an addictive drug since it does not produce compulsive drug-seeking behavior.

*Drug Interactions:* Cross-tolerance with mescaline and psilocybin has been demonstrated in animal models. LSD blocks subjective alcohol effects in many subjects. Possible seizures when concurrently taken with lithium or fluoxetine.

*Performance Effects:* LSD produces significant psychedelic effects with doses as little as 25-50 µg. LSD impairs reaction time (auditory and visual), choice reaction time, and visual acuity for up to 4 hours. Impaired divided attention, ataxia, and grossly distorted perception have also been reported following LSD use.

*Effects on Driving:* Epidemiology studies suggest the incidence of LSD in driving under the influence cases is extremely rare. In Denver, Colorado between Jan 1988 to June 1990, 242 drivers detained for driving while impaired were evaluated by drug
recognition examiners; only 1 case of LSD was confirmed following urine toxicology screens.

**DEC Category:** Hallucinogen.

**DEC Profile:** Horizontal gaze nystagmus not present; vertical gaze nystagmus not present; lack of convergence not present; pupil size dilated; reaction to light normal; pulse rate elevated; blood pressure elevated; body temperature elevated. Other characteristic indicators may include extreme changes in behavior and mood, trance-like state, sweating, body tremors, piloerection, hallucinations, paranoia, and changes in sense of light, hearing, touch and smell.

**Panel’s Assessment of Driving Risks:** The use of LSD is not compatible with the skills required for driving due to its severe psychomotor, cognitive and residual effects.

**References and Recommended Reading:**


Methadone
Methadone hydrochloride is a white crystalline powder or colorless crystals. Available primarily in tablet or liquid form.

Synonyms: 6-dimethylamino-4,4-diphenyl-3-heptanone; Dolophine® Hydrochloride, Methadose®, Methadone Hydrochloride Intensol™.

Source: Methadone is a synthetic narcotic analgesic and is a schedule II controlled substance. Methadone is available by prescription as oral solutions (1-2 mg/mL strength), tablets (5-10 mg), dispersible tablets (40 mg), or injectable solutions (10 mg/mL).

Drug Class: Narcotic analgesic.

Medical and Recreational Uses: Methadone is an analgesic prescribed for the relief of moderate to severe pain, and is used in detoxification treatment of opioid dependence and maintenance in narcotic addiction. Compared to morphine, methadone has a much longer duration of action, suppressing opiate withdrawal symptoms and remaining efficacious for an extended period of time with repeated administration. Recreationally, methadone is abused for its sedative and analgesic effects.

Potency, Purity and Dose: Available as the racemic mixture, (R)- or l-methadone is 8-50 times more potent than the (S)- or d-isomer. For relief of severe acute pain the usual adult dose is 2.5-10 mg every 3-4 hours. For methadone maintenance the daily dose is generally 60-80 mg, but can vary from 30-120 mg. For detoxification treatment an initial oral dose of 15-20 mg is administered, with an additional dose if withdrawal symptoms are not suppressed; a stabilizing dose of 40 mg in single or divided dosages is prescribed for 2-3 weeks, then the dose is gradually decreased. Concurrent use of other prescription medication is common.

Route of Administration: Oral ingestion, intravenous, intramuscular or subcutaneous injection.

Pharmacodynamics: Methadone is a long acting µ opioid receptor agonist with potent central analgesic, sedative, and antitussive actions. Methadone inhibits ascending pain pathways, alters perception of and response to pain (dissociative effect), and produces generalized CNS depression. Respiratory depression also occurs due to complete blockade of respiratory centers to pCO2. (S)-Methadone lacks significant respiratory depressive action and addiction liability.

Pharmacokinetics: When administered orally, methadone is rapidly absorbed from the gastrointestinal tract and can be detected in the blood within 30 minutes. Oral bioavailability varies from 41-99% and plasma protein binding is 60-90%. After repeated administration there is gradual accumulation in tissues. As for most lipid soluble drugs, a large between and within subject variability is observed. The half-life of (R,S)-methadone is 15-60 hours, and 10-40 hours for (R)-methadone. Methadone undergoes extensive biotransformation in the liver primarily to two inactive metabolites,
2-ethylidene-1.5-dimethyl-3.3-diphenylpyrrolidine (EDDP) and 2-ethyl-5-methyl-3,3-diphenyl-1-pyrroline (EMDP), through N-demethylation and cyclization. These are eliminated by the kidney and excreted through the bile. In total, nine metabolites have been identified including two minor active metabolites, methadol and normethadol.

**Molecular Interactions / Receptor Chemistry:** Methadone is metabolized to EDDP via the cytochrome P450 CYP3A4 isoform. Potential inhibitors of this isoform could decrease the rate of methadone elimination if administered concurrently, while potential inducers could increase the rate of elimination. Methadone itself inhibits cytochrome P450 2D6 isoform.

**Blood to Plasma Concentration Ratio:** 0.75 and 0.77 reported.

**Interpretation of Blood Concentrations:** Methadone can be detected in plasma within 30 minutes following oral ingestion, reaching a peak concentration at ~4 hours. Mean EDDP concentration are ~15% that of methadone. There is often a large overlap between reported therapeutic (0.03-0.56 mg/L) and fatal concentrations (0.06-3.1 mg/L). Peak serum concentrations following a single oral dose of 15 mg were 0.075 mg/L, 0.86 mg/L for 100 mg, and 0.83 mg/L for 120 mg; all at 4 hours. Chronic oral administration of 100-200 mg to tolerant subjects produced average peak plasma concentrations of 0.83 mg/L at 4 hours, decreasing to 0.46 mg/L at 24 hours. Peak plasma methadone concentrations of 0.034 mg/L were obtained at 50 minutes following intramuscular injection of 10 mg, while intravenous administration of 10 mg produced concentrations of 0.096 mg/L at 34 minutes. Concentrations greater than 0.10 mg/L are required for prevention of opiate withdrawal symptoms. In cancer patients treated for pain relief and sedation, methadone concentrations were 0.35 ± 0.18 mg/L.

**Interpretation of Urine Test Results:** The percentage of a dose excreted in the urine as unchanged methadone and EDDP will vary with the pH of the urine. Urinary excretion of unchanged parent drug is 5-50% and EDDP 3-25%. It may be possible to use excretion data to monitor individuals’ compliance in a methadone program after establishing their intraindividual variation in excretion patterns through long-term monitoring.

**Effects:**

*Psychological:* Drowsiness, sedation, dizziness, lightheadedness, mood swings (euphoria to dysphoria), depressed reflexes, altered sensory perception, stupor, and coma.

*Physiological:* Strong analgesia, headache, dry mouth, facial flushing, nausea, constipation, respiratory depression, muscle flaccidity, pupil constriction, and decreased heart rate.

**Duration of Effects:** Onset of analgesia occurs 10-20 minutes following parenteral administration and 30-60 minutes after oral administration. Oral administration results in a delay in onset, lower peak concentration and longer duration of action. Following single oral doses effects may last 6-8 hours, increasing to 22-48 hours in cases of chronic administration.
Side Effect Profile: Sedation, alteration in cognitive and sensory efficiency, respiratory depression, nausea, vomiting, headache, constipation, urinary retention, sweating, sleep disorders, and concentration disorders. Infrequent side effects include urticaria, hypersensitivity reaction, shock, and pulmonary edema. Overdose can include slow, shallow breathing, respiratory depression, clammy skin, convulsions, extreme somnolence, apnea, circulatory collapse, cardiac arrest, coma, and possible death.

Tolerance, Dependence and Withdrawal Effects: Upon repeated administration, tolerance may develop to the nauseant, miotic, sedative, respiratory depressant, and cardiovascular effects of methadone. Tolerance develops more slowly to methadone than to morphine in some patients. Methadone can produce physiological and psychological drug dependence of the morphine type, and has the potential for being abused. Withdrawal symptoms are similar to those of other opioids but are less severe, slower in onset, and last longer. Symptoms include watery eyes, runny nose, nausea, loss of appetite, diarrhea, cramps, muscle aches, dysphoria, restlessness, irritability, anxiety, pupillary dilation, piloerection, tremors, chills, sweating, increased sensitivity to pain, insomnia, and tachycardia.

Drug Interactions: There is additive CNS depressive effects with concurrent use of sedatives, hypnotics, tranquilizers, other narcotic analgesics, tricyclic antidepressants, alcohol and other CNS depressant drugs, resulting in exaggerated respiratory depression and sedation. Methadone can potentiate the deleterious effects of alcohol. Pentazocine, nalbuphine, butorphanol and buprenorphine are partial agonists and will behave as antagonists in the presence of methadone, resulting in the precipitation of withdrawal symptoms. Rifampin reduces blood concentrations of methadone and may lead to withdrawal. Blood levels of desipramine have increased with concurrent methadone therapy.

Performance Effects: In general, laboratory studies have shown that non-tolerant individuals receiving single doses of methadone have produced dose-dependent reductions in reaction time, visual acuity, information processing, and sedation. Significant psychomotor impairments are seldom evident when tolerant subjects have been tested, including performance deficits in reaction time, attention, and peripheral vision. In the majority of experimental clinical trials, psychophysical performance tests have yielded the same results for methadone substitution patients as for control groups. However, variable results have been observed. Attention and perception tasks have been impaired in methadone maintenance patients, but sociodemographic factors may have played a role. In patients receiving 35-85 mg methadone daily, significant impairment was measured on attention, perception and learning tasks but there was no reaction time deficit. In patients receiving a daily average of 63 mg methadone, significant impairment in distance perception, attention span and time perception was observed. No significant adverse effects were measured with addicts stabilized for at least 1 year on daily oral doses of methadone.

Effects on Driving: The drug manufacturer cautions that methadone may impair the mental and/or physical abilities required for the performance of potentially hazardous
tasks, and that the sedative effects of the drug may be enhanced by concurrent use of other CNS depressants, including alcohol. In healthy, non-methadone using volunteers, single doses of methadone will impair driving ability. Numerous European studies of long-term methadone maintenance patients have shown that appropriately administered methadone does not cause significant psychomotor or cognitive impairment when administered regularly and when the subject abstains from all other drugs. However, in the majority of cases, patients did not exhibit stable abstinence from drug use and had an increased occurrence of simultaneous psychiatric/neurotic disorders or personality disturbances which, by themselves, could be a reason to doubt their driving ability. In Germany, the Joint Advisory Council for Traffic Medicine at the Federal Ministry of Transport, Building and Housing and the Federal Ministry for Health issued the following recommendation: Heroin addicts treated with methadone are generally not fit to drive; however, these patients may be considered fit to drive if they show a period of methadone substitution for more than a year; stable psychosocial integration; no evidence of the consumption of additional psychotropic substances; evidence of a subject’s readiness to feel responsible for himself/herself; therapy compliance; and no evidence of serious personality defects.

**DEC Category:** Narcotic Analgesic.

**DEC Profile:** Horizontal gaze nystagmus not present; vertical gaze nystagmus not present; lack of convergence not present; pupil size constricted; little to no reaction to light; pulse rate down; blood pressure down; body temperature down. Other characteristic indicators may include muscle tone flaccidity, droopy eyelids, drowsiness, depressed reflexes, and dry mouth.

**Panel’s Assessment of Driving Risks:** Moderate to severely impairing in naïve or non-tolerant individuals, causing dose-dependent reductions in reaction time, visual acuity and information processing. Significant psychomotor impairment is not expected in tolerant individuals. Driving ability and driving fitness are nevertheless often limited because of consumption of additional psychotropic substances and psychopathological findings.

**References and Recommended Reading:**


**Methamphetamine (and Amphetamine)**

Methamphetamine hydrochloride is a white to light brown crystalline powder, or clear chunky crystals resembling ice. Methamphetamine base is a liquid.

**Synonyms:** Methamphetamine: chalk, chrissy, crank, crystal, glass, go, hydro, ice, meth, rock candy, speed, whiz; Desoxyn®; Amphetamine: dextroamphetamine; Dexedrine®, Adderall®, Benzedrine®, DextroStat®, Biphetamine®, Gradomet®.

**Source:** The majority of street methamphetamine is produced in clandestine laboratories (e.g. reduction of *l*-ephedrine or *d*-pseudoephedrine over red phosphorus with hydroiodic acid, or reduction with sodium or lithium in condensed liquid ammonia). Methamphetamine remains concentrated in western U. S. states and some rural areas elsewhere. *d*-Methamphetamine is a schedule II controlled substance (Desoxyn®) available in 5 mg white, 10 mg pink, and 15 mg yellow strength tablets. Amphetamine is also a Schedule II controlled substance and is usually supplied as the sulfate salt of the *d*-isomer (Dexedrine®), or as the racemic mixture (Benzedrine®), or a mixture of the two (Adderall®). Dexedrine® is available in 5, 10, and 15 mg strength, orange/black capsules, or 5 mg tablets. Adderall® is available in 5, 7.5, 10, 12.5, 20, and 30 mg strength, blue or orange tablets.

**Drug Class:** CNS stimulant, sympathomimetic, appetite suppressant.

**Medical and Recreational Uses:** Medicinally, methamphetamine is used in the treatment of narcolepsy, attention deficit disorder (ADD), and attention deficit hyperactivity disorder (ADHD). Typical doses are 10 mg/day or up to 40 mg daily, and a course of greater than six weeks is not recommended. Methamphetamine is infrequently used in the treatment of obesity, overeating disorders, and weight loss due to its abuse potential. Amphetamine is also used in ADD, narcolepsy, and weight control. Recreationally, methamphetamine is abused to increase alertness, relieve fatigue, control weight, treat mild depression, and for its intense euphoric effects.

**Potency, Purity and Dose:** Purity of methamphetamine is currently very high, at 60-90%, and is predominantly *d*-methamphetamine which has greater CNS potency than the *l*-isomer or the racemic mixture. Common abused doses are 100-1000 mg/day, and up to 5000 mg/day in chronic binge use. Therapeutic doses of Desoxyn® are 2.5-10 mg daily, with dosing not exceed 60 mg/day. To treat narcolepsy, 5-60 mg/day of amphetamine is ingested in divided doses; and in ADD and ADHD doses of 2.5-10 mg/day is administered, depending on age.

**Route of Administration:** Methamphetamine users often begin with intranasal or oral use and progress to intravenous use, and occasionally smoking. In contrast to cocaine, the hydrochloride salt of methamphetamine can itself be smoked. Methamphetamine is used sometimes with alcohol or marijuana, particularly during the withdrawal phase.

**Pharmacodynamics:** Methamphetamine increases synaptic levels of the neurotransmitters dopamine, serotonin (5-HT) and norepinephrine, and has α and β
adrenergic agonist effects. Norepinephrine is responsible for methamphetamine’s alerting, anorectic, locomotor and sympathomimetic effects; dopamine stimulates locomotor effects, psychosis, and perception disturbances; and 5HT is responsible for delusions and psychosis. Methamphetamine’s effects are similar to cocaine but its onset is slower and the duration is longer. Racemic amphetamine and d-amphetamine have similar chemical properties and actions to methamphetamine but are less potent.

**Pharmacokinetics:** Following oral administration, peak methamphetamine concentrations are seen in 2.6-3.6 hours and the mean elimination half-life is 10.1 hours (range 6.4-15 hours). The amphetamine metabolite peaks at 12 hours. Following intravenous injection, the mean elimination half-life is slightly longer (12.2 hours). Methamphetamine is metabolized to amphetamine (active), p-OH-amphetamine and norephedrine (both inactive). Several other drugs are metabolized to amphetamine and methamphetamine and include benzphetamine, selegeline, and famprofazone.

**Molecular Interactions / Receptor Chemistry:** Methamphetamine is metabolized to amphetamine via cytochrome P450 2D6. Potential inhibitors of the 2D6 isoenzyme could decrease the rate of methamphetamine elimination if administered concurrently, while potential inducers could increase the rate of elimination.

**Blood to Plasma Concentration Ratio:** 0.65 (N=1).

**Interpretation of Blood Concentrations:** Blood concentrations can generally be used to distinguish therapeutic use from abuse. Concentrations of 0.02-0.05 mg/L are typical for therapeutic use, and up to 0.2 mg/L have been documented. Concentrations greater than this represent abuse. Concentrations do not disclose phase of use. Normal concentrations in recreational use are 0.01 to 2.5 mg/L (median 0.6 mg/L). Concentrations above this range will likely be associated with severe, possibly life threatening, toxicity. There is no evidence for improved performance in any task or test following use of doses greater than 40 mg (or concentrations greater than 0.2 mg/L).

Peak blood methamphetamine concentrations occur shortly after injection, a few minutes after smoking, and around 3 hours after oral dosing. Peak plasma amphetamine concentrations occur around 10 hours after methamphetamine use.

**Interpretation of Urine Test Results:** Positive results generally indicate use within 1-4 days but could be up to a week following heavy chronic use. Rate of excretion into the urine is heavily influenced by urinary pH. Between 30-54% of an oral dose is excreted in urine as unchanged methamphetamine and 10-23% as unchanged amphetamine. Following an intravenous dose, 45% is excreted as unchanged parent drug and 7% amphetamine.

**Effects:** Methamphetamine effects are less intense after oral ingestion than following smoked or intravenous use.

**Early phase – Psychological:** Euphoria, excitation, exhilaration, rapid flight of ideas, increased libido, rapid speech, motor restlessness, hallucinations, delusions, psychosis, insomnia, reduced fatigue or drowsiness, increased alertness, heightened sense of well
being, stereotypes behavior, feelings of increased physical strength, and poor impulse control.

*Early phase – Physiological:* Increased heart rate, increased blood pressure, increased respiration rate, elevated temperature, palpitations, irregular heartbeat, dry mouth, abdominal cramps, appetite suppressed, twitching, pallor, dilated pupils, HGN at high doses, faster reaction time, increased strength, and more efficient glucose utilization.

*Late phase – Psychological:* Dysphoria, residual stimulation, restlessness, agitation, nervousness, paranoia, violence, aggression, lack of coordination, pseudo-hallucinations, delusions, psychosis, and drug craving.

*Late phase – Physiological:* Fatigue, sleepiness with sudden starts, itching/picking/scratching, normal heart rate, and normal to small pupils which are reactive to light.

Binge use of methamphetamine can be broken down into the following phases:

- **Rush** – (5 minutes) intense euphoria, rapid flight of ideas, sexual stimulation, high energy, obsessive/compulsive activity, thought blending, dilated pupils; **Shoulder** – (1 hour) less intense euphoria, hyperactivity, rapid flight of ideas, obsessive/compulsive activity, thought blending, dilated pupils; **Binge use** – (1-5 days) the drug is frequently readministered in an attempt to regain or maintain euphoria; **Tweaking** – (4-24 hours) dysphoria, scattered and disorganized thought, intense craving, paranoia, anxiety and irritability, hypervigilance, auditory and tactile hallucinations, delusions, and normal pupils; **Crash** – (1-3 days) intense fatigue, uncontrollable sleepiness and catnapping, continuing stimulation, drug craving; **Normal** – (2-7 days) apparent return to “normalcy” although drug craving may appear; **Withdrawal** – anergia, anhedonia, waves of intense craving, depression, hypersomnia, exhaustion, extreme fatigue.

**Side Effect Profile:** Light sensitivity, irritability, insomnia, nervousness, headache, tremors, anxiety, suspiciousness, paranoia, aggressiveness, delusions, hallucinations, irrational behavior, and violence. In overdose, symptoms may include hyperthermia, tachycardia, severe hypertension, convulsions, chest pains, stroke, cardiovascular collapse, and possible death. Other common side effects following abuse of amphetamines include viral hepatitis, Sexually Transmitted Diseases (STDs), HIV, septicemia, abscesses, collapsed blood vessels, and malnutrition. Chronic abuse generally produces a psychosis that resembles schizophrenia and is characterized by paranoia, picking at the skin, preoccupation with one’s own thoughts, and auditory and visual hallucinations. Violent and erratic behavior is frequently seen among chronic abusers. Over time, methamphetamine appears to cause reduced levels of dopamine, which can result in symptoms like those of Parkinson’s disease.

**Duration of Effects:** Onset of effects is rapid following intravenous use and smoking, while effects onset more slowly following oral use. Overall effects typically last 4-8 hours; residual effects can last up to 12 hours.

**Tolerance, Dependence and Withdrawal Effect:** Methamphetamine has a high potential for abuse and dependence. Tolerance may develop and users may quickly become addicted and use it with increasing frequency and in increasing doses. Abrupt
discontinuation of use can produce extreme fatigue, mental depression, apathy, long periods of sleep, irritability, and disorientation.

**Drug Interactions:** Phenobarbital, propoxyphene, phenytoin and MAOI’s slow the metabolism of amphetamines and increases their effect on the release of norepinephrine and other monoamines from adrenergic nerve endings. Amphetamines may counteract sedative effects of antihistamines. Methamphetamine may restore ethanol induced impairment in simple repetitive tasks of short duration, however, there is no restoration of ethanol-induced deficits of balance and steadiness. In general, high doses of amphetamines are likely to increase the impairing effects of alcohol. Chlorpromazine and haloperidol block dopamine and norepinephrine reuptake, thus inhibiting the central stimulant effects of amphetamines. Amphetamine potentiates the analgesic effect of meperidine.

**Performance Effects:** Laboratory studies have been limited to much lower doses than those used by methamphetamine abusers. Doses of 10-30 mg methamphetamine have shown to improve reaction time, relief fatigue, improve cognitive function testing, increase subjective feelings of alertness, increase time estimation, and increase euphoria. However, subjects were willing to make more high-risk choices. The majority of laboratory tests were administered 1 hour post dose. Expected performance effects following higher doses may include agitation, inability to focus attention on divided attention tasks, inattention, restlessness, motor excitation, increased reaction time, and time distortion, depressed reflexes, poor balance and coordination, and inability to follow directions.

**Effects on Driving:** The drug manufacturer states that patients should be informed that methamphetamine andamphetamine may impair the ability to engage in potentially hazardous activities such as driving a motor vehicle. In epidemiology studies drive-off-the-road type accidents, high speed, failing to stop, diminished divided attention, inattentive driving, impatience, and high risk driving have been reported. Significant impairment of driving performance would also be expected during drug withdrawal. In a recent review of 101 driving under the influence cases, where methamphetamine was the only drug detected, blood concentrations ranged from <0.05-2.36 mg/L (mean 0.35 mg/L, median 0.23 mg/L). Driving and driver behaviors included speeding, lane travel, erratic driving, accidents, nervousness, rapid and non-stop speech, unintelligible speech, disorientation, agitation, staggering and awkward movements, irrational or violent behavior, and unconsciousness. Impairment was attributed to distraction, disorientation, motor excitation, hyperactive reflexes, general cognitive impairment, or withdrawal, fatigue and hypersomnolence.

**DEC Category:** CNS stimulant.

**DEC Profile:** Horizontal gaze nystagmus not present; vertical gaze nystagmus not present; lack of convergence not present; pupil size dilated; reaction to light slow; pulse rate elevated; blood pressure elevated; body temperature normal to down. Other
characteristic indicators may include restlessness, body tremors, talkativeness, exaggerated reflexes, anxiety, and track marks or recent injection sites.

**Panel’s Assessment of Driving Risks:** At lower dose, amphetamines have few effects on cognitive functioning and may result in an enhancement of some psychomotor tasks, but risk-taking increases at higher doses and responses become inappropriate. Drug withdrawal could also lead to the impairment of psychomotor skills required for safe driving.

**References and Recommended Reading:**
Baselt RC. *Drug effects on psychomotor performance*. Biomedical Publications, Foster City, CA; pp 30-5, pp 244-6;2001.
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Jerome L, Segal A. Benefit of long-term stimulus on driving in adults with ADHD. *J Nerv Ment Dis* 2001(1);189:63-4.
National Transportation Safety Board safety study: Fatigue, alcohol, other drugs, and medical factors in fatal-to-the-driver heavy truck crashes (vol I and II). Accession# PB90-917002, report# NTSB/SS-90/01/02, National Transportation Safety Board, Washington DC, 1990.
Methylenedioxymethamphetamine (MDMA, Ecstasy)
MDMA is a white, tan or brown powder. Available primarily in tablet form.

**Synonyms:** 3,4-methylenedioxymethamphetamine; ecstasy, ADAM, candy canes, disco biscuit, doves, E, eckie, essence, hug drug, love drug, M&M, rolls, white doves, X, XTC.

**Source:** MDMA is the methylenedioxy derivative of methamphetamine. Starting materials in its illicit manufacture include isosafrole (Leuckart reaction) and safrole (Merck patent). MDMA is most commonly found in tablet forms of various colors, carrying distinctive markings on one side such as a dove, E, yin/yang symbol, Mitsubishi symbol, etc. MDMA is a Schedule I controlled substance.

**Drug Class:** Mild CNS stimulant, empathogen, entactogen, mild hallucinogen and psychedelic, appetite suppressant.

**Medical and Recreational Uses:** Originally patented as an appetite suppressant and used as a possible adjunct to psychotherapy, there is currently no legitimate medical use in the U. S. MDMA is recreationally used as a party, rave or dance drug for its stimulant, mild hallucinogenic, and empathogenic properties.

**Potency, Purity and Dose:** MDMA exists as a racemic mixture, with the S-(+)-enantiomer having greater CNS potency compared to the R-(-)-enantiomer. Potency of street samples is highly variable, and tablets sold as ‘ecstasy’ may in fact contain little or no MDMA, but may contain caffeine, ephedrine, phenylpropanolamine, paramethoxyamphetamine (PMA), methylenedioxymethamphetamine (MDA), dextromethorphan, amphetamine, methamphetamine, and ketamine. Some tablets have been reported to contain LSD or heroin. Typical doses in a series of pills can range between 10–150 mg of MDMA. User surveys report a range of doses between 50-700 mg in a session, with an average of 120 mg. Most common pattern of use is binge consumption at all night rave or dance parties. MDMA is frequently taken with other recreational drugs such as ethanol, marijuana, cocaine, methamphetamine, nitrous oxide, and GHB.

**Route of Administration:** Primarily oral administration, although MDMA could conceivably be dissolved and injected, or crushed and snorted.

**Pharmacodynamics:** MDMA is a phenylethylamine that has stimulant as well as psychedelic effects. MDMA is related in structure and effects to methamphetamine, however, it has significantly less CNS stimulant properties than methamphetamine. MDMA has a high affinity for 5-HT\textsubscript{2} receptors. Both S- and R- enantiomers of MDMA cause acute depletion of presynaptic serotonin (5-HT), depression of 5-HT synthesis by tryptophan hydroxylase, and retrograde destruction of 5-HT neurons following high doses. MDMA also increases levels of norepinephrine and dopamine. The MDMA metabolite, S-(+)- MDA, elicits more stereotypic behavior and is an even more potent...
neurotoxin than the parent drug. MDA destroys serotonin-producing neurons which play a direct role in regulating aggression, mood, sexual activity, sleep, and sensitivity to pain.

**Pharmacokinetics:** MDMA is rapidly absorbed and the half-life of MDMA is ~ 7 hours, although non-linear pharmacokinetics have been observed due to stereoselective pharmacokinetics of the enantiomers. MDMA is metabolized to MDA which is the only metabolite reported in blood and plasma. S-(+)-MDA accumulates in blood due to stereoselective metabolism of S-(+)-MDMA. MDA is further metabolized to its 3-hydroxy-4-methoxy and 3,4-dihydroxy derivatives (HMA and HHA). Additional MDMA metabolites include 3-hydroxy-4-methoxymethamphetamine (HMMA) and 3,4-dihydroxymethamphetamine (HHMA). These polar hydroxylated metabolites are conjugated prior to their excretion in urine.

**Molecular Interaction / Receptor Chemistry:** The majority of MDMA N-demethylation to MDA is via the cytochrome P450 2D6 isoenzyme, with minor contributions by the 1A2 isoform. Potential inhibitors of these isoenzymes could decrease the rate of MDMA elimination if administered concurrently, while potential inducers could increase the rate of elimination. Both extensive and poor MDMA metabolizers have been identified.

**Blood to Plasma Concentration Ratio:** Data not available.

**Interpretation of Blood Concentrations:** No clear correlation exists between MDMA blood concentrations and effects. MDMA and MDA are the analytes detected in blood, with MDA concentrations typically only 5-10% of the corresponding MDMA concentrations. Higher MDA:MDMA ratios may indicate co-administration of MDA. Plasma concentrations following single oral doses of 50, 75, 100, 125 and 150 mg of MDMA were 0.02-0.08 mg/L, 0.13 mg/L, 0.19-0.21 mg/L, 0.24 mg/L, and 0.44 mg/L, respectively. Peak concentrations of MDMA and MDA are observed at 1.5-2 hours and 4 hours, respectively.

**Interpretation of Urine Test Results:** MDMA, MDA, HMMA, HHMA, HMA and HHA are typically found in urine following their hydrolysis. MDA and HMMA concentrations in urine are typically 10-15% of the corresponding MDMA concentrations.

**Effects:**

**Psychological:** Low to moderate doses (50-200 mg) produce mild intoxication, relaxation, euphoria, an excited calm or peace, feelings of well-being, increase in physical and emotional energy, increased sociability and closeness, heightened sensitivity, increased responsiveness to touch, changes in perception, and empathy. At higher doses, agitation, panic attacks, and illusory or hallucinatory experiences may occur.

**Physiological:** Low to moderate doses (50-200 mg) produce mild visual disturbances (blurred or double vision, increased light sensitivity), dilated pupils, dry mouth, sweating, ataxia, muscle tension, and involuntary jaw clenching.
**Side Effect Profile:** Impairment of cognitive, perception, and mental associations. Psychological difficulties include confusion, depression, sleep problems, drug craving, severe anxiety, and paranoia. Subjects may experience fatigue, uncoordinated gait, decreased fine motor skills, attentional dysfunction (difficulty to maintain attention during complex tasks), preoccupation, hyperthermia, tachycardia, hyperthermia, hyponatremia, convulsions, and catatonic stupor. Prolonged cognitive and behavioral effects may occur including poor memory recall, flashbacks, panic attacks, psychosis, and depersonalization due to serotonergic neuron damage and decreased serotonin production as a result of long-term use.

**Duration of Effects:** Following oral administration, effects onset in 20-30 minutes and desired effects may last only an hour or more, depending on dose. Other general effects last for approximately 2-3 hours. LSD is sometimes used in combination with MDMA to increase its duration of effects. Residual and unwanted effects are generally gone within 24 hours although confusion, depression and anxiety may last several weeks.

**Tolerance, Dependence and Withdrawal Effect:** Drug stacking refers to the ingestion of single doses consecutively as effects begin to wane, similar to cocaine or methamphetamine binges. Such extensive or binge use usually occurs over weekends, and can result in exhaustion, apathy, depression, irritability, insomnia and muscle tension early the next week (often referred to as “terrible Tuesdays”). Tolerance does develop, however, the occurrence of physical and/or psychological dependence is unknown. Persistent neurological deficits may occur, including serotonergic neuron damage which leads to less production of serotonin.

**Drug Interactions:** The dopamine D₂ receptor antagonist, haloperidol, attenuates psychological effects of MDMA but has no effect on physiological effects.

**Performance Effects:** MDMA can enhance impulsivity and make it difficult for a person to maintain attention during complex tasks (selective attention, divided and sustained attention, and complex attention tasks). Laboratory studies have demonstrated changes in cognitive, perception and mental associations, instability, uncoordinated gait, and poor memory recall. Distortion of perception, thinking, and memory, impaired tracking ability, disorientation to time and place, and slow reactions are also known performance effects. Single oral doses of MDMA causes subjective excitability, anxiety, perceptual changes, and thought disorders 1-3 hours post dose.

**Effects on Driving:** In an advanced driving simulator study, subjects were given a mean single dose of 56 mg MDMA. Compared to a sober state, moderate effects on vehicle control, acceptance of higher levels of risk, acute changes in cognitive performance, and impaired information processing ability were observed. In six subjects arrested for driving under the influence, MDMA was the only drug detected at blood concentrations ranging from <0.05-0.58 mg/L. The subjects were cooperative and laid back, and experienced muscle twitching, body tremors, perspiring, dilated pupils, slow reaction to light, and poor performance on field sobriety tests. The following concentrations of MDMA have also been measured in other retrospective studies; serum...
MDMA concentrations ranging from 0.001-0.514 mg/L (mean 0.076 mg/L) in 18 cases of driving impairment; blood MDMA concentrations ranging from 0.04-0.38 mg/L (mean 0.18±0.14 mg/L; median 0.19 mg/L) in 9 impaired driving cases; blood MDMA concentrations of 0.12, 0.08, and 0.14 mg/L in 3 impaired driving cases; and a blood MDMA concentration of 2.14 mg/L and urine 118.8 mg/L in one driving fatality case. Another study reported the occurrence of speeding, jumping red lights, hallucinations/delusions, and a sense of detachment in five impaired driving cases, however, no MDMA concentrations were mentioned.

**DEC Category:** Hallucinogen; (with many characteristics similar to a CNS stimulant)

**DEC Profile:** Horizontal gaze nystagmus not present; vertical gaze nystagmus not present; lack of convergence not present; pupil size dilated; reaction to light slow; pulse rate elevated; blood pressure normal to elevated; body temperature normal to elevated. Other characteristic indicators may include profuse sweating, muscle twitching, body tremors, and poor performance in field sobriety tests. Subjects are usually described as very cooperative and “laid-back”. Note that elevated blood pressure and body temperature are not always observed.

**Panel’s Assessment of Driving Risks:** Low to moderate single doses of MDMA can cause acute changes in cognitive performance and impair information processing, which in turn would impair driving ability. Basic vehicle control is only moderately affected, however, subjects may accept higher levels of risk.

**References and Recommended Reading:**
Brookhuis KA, DeWaard D, Pernot LMC. A driving simulator study on driving performance and traffic safety after multiple drug use, consisting of MDMA (Ecstasy) and various other psychoactive compounds. Proceedings of the International Council on Alcohol Drugs and Traffic Safety (ICADTS), Stockholm Sweden, May 2000.


Morgan MJ. Recreational use of “ecstasy” (MDMA) is associated with elevated impulsivity. *Neuropsychopharm* 1998;19(4):252-64.


Omtzigt JGC, Vermasse CJ, Zweipfenning PGM. Deaths associated with amphetamine, 3,4-methylenedioxymethamphetamine (MDMA), 3,4-methylenedioxyethamphetamine (MDEA), or 3,4-methylenedioxyamphetamine (MDA) abuse. Proceedings of the 23rd meeting of the International Association of Forensic Toxicologists (TIAFT), Tampa, FL 1994.


Morphine (and Heroin)

Morphine and heroin are white, crystalline powders. Illicit heroin may vary in color from white to dark brown due to impurities, or may appear as a black tar-like material.

**Synonyms:** Morphine: Astramorph®, Duramorph®, Infumorph®, Kadian®, Morphine Sulfate®, MSIR®, MS-Contin®, Oramorph SR®, Roxanol®. Heroin: diacetylmorphine, diamorphine; Mexican brown or Mexican black tar heroin; bags, blue-steel, China white, H, horse, junk, no-name, silk, skag, smack. Scramble (cut heroin), bone (uncut heroin for smoking), chippers (occasional users).

**Source:** Morphine is a naturally occurring substance extracted from the seedpod of the poppy plant, *Papaver somniferum*. The milky resin that seeps from incisions made in the unripe seedpod is dried and powdered to make opium, which contains a number of alkaloids including morphine. Morphine concentration in opium can range from 4-21%. An alternate method of harvesting morphine is by the industrial poppy straw process of extracting alkaloids from the mature dried plant, which produces a fine brownish powder. Morphine is a schedule II controlled substance and is available in a variety of prescription forms: injectables (0.5-25 mg/mL strength); oral solutions (2-20 mg/mL); immediate and controlled release tablets and capsules (15-200 mg); and suppositories (5-30 mg). Heroin is a schedule I controlled substance and is produced from morphine by acetylation at the 3 and 6 positions. The majority of heroin sold in the U. S. originates from Southeast Asia, South America (Columbia) and Mexico. Low purity Mexican black tar heroin is most common on the West coast, while high purity Columbian heroin dominates in the East and most mid-western states.

**Drug Class:** Narcotic analgesic.

**Medical and Recreational Uses:** Morphine is used medicinally for the relief of moderate to severe pain in both acute and chronic management. It can also be used to sedate a patient pre-operatively and to facilitate the induction of anesthesia. Heroin has no currently accepted medical uses in the U.S., however, it is an analgesic and antitussive.

**Potency, Purity and Dose:** The dosage of morphine is patient-dependent. A usual adult oral dose of morphine is 60-120 mg daily in divided doses, or up to 400 mg daily in opioid tolerant patients. Recreationally, daily heroin doses of 5-1500 mg have been reported, with an average daily dose of 300-500 mg. Addicts may inject heroin 2-4 times per day. Depending on the demographic region, the street purity of heroin can range from 11-72% (average U.S. purity is ~38%). Heroin may be cut with inert or toxic adulterants such as sugars, starch, powdered milk, quinine, and ketamine. Heroin is often mixed with methamphetamine or cocaine (“speedball”) and injected; or co-administered with alprazolam, MDMA (Ecstasy), crack cocaine, or diphenhydramine.

**Route of Administration:** Morphine: oral, intramuscular, intravenous, rectal, epidural, and intrathecal administration. Morphine tablets may be crushed and injected, while opium can be smoked. Heroin: smoked, snorted, intravenous (“mainlining”), and
subcutaneous (“skin popping”) administration. Black tar heroin is typically dissolved, diluted and injected, while higher purity heroin is often snorted or smoked.

**Pharmacodynamics:** Morphine produces its major effects on the CNS primarily through μ-receptors, and also at κ- and δ-receptors. μ₁-receptors are involved in pain modulation, analgesia, respiratory depression, miosis, euphoria, and decreased gastrointestinal activity; μ₂-receptors are involved in respiratory depression, drowsiness, nausea, and mental clouding; κ-receptors are involved in analgesia, diuresis, sedation, dysphoria, mild respiratory depression, and miosis; and δ-receptors are involved in analgesia, dysphoria, delusions, and hallucinations. Heroin has little affinity for opiate receptors and most of its pharmacology resides in its metabolism to active metabolites, namely 6-acetylmorphine, morphine, and morphine-6-glucuronide.

**Pharmacokinetics:** The oral bioavailability of morphine is 20-40%, and 35% is bound in plasma. Morphine has a short half-life of 1.5 - 7 hours and is primarily glucuroconjugated at positions 3 and 6, to morphine-3-glucuronide (M3G) and morphine-6-glucuronide (M6G), respectively. A small amount (5%) is demethylated to normorphine. M6G is an active metabolite with a higher potency than morphine, and can accumulate following chronic administration or in renally impaired individuals. The half-life of M6G is 4 +/- 1.5 hours. Close to 90% of a single morphine dose is eliminated in the 72 hours urine, with 75% present as M3G and less than 10% as unchanged morphine. Heroin has an extremely rapid half-life of 2-6 minutes, and is metabolized to 6-acetylmorphine and morphine. The half-life of 6-acetylmorphine is 6-25 minutes. Both heroin and 6-acetylmorphine are more lipid soluble than morphine and enter the brain more readily.

**Molecular Interactions / Receptor Chemistry:** The uridine 5’-diphosphate-glucuronosyltransferase (UGT) 2B7 isoform is primarily involved in the metabolism of morphine. Potential inhibitors of this UGT isoform could decrease the rate of morphine elimination if administered concurrently, while potential inducers could increase the rate of elimination.

**Blood to Plasma Concentration Ratio:** Morphine 1.02; M6G 0.57; M3G 0.59

**Interpretation of Blood Concentrations:** Tolerance makes interpretation of blood or plasma morphine concentrations extremely difficult. Peak plasma morphine concentrations occur within an hour of oral administration, and within 5 minutes following intravenous injection. Average plasma concentrations of 0.065 mg/L are necessary for adequate therapeutic analgesia in ambulatory patients. Anesthetic concentrations can reach beyond 2 mg/L in surgical patients. Following oral doses of 10-80 mg, corresponding peak morphine concentrations in serum were 0.05-0.26 mg/L. Following an intravenous dose of 8.75g/70 kg, a peak serum concentration of 0.44 mg/L was reached. In 10 intravenous drug fatalities, where morphine was the only drug detected, postmortem whole blood morphine concentrations averaged 0.70 mg/L (range 0.20-2.3 mg/L). Following a single 12 mg intravenous mg dose of heroin, a peak heroin concentration of 0.141 mg/L was obtained at 2 minutes, while the 6-acetylmorphine and
morphine concentrations were 0.151 and 0.044, respectively. A single 5 mg intravenous dose of heroin produced a peak plasma morphine concentration of 0.035 mg/L at 25 minutes, while intravenous doses of 150-200 mg have produced plasma morphine concentrations of up to 0.3 mg/L. Intranasal administration of 12 mg heroin in 6 subjects produced average peak concentrations of 0.016 mg/L heroin in plasma within 5 minutes; 0.014 mg/L of 6-acetylmorphine at 0.08-0.17 hours; and 0.019 mg/L of morphine at 0.08-1.5 hours.

Interpretation of Urine Test Results: Positive morphine urine results generally indicate use within the last two to three days, or longer after prolonged use. Detection of 6-acetylmorphine in the urine is indicative of heroin use. High concentrations may indicate chronic use of the drug. It is important to hydrolyze urine specimens to assess a urine morphine concentration.

Effects: Depends heavily on the dose of morphine or heroin, the route of administration, and previous exposure. Following an intravenous dose of heroin, the user generally feels an intense surge of euphoria ("rush") accompanied by a warm flushing of the skin, dry mouth, and heavy extremities. The user then alternates between a wakeful and drowsy state ("on the nod").

Psychological: Euphoria, feeling of well-being, relaxation, drowsiness, sedation, lethargy, disconnectedness, self-absorption, mental clouding, and delirium.

Physiological: Analgesia, depressed heart rate, respiratory depression, CNS depression, nausea and vomiting, reduced gastrointestinal motility, constipation, flushing of face and neck due to dilatation of subcutaneous blood vessels, cramping, sweating, pupils fixed and constricted, diminished reflexes, and depressed consciousness.

Side Effect Profile: Drowsiness, inability to concentrate, apathy, lessened physical activity, constipation, urinary retention, nausea, vomiting, tremors, itching, bradycardia, severe respiratory depression, and pulmonary complications such as pneumonia. Medical complications among abusers arise primarily from adulterants found in street drugs and in non-sterile injecting practices, and may include skin, lung and brain abscesses, collapsed veins, endocarditis, hepatitis and HIV/AIDS. Overdose can include slow, shallow breathing, clammy skin, convulsions, extreme somnolence, severe respiratory depression, apnea, circulatory collapse, cardiac arrest, coma, and death.

Duration of Effects: Depending on the morphine dose and the route of administration, onset of effects is within 15-60 minutes and effects may last 4-6 hours. The duration of analgesia increases progressively with age although the degree of analgesia remains unchanged. Following heroin use, the intense euphoria lasts from 45 seconds to several minutes, peak effects last 1-2 hours, and the overall effects wear off in 3-5 hours, depending on dose.

Tolerance, Dependence and Withdrawal Effects: Both morphine and heroin have high physical and psychological dependence. With regular use, tolerance develops early to the duration and intensity of euphoria and analgesia. Withdrawal symptoms may occur if use is abruptly stopped or reduced. Withdrawal can begin within 6-12 hours after the last
dose and may last 5-10 days. Early symptoms include watery eyes, runny nose, yawning and sweating. Major withdrawal symptoms peak between 48-72 hours after the last dose and include drug craving, restlessness, irritability, dysphoria, loss of appetite, tremors, severe sneezing, diarrhea, nausea and vomiting, elevated heart rate and blood pressure, chills alternating with flushing and excessive sweating, goose-flesh, abdominal cramps, body aches, muscle and bone pain, muscle spasms, insomnia, and severe depression.

**Drug Interactions:** Alcohol increases the CNS effects of morphine such as sedation, drowsiness, and decreased motor skills. There is a higher risk of respiratory depression, hypotension and profound sedation or coma with concurrent treatment or use of other CNS depressant drugs such as barbiturates, benzodiazepines, hypnotics, tricyclic antidepressants, general anesthetics, MAO inhibitors, and antihistamines. Morphine may enhance the neuromuscular blocking action of skeletal muscle relaxants and produce an increased degree of respiratory depression. Small doses of amphetamine substantially increase the analgesia and euphoriant effects of morphine and may decrease its sedative effects. Antidepressants may enhance morphine’s analgesia. Partial agonists such as buprenorphine, nalbuphine, butorphanol, and pentazocine will precipitate morphine withdrawal.

**Performance Effects:** Laboratory studies have shown that morphine may cause sedation and significant psychomotor impairment for up to 4 hours following a single dose in normal individuals. Early effects may include slowed reaction time, depressed consciousness, sleepiness, and poor performance on divided attention and psychomotor tasks. Late effects may include inattentiveness, slowed reaction time, greater error rate in tests, poor concentration, distractibility, fatigue, and poor performance in psychomotor tests. Subjective feelings of sedation, sluggishness, fatigue, intoxication, and body sway have also been reported. Significant tolerance may develop making effects less pronounced in long-term users for the same dose. In a laboratory setting, heroin produced subjective feelings of sedation for up to 5-6 hours and slowed reaction times up to 4 hours, in former narcotic addicts. Euphoria and elation could also play a role on perception of risks and alteration of behaviors.

**Effects on Driving:** The drug manufacturer states that morphine may impair the mental and/or physical abilities needed to perform potentially hazardous activities such as driving a car, and patients must be cautioned accordingly. Driving ability in cancer patients receiving long-term morphine analgesia (mean 209 mg daily) was considered not to be impaired by the sedative effects of morphine to an extent that accidents might occur. There were no significant differences between the morphine treated cancer patients and a control group in vigilance, concentration, motor reactions, or divided attention. A small but significant slowing of reaction time was observed at 3 hours. In several driving under the influence case reports, where the subjects tested positive for morphine and/or 6-acetylmorphine, observations included slow driving, weaving, poor vehicle control, poor coordination, slow response to stimuli, delayed reactions, difficulty in following instructions, and falling asleep at the wheel.

**DEC Category:** Narcotic Analgesic.
**DEC Profile:** Horizontal gaze nystagmus not present; vertical gaze nystagmus not present; lack of convergence not present; pupil size constricted; little or no reaction to light; pulse rate down; blood pressure down; body temperature down. Other characteristic indicators may include presence of fresh injection marks, track marks, flaccid muscle tone, droopy eyelids, drowsiness or “on-the-nod”, and low raspy slow speech.

**Panel’s Assessment of Driving Risks:** Classification of risk depends on tolerance, dose, time of exposure, acute or chronic use, presence or absence of underlying pain, physiological status of individual, and the presence of other drugs. Moderately to severely impairing in non-tolerant individuals. Mild to moderately impairing if morphine is used as medication on a regular basis for chronic pain. Severely impairing in acute situations if used orally, or as an intravenous medication, or if either drug is taken illicitly.

**References and Recommended Reading:**


**Phencyclidine (PCP)**

PCP is a white, crystalline powder (contaminants may cause tan to brown color), or a clear, yellowish liquid.

**Synonyms:** 1-phenylcyclohexylpiperidine; amp, angel dust, animal tranquilizer, dips, dust, elephant, embalming fluid, formaldehyde, fry, hog, ozone, peace pill, rocket fuel, Sernyl, Sernylan, super kools, TicTac, tranq, water, wet.

**Source:** Synthetic chemical made in clandestine laboratories, or diverted from veterinary sources. PCP is currently a Schedule II controlled substance. In illicit synthesis, piperidine is reacted with cyanide and cyclohexanone to make piperidinocyclohexanecarbonitrile (PCC), which is then reacted with phenylmagnesium bromide to make PCP. PCP can be mixed with dyes and sold in a variety of tablets, capsules and colored powders. PCP is also sold as a liquid in small shaker bottles. PCP analogs are also available: cyclohexamine (PCE), phenylcyclohexylpyrrolidine (PHP), phenylcyclopentylpiperidine (PCPP), and thienylcyclohexylpiperidine (TCP).

**Drug Class:** Hallucinogen, dissociative anesthetic, psychotomimetic, sedative-hypnotic.

**Medical and Recreational Uses:** Formerly used as a surgical anesthetic, however, there is no current legitimate medical use in humans. Used as a veterinary anesthetic or tranquilizer. Recreationally used as a psychedelic and hallucinogen.

**Potency, Purity and Dose:** A light dose typically consists of 3-5 mg; a common dose is 5-10 mg; while a strong dose is greater than 10 mg. Lighter doses are usually smoked, intravenously or intranasally administered, while heavier doses are commonly ingested orally. The liquid can be sprinkled on tobacco or marijuana then smoked, or the cigarettes or joints themselves can be dipped in PCP solution; the resulting PCP dose can therefore vary widely. Due to difficulty of synthesis, street preparations have highly variable concentrations of PCP and byproducts. PCC, the PCP precursor, is found in approximately 20% of illicit samples and is more toxic than PCP as it releases cyanide. Abuse of PCP precursors or analog chemicals leads to similar or more devastating pharmacological effects than PCP. PCP is often administered or mixed with other drugs such as crack cocaine (“beam me up”), cocaine hydrochloride (“lovelies”), and marijuana (“crystal supergrass”, “donk”, “killer joints”, “sherm”, “wacky weed”, “wicky stick”).

**Route of Administration:** Smoked, intravenous injection, snorted, added as eye drops, oral ingestion, and transdermal absorption.

**Pharmacodynamics:** Dopaminergic, anticholinergic and opiate-like activities exist. PCP is a non-competitive NMDA-receptor antagonist, and blocks dopamine reuptake and elevates synaptic dopamine levels. It has high affinity to sites in the cortex and limbic structures.

**Pharmacokinetics:** Well absorbed following all routes of administration, although ~50% of PCP in cigarette smoke is converted to an inactive thermal degradation product.
PCP is highly lipid soluble and is stored in fat and brain tissue. The plasma binding of PCP is 65% and its half-life ranges from 7-46 hours (average 21 hours). PCP is extensively metabolized to inactive metabolites by a variety of metabolic routes.

**Molecular Interaction / Receptor Chemistry:** The cytochrome P450 3A isoenzyme plays a major role in PCP biotransformation. Potential inhibitors of this isoenzyme could decrease the rate of PCP elimination if administered concurrently, while potential inducers could increase the rate of elimination. PCP itself may inhibit 2B1 and 2C11 isoforms.

**Blood to Plasma Concentration Ratio:** 0.94 and 1.0 reported.

**Interpretation of Blood Concentrations:** There is no direct correlation between PCP concentration and behavioral or physical findings. Blood levels peak 1-4 hours after ingestion. Average peak plasma concentrations of 2.7 and 2.9 ng/mL were achieved after a 1 mg oral and intravenous dose, respectively. PCP concentrations ranged from 0.3 to 143 ng/mL in 63 patients presenting at a psychiatric hospital emergency room and were associated with a wide variety of psychotic clinical pictures resembling mania, depression or schizophrenia. All these patients had at least one manifestation of toxic psychosis and/or acute delirium, in addition to other symptoms. Similarly, plasma PCP concentrations ranged up to 812 ng/mL in 22 patients with nonfatal PCP intoxication. The most common physical findings were combativeness-agitation (64%), depressed level of consciousness (50%), hypertension (43%), miosis (43%) and tachycardia (43%). Blood PCP concentrations ranged from 12 to 118 ng/mL in 26 individuals arrested for public intoxication.

**Interpretation of Urine Test Results:** Elimination of PCP in 72 hours urine ranges from 4 to 19% for unchanged drug and 25 to 30% for conjugated metabolites. Approximately 97% of a dose is excreted in 10 days, and PCP use can be detected in urine by immunoassay up to a week following a high dose. Urine PCP concentrations ranged from 0.4-340 mg/L in 19 intoxicated patients.

**Effects:**

*Psychological:* Effects are usually dose dependent, and include euphoria, calmness, feelings of strength and invulnerability, lethargy, disorientation, loss of coordination, distinct changes in body awareness, distorted sensory perceptions, impaired concentration, disordered thinking, illusions and hallucinations, agitation, combativeness or violence, memory loss, bizarre behavior, sedation, and stupor.

*Physiological:* Rise in blood pressure and heart rate, flushing, profuse sweating, generalized numbness of extremities, blurred vision, grimacing facial expression, speech difficulties, ataxia, muscular incoordination, marked analgesia, nystagmus, and anesthesia. In the anesthetized state, the patient remains conscious with a staring gaze and rigid muscles.

**Side Effect Profile:** Excessive salivation, nausea, vomiting, amnesia, combativeness, severe anxiety, paranoia, flashbacks, seizures, coma, and death. PCP can simulate
schizophrenic-like symptomatology such as flattened affect, dissociative thought disorder, depersonalization and catatonic states. Long periods of use may lead to memory loss, difficulties with speech and thinking, depression, weight loss, liver function abnormalities, and rhabdomyolysis.

**Duration of Effects:** Onset of effects is very rapid when smoked or injected (1-5 minutes) and are delayed when snorted or orally ingested (30 minutes), with a gradual decline of major effects over 4-6 hours. A return to ‘normal’ may take up to 24 hours. Consciousness is regained within 10-60 minutes following intravenous administration, with a prolonged recovery period of 3-18 hours. Long-term psychological effects are possible and PCP may precipitate a psychotic reaction lasting a month or more that clinically appears like schizophrenia.

**Tolerance, Dependence and Withdrawal Effects:** Most PCP users administer the drug intermittently, although daily use has been reported and tolerance may develop. There is evidence of tolerance to behavioral effects of PCP in animals. PCP can be addicting and use can lead to psychological dependence, craving and drug seeking behavior. There has been no demonstration of physical dependency in humans. Upon abrupt discontinuation, physical distress, lack of energy, and depression are reported. Long periods of use may lead to memory loss, difficulties with speech and thinking, depression, and weight loss. These can last up to a year after cessation of use.

**Drug Interactions:** Benzodiazepines can decrease hypertensive effects and reverse seizure activity of PCP. Chlorpromazine and PCP use can cause severe hypotension. PCP may enhance effects of other CNS depressants like barbiturates and alcohol.

**Performance Effects:** Laboratory studies have shown that PCP causes disorientation, drowsiness, dizziness, ataxia, double or blurred vision, body image changes, disorganization of thoughts, combativeness, impairment of eye-hand coordination, memory impairment, paresthesia, slowed reaction time, distorted perceptions of space. Effects generally occur within 1 hour post dose. Subjective sensation of intoxication has been reported up to 8 hours and slowed reaction time up to 14 hours.

**Effects on Driving:** Fifty-six (56) subjects were arrested for erratic driving and were evaluated by a drug recognition examiner. All subjects were judged to be driving under the influence of PCP, and blood PCP concentrations ranged from 12 to 188 ng/mL (mean 51 ng/mL). Similarly, blood PCP concentrations ranged from 10 to 180 ng/mL (mean 73 ng/mL) in 50 subjects arrested for driving under the influence of PCP.

**DEC Category:** Phencyclidine.

**DEC Profile:** Horizontal gaze nystagmus present; vertical gaze nystagmus present; lack of convergence present; pupil size normal; reaction to light normal; pulse rate elevated; blood pressure elevated; body temperature elevated. Other characteristic indicators may include rigid muscles, cyclic behavior, sudden turn to violence, lack of response to
painful stimuli, trance-like state or blank stare, sweating, incomplete or delayed verbal responses.

**Panel’s Assessment of Driving Risks:** The use of PCP is not compatible with skills required for safe driving. Severe impairment of mental and physical abilities can occur following single doses.

**References and Recommended Reading:**


Cook CE. Pyrolytic characteristics, pharmacokinetics, and bioavailability of smoked heroin, cocaine, phencyclidine and methamphetamine. NIDA Res Mon 115 (pp. 6-23);1991.


Toluene
Toluene is a colorless, flammable liquid with a sweet pungent odor.

**Synonyms:** Toluol, methylbenzene, methyl benzol, and phenylmethane.

**Source:** Toluene is an aromatic hydrocarbon, occurring naturally in crude oil and in the tolu tree. It is produced during the process of making gasoline and other fuels from crude oil, in making coke from coal, and as a by-product in the manufacture of styrene. Toluene has numerous commercial and industrial applications and is a solvent in paints, lacquers, thinners, glues, correction fluid and nail polish remover, and is used in the printing and leather tanning processes. Due to its easy accessibility, low cost and ease of concealment, some U.S. states have placed restrictions on the sale of these products to minors.

**Drug Class:** Volatile solvent, CNS depressant.

**Medical and Recreational Uses:** No approved medical use of toluene. It is frequently abused for its intoxicating effects. Recreational use is most common among younger adolescents primarily because it is readily available, inexpensive and legal.

**Potency, Purity and Dose:** Solvents in many commercial and industrial products are often mixed and the solvent “sniffer” is often exposed to other solvents in addition to toluene. Acute and chronic accidental exposure to toluene can also occur, particularly in work environments. Regulatory Limits: OSHA recommends a maximum of 200 ppm toluene in workplace air for an 8-hour work day, 40-hour work week; NIOSH recommends an exposure limit of 100 ppm toluene in workplace air; and ACGIH recommends an exposure limit of 50 ppm in workplace air.

**Route of Administration:** Inhalation of vapor. May be sniffed directly from an open container, or “huffed” from a rag soaked in the substance and held to the face. Alternatively, the open container or soaked rag can be placed in a bag where the vapors can concentrate before being inhaled. Exposure can also occur by ingesting the liquid or via skin contact.

**Pharmacodynamics:** Solvents have three proposed mechanisms of action: they may alter the structure of membrane phospholipid bi-layers, impairing various ion channels; they may alternatively alter membrane bound enzymes or receptor-site specificity for endogenous substrates; or they may produce toxic metabolites modifying the hepatic microsomal system and possibly adducting RNA and DNA molecules. Toluene depresses neuronal activity and reversibly enhances GABA_A receptor-mediated synaptic currents and α1-glycine receptor-activated ion channel function. Toluene also inhibits glutamatergic neurotransmission via NMDA receptors and alters dopaminergic transmission.

**Pharmacokinetics:** Toluene is well-absorbed following oral ingestion and rapidly absorbed following inhalation. Toluene is detectable in the arterial blood within
10 seconds of inhalation exposure. It is highly lipid soluble and accumulates in adipose
tissue, tissues with high fat content, and highly vascularized tissues. Highest
concentrations are found in the liver, kidney, brain and blood. The initial half-life in
whole blood averages 4.5 hours, (range of 3-6 hours), with a terminal phase half-life of
72 hours. The half-life in adipose tissue ranges from 0.5-2.7 days, increasing with
amounts of body fat. Approximately 80% of a dose is metabolized in the liver. Side-chain
hydroxylation to benzyl alcohol is followed by oxidation to benzaldehyde by alcohol
dehydrogenase, oxidation to benzoic acid by aldehyde dehydrogenase and conjugation
with glycine to hippuric acid or reaction with glucuronic acid to form benzoyl
glucuronide. Ring hydroxylation to o- and p-cresol is a minor (~1%) metabolic pathway.
4%-20% is excreted unchanged by the lungs and <0.1% is excreted unchanged in the
urine. 60%-70% is excreted in urine as hippuric acid (glycine conjugate), and 10%-20%
as benzoic acid glucuronide conjugate.

**Molecular Interactions / Receptor Chemistry:** Toluene is metabolized to benzyl alcohol
via the cytochrome P450 2E1 isoform, and to a lesser extent to benzyl alcohol, o-cresol,
and p-cresol by 2B6, 2C8, 1A2 and 1A1 isoforms. Potential inhibitors of these
isoenzymes could decrease the rate of toluene elimination if administered concurrently,
while potential inducers could increase the rate of elimination.

**Blood to Breath Concentration Ratio:** Ranges from 7 to 15

**Interpretation of Blood Concentrations:** In non-exposed individuals, average toluene
concentrations have been measured at 0.47 µg/L (non-smokers) and 1.14 µg/L (smokers).
Toluene is detectable in arterial blood within 10 seconds of inhalation exposure.
Exposure to 38 ppm for 8 hours resulted in blood toluene concentrations of 0.59 mg/L.
Similarly, exposure to 34 ppm for 8 hours resulted in blood toluene concentrations of
0.457 mg/L, decreasing to 0.038 mg/L after 16 hours. Exposure to 100 ppm for
30 minutes produced 0.4 mg/L of blood toluene in resting individuals and 1.2 mg/L after
exercise. In 136 toluene abusers hospitalized or arrested while intoxicated, blood toluene
concentrations ranged from 0.3-30 mg/L. Three fatalities from acute toluene inhalation
had blood concentrations of 50, 60, and 79 mg/L. In 8 fatal cases of accidental or
intentional acute exposure of toluene, blood concentrations ranged from 10-48 mg/L
(mean 22 mg/L).

In 53 toluene abusers, blood concentrations of less than 1.0 mg/L corresponded to
an odor of “chemical” on the subject’s breath; some signs of impairment were observed
at concentrations of 1.0-2.5 mg/L; 50% of subjects with concentrations of 2.5-10 mg/L
were hospitalized with marked intoxication including hallucinations; and
unconsciousness or death were reported at concentrations of 10 mg/L or greater. In 6
subjects with blood toluene concentrations ranging from 9.8-31 mg/L, slurred speech,
slow movements, and an inability to concentrate were observed within minutes of
cessation of use.

**Interpretation of Urine Test Results:** In 136 toluene abusers hospitalized or arrested
while intoxicated, urine toluene concentrations ranged from 0-5 mg/L. In 120 glue
sniffers, concentrations of toluene in the urine ranged from 0.1-40.3 mg/L. Urinary o-
cresol and hippuric acid concentrations may have a high correlation with blood toluene concentrations. Hippuric acid excretion increases during the first 4 hours of exposure to up to 4 times the background level, then decreases rapidly to background levels within 6 hours. O-cresol excretion peaks during the last hour of chronic exposure or in the period immediately after acute exposure. Exercise increases the rate of both hippuric acid and o-cresol excretion. Hippuric acid concentrations (not corrected for creatinine) in non-exposed persons averaged 800 mg/L (range 400-1400); daily exposure to 50 ppm averaged 1920 mg/L (range 1260-2930); 100 ppm ranged from 2800-3500 mg/L; and 200 ppm averaged 5970 mg/L (range 4120-8650). O-cresol is not normally detected in the urine of non-exposed persons, while exposure to 200 ppm results in concentrations of 1-3 mg/L.

**Effects:**

*Psychological*: Dizziness, euphoria, grandiosity, floating sensation, drowsiness, reduced ability to concentrate, slowed reaction time, distorted perception of time and distance, confusion, weakness, fatigue, memory loss, delusions, and hallucinations.

*Physiological*: Irritation to the nose, throat, and eyes, headache, nystagmus, slurred speech, ataxia, staggering, impaired color vision, vigilance, nausea, vomiting, respiratory depression, convulsions, severe organ damage, coma, and death.

Mild exposure (100-1500 ppm) dose-dependently results in euphoria, dizziness, reduced inhibitions, feelings of inebriation similar to alcohol intoxication, headache, nausea, lethargy, slow thought and speech, impairment of coordination, loss of memory, slowed reaction time, fatigue, sedation, confusion, impared cognition function, impaired visual perception, staggering gait, muscular fatigue, and insomnia. More severe intoxication (10,000-30,000 ppm) will lead to tremors, arrhythmias, paralysis, unconsciousness, coma, and death. Chronic exposure may result in paranoid psychosis, temporal lobe epilepsy, mental retardation, and visual impairment.

**Side Effect Profile**: Toluene can cause brain, liver and kidney damage, hearing loss, memory impairment, and attention deficits. Death can result from heart failure, asphyxiation or aspiration. Toluene also owes its pharmacology to a mucosal irritant effect from an exothermic reaction with water. This results in vomiting, lacrimation and ocular burning, cough, chest pain, wheezing and possible interstitial edema, and kidney toxicity with tubular acidosis. Toluene exposure is also associated with a transient liver injury.

**Duration of Effects**: Once inhaled, the extensive capillary surface of the lungs allows rapid absorption of toluene and blood levels peak rapidly. Entry into the brain is extremely fast and onset of effects is almost immediate. Toluene effects generally last several hours.

**Tolerance, Dependence and Withdrawal Effects**: Tolerance to the effects of toluene has been shown in rats. Toluene has the potential to produce physical and psychological dependence, and its abuse liability is significant. Signs of physical dependence are observed on withdrawal.
**Drug Interactions:** There is a likely synergy or potentiation of effects with other solvents and CNS depressants. Acute consumption of ethanol inhibits toluene elimination resulting in increased blood toluene concentrations and tissue exposure. This is probably due to competition for alcohol dehydrogenase.

**Performance Effects:** Most analyses on performance have been on subjects exposed to 50-200 ppm over a 6-8 hour work period. Marked impairment in neurological and neuropsychological test performance have been observed, including impaired working memory and executive cognitive functions, impairment of visual-vigilance tasks, loss in color vision and visual perception, inability to concentrate, slow movements, and decreased response time to simple brief tests.

**Effects on Driving:** No driving or simulator studies exist for toluene. Blood toluene concentrations were above ~1.0 mg/L in 114 drivers arrested on suspicion of driving while intoxicated in Norway between 1983-1987. In 29 of these cases toluene was the only detected drug, with mean blood concentrations of 10 mg/L (range 1-29.3 mg/L). The authors stated there was no simple relation between blood toluene concentrations and degree of impairment, however, almost all drivers with blood toluene concentrations greater than 9.2 mg/L were considered impaired or highly probably impaired. No driving observations were documented.

**DEC Category:** Inhalant

**DEC Profile:** Horizontal gaze nystagmus present in high doses; vertical gaze nystagmus present in high doses; lack of convergence present; pupil size normal; reaction to light slow; pulse rate elevated; blood pressure elevated; body temperature normal. Other characteristic indicators may include strong odor of solvent or chemical on breath or clothes, residue of substance around nose, mouth or hands, slurred speech, and general intoxication.

**Panel’s Assessment of Driving Risks:** Acute and chronic exposure to toluene can result in severe impairment.

**References and Recommended Reading:**
ACGIH – American Conference of Government Industrial Hygienists.


OSHA – Occupational Safety and Health Administration.

NIOSH – National Institute for Occupational Safety and Health.


Zolpidem (and Zaleplon, Zopiclone)

Zolpidem is a white to off-white crystalline powder.

**Synonyms:** N,N, 6-trimethyl-2-p-tolyl imidazo[1,2-a]pyridine-3-acetamide L-(+)-tartrate; zolpidem tartrate; Ambien®.

**Source:** Zolpidem is available by prescription and is a Schedule IV controlled substance. Ambien® is available in strengths of 5 mg and 10 mg (white and pink oval tablets, respectively). Sonata® contains zaleplon. Imovane® contains zopiclone.

**Drug Class:** Non-benzodiazepine sedative-hypnotic, CNS depressant, sleep aid.

**Medical and Recreational Uses:** Zolpidem is a non-benzodiazepine hypnotic used in short-term treatment (up to 4 weeks) of insomnia. Zaleplon and zopiclone also are indicated for the treatment of insomnia.

**Potency, Purity and Dose:** Recommended zolpidem dose is 10 mg immediately before bedtime (5 mg in the elderly). Recommended nighttime zaleplon and zopiclone doses are 5-20 mg and 7.5 mg, respectively. Patients treated with zolpidem often concurrently use other medications such as antidepressants, narcotic analgesics, and muscle relaxants.

**Route of Administration:** Oral.

**Pharmacodynamics:** While zolpidem has a chemical structure unrelated to benzodiazepines, it is a GABA<sub>A</sub> receptor agonist and shares some of the pharmacological properties of benzodiazepines. Zolpidem preferentially binds to receptors containing an α<sub>1</sub> subunit (also known as BZ1- or ω<sub>1</sub>-receptor subtypes). Zolpidem shortens sleep latency and prolongs total sleep time in patients with insomnia, but has little effect on the stages of sleep in normal subjects. It also has weak anticonvulsant properties. Zaleplon binds preferentially to BZ-1, but also to BZ-2 and BZ-3; while zopiclone binds equally to BZ-1 and BZ-2.

**Pharmacokinetics:** Zolpidem is absorbed readily from the gastrointestinal tract. First-pass hepatic metabolism results in an oral bioavailability of 67%, and 92% is bound in plasma. Zolpidem has a short elimination half-life (2.2 ± 0.4 hours), which is reduced in children (~ 1.4 hours) and increased in the elderly (~ 2.8 hours) and patients with hepatic cirrhosis (~ 9.9 hours). Peak plasma concentrations are detected at 1.5-2.5 hours. Peak concentrations are decreased with food and increased in patients with hepatic insufficiency. Zaleplon has a bioavailability of 30% and has a shorter half-life (1.1 hours) compared to zolpidem.

**Molecular Interactions / Receptor Chemistry:** Zolpidem is converted to hydroxylated metabolites principally by cytochrome P450 3A4 isoenzymes, with minor contributions by 1A2 and 2C9 isoforms. Potential inhibitors of these isoenzymes could decrease the
rate of zolpidem elimination if administered concurrently, while potential inducers could increase the rate of elimination

**Blood to Plasma Concentration Ratio:** Data not available.

**Interpretation of Blood Concentrations:** Single doses of 5 mg zolpidem resulted in average peak concentrations of 0.06 mg/L at 1.6 hours; 10 mg produced 0.12 mg/L at 1.6 hours; 15 mg produced 0.20 mg/L at 1.5 hours; and 20 mg produced 0.23 mg/L at 2.1 hours.

**Interpretation of Urine Test Results:** Urinary excretion of unchanged zolpidem is less than 1%.

**Effects:**
*Psychological:* Sleep induction, drowsiness, dizziness, lightheadedness, amnesia, confusion, concentration difficulties, and memory impairment.
*Physiological:* Nausea, ataxia, slow and slurred speech, slow reflexes, and difficulty with coordination.

**Side Effect Profile:** Somnolence, lightheadedness, vertigo, headache, nausea, fatigue, cognitive deficits, and impairment of consciousness ranging from somnolence to light coma. Infrequently reported side effects include agitation, depressive syndrome, detachment, nightmares, hallucination, leg cramp, paresthesia, speech disorder, double vision, dry mouth, and diarrhea. Hangover effects are unlikely with zolpidem, although morning-after anterograde amnesia may occur. In overdose, patients mainly suffer somnolence and drowsiness, pinpoint pupils, respiratory depression, and in extreme cases, coma and respiratory failure.

**Duration of Effects:** Following 10-20 mg oral doses of zolpidem, effects can last up to 4-5 hours (dose-dependent). There are generally no residual effects the morning after a nighttime dose of zolpidem. Sedation may extend for 8-16 hours following intoxication. Zaleplon has a more rapid onset and shorter duration of effects compared to zolpidem, while zopiclone has longer duration of effects.

**Tolerance, Dependence and Withdrawal Effects:** Tolerance and dependency are not typically detected after 4 weeks of therapeutic use; however, tolerance may develop with chronic use. There is some evidence of tolerance and physical dependency observed with chronic administration of zolpidem in animal models. Withdrawal following abrupt discontinuation may include mild dysphoria and insomnia, abdominal and muscle cramps, vomiting, sweating, tremors, convulsions, fatigue, flushing, lightheadedness, nervousness, and panic attacks.

**Drug Interactions:** Imipramine has an additive effect of decreased alertness; chlorpromazine has an additive effect of decreased alertness and decreased psychomotor performance; ritonavir decreases clearance though inhibiting CYP3A hydroxylation; ketoconazol also decreases clearance; and flumazenil is an effective and therapeutic
pharmacodynamic antagonist. Alcohol increases the sedation and decreases psychomotor performance produced by zolpidem. Other CNS depressant drugs may potentiate the effects of zolpidem. Zopiclone has additional performance decrements when concurrently taken with alcohol, carbamazepine, and diazepam.

**Performance Effects:** Unsteady gait, confusion, disorientation, and significant cognitive and psychomotor impairment can be observed within 1-5 hours following zolpidem doses of 10-20 mg. Memory impairment (learning, recall and recognition of words, pictures, and numbers) psychomotor slowing (digit symbol substitution task, circular light tasks), reduced attentional capacity (impaired divided and sustained attention), impaired balance (ataxia, dizziness), visual disturbances (double vision), and impaired time estimation have been recorded. Psychomotor impairment can be found up to 5 hours after a single 15 mg oral dose and up to 8.25 hours after a 20 mg dose. Memory and learning impairment can be found up to 8.25 hours following a 10-20 mg dose. There has been no significant residual effect on memory or actual driving when subjects have been tested the morning after a single 10 mg dose.

Following a single 10-20 mg dose of zaleplon, studies have shown no residual effects on actual driving (5-10 hours) or on body sway, reasoning, retrieval and spatial memory (4-9 hours); however, significant impairment has been reported within 1-3 hours of dosing. Minor impairment of delayed free recall has occurred 4 hours after 20 mg dose of zaleplon. For zopiclone, a single 7.5 mg dose can cause severe residual effects on actual driving at 5 and 10 hours, severe residual effects on body sway and memory at 4 hours, and minor impairment of delayed free recall 9 hours after dosing.

**Effects on Driving:** The drug manufacturer states that patients should be cautioned against engaging in hazardous occupations requiring complete mental alertness or motor coordination such as driving a motor vehicle. Within the first 4-5 hours, zolpidem can produce significantly impaired coordinative, reactive and cognitive skills following single oral doses of 10-20 mg. However, no significant adverse effects were observed during a 1.5 hour driving test on a rural road, 10-12 hours after drug administration. In five reported cases of driving impairment in which zolpidem was the only drug detected, blood concentrations of zolpidem ranged from 0.08 to 1.4 mg/L (mean 0.65 mg/L). Symptoms and observed behavior included erratic driving (weaving, lane travel), slow and slurred speech, slow reflexes, dazed appearance, disorientation, confusion, loss of balance and coordination, loss of short-term memory, blacking out, somnolence, dilated pupils, double vision, poor performance on field sobriety tests, poor attention, and an inability to stand or walk unassisted. In another six reported cases of driving under the influence of zolpidem, blood concentrations ranged from 0.1 to 0.73 mg/L (mean 0.31 mg/L). The subjects were involved in automobile accidents or were seen to drive erratically, and symptoms included slow and slurred speech, ataxia, unsteady gait, confusion and disorientation.

**DEC Category:** CNS depressant

**DEC Profile:** Horizontal gaze nystagmus present; vertical gaze nystagmus present for high doses; lack of convergence present; pupil size normal; reaction to light slow; pulse
rate down; blood pressure down; body temperature normal. Other characteristic indicators may include slow and slurred speech, somnolence, and poor performance on field sobriety tests.

**Panel’s Assessment of Driving Risks:** Zolpidem causes significant effects when driving within 5 hours of use (10 mg dose). Zaleplon causes significant impairment within 3 hours of use (10 mg), but no significant impairment after 4 hours (10 mg) and 5 hours (20 mg). Zolpidem and zaleplon are relatively free of residual morning-after effects. Zopiclone causes severe impairment 1-5 hours after dosing (7.5 mg), with residual hangover effects up to 10-11 hours.

**References and Recommended Reading:**
Hindmarch I, Patat A, Stanley N, Paty N, Rigney I. Residual effects of zaleplon and zolpidem following middle of the night administration five hours to one hour before awakening. *Human Psychopharmac* 2001;16(2):159-67.
Meeker JE, Baselt RC. Six cases of impaired driving following recent use of the sleep inducer zolpidem (Ambien®). Presented at the American Academy of Forensic Sciences annual meeting, Nashville, TN, February 1996.
Biographical Sketches of Lead Authors and Main Contributors

Lead Authors

Fiona Couper, Ph.D.

Dr. Fiona J. Couper received her B.Sc. (Honors) degree in Pharmacology/Toxicology and her Ph.D. degree in Forensic Medicine/Toxicology from Monash University, Melbourne, Australia. During this period, Dr. Couper also worked as a forensic toxicologist at the Victorian Institute of Forensic Medicine (VIFM) in Melbourne. From 1997-1998, Dr. Couper held a postdoctoral fellowship position at the National Institute of Forensic Sciences and the VIFM, and in late 1998 became a senior research fellow at the University of Washington and the Washington State Toxicology Laboratory, in Seattle, U.S.A. Dr. Couper is now the Chief Toxicologist at the Office of the Chief Medical Examiner, Washington D.C. Dr. Couper’s research has focused on the effects of prescription and illicit drugs on driving impairment, the use of drugs to facilitate sexual assaults, GHB and drug overdoses in the emergency room, and the prevalence of drug use in various community groups. Dr. Couper is also an active member of the Society of Forensic Toxicologists (SOFT), the American Academy of Forensic Sciences (AAFS), and the International Association of Forensic Toxicologists. Additionally, she is the chair of the Joint AAFS/SOFT Drugs and Driving Committee.

Barry Logan, Ph.D.

Dr. Barry K. Logan was born in Bearsden, Scotland, and earned his bachelor's degree in chemistry and Ph.D. in forensic toxicology from the University of Glasgow. In 1986 he accepted a research position in the Department of Toxicology and Chemical Pathology at the University of Tennessee in Memphis. In 1990 he joined the faculty of the University of Washington (UW) in the Department of Laboratory Medicine and was appointed Washington State Toxicologist. In 1999 the Washington State Toxicology Laboratory merged with the Washington State Patrol, and Dr. Logan was named Director of the newly created Forensic Laboratory Services Bureau. In addition to his duties as State Toxicologist and Clinical Assistant Professor at UW, he oversees operations of the State Patrol Crime Laboratories, Breath Test Section, and Implied Consent Section. Dr. Logan has more than 70 publications in the field of forensic toxicology and drug analysis, and is Board Certified by the American Board of Forensic Toxicology. He has been elected to the National Safety Council's Committee on Alcohol and Other Drugs and to the International Council on Alcohol, Drugs, and Traffic Safety, and has served as a consultant to the National Institute of Justice, the United Nations Drug Control Program, and numerous state agencies. He is a Fellow of the American Academy of Forensic Sciences, an active member of the Society of Forensic Toxicologists, and serves on the editorial boards of the Journal of Forensic Sciences and the Journal of Analytical Toxicology. His current research interests include stimulant use and driving impairment, drug interactions and postmortem toxicology, and drug facilitated sexual assault.
Main Contributors

Michael Corbett, Ph.D.

Dr. Michael R. Corbett received his B.Sc., M.Sc. and Ph.D. degrees in chemistry from the University of Toronto, the last being conferred in 1989. He is also the coordinator, and an instructor, in the forensic science courses offered through the School of Continuing Studies at the University of Toronto, and has supervised undergraduate students in research projects at the Department of Pharmacology. Dr. Corbett received the prestigious "Excellence in Teaching Award" for overall cumulative achievement in 2001. Dr. Michael Corbett is currently a senior forensic toxicologist in the Province of Ontario in Canada. In the area of alcohol, other drugs, and the operation of motor vehicles, Dr. Corbett has been directly involved in over 2500 cases. He is a designated analyst pursuant to the Criminal Code of Canada. He has provided educational programs on alcohol screening devices and instruments, including human subject testing, to police, lawyers, judges, media, and university students. Dr. Corbett serves as a member of the editorial board of the Journal of Analytical Toxicology. He belongs to numerous professional peer organizations including the AAFS, SOFT and The International Association of Forensic Toxicologists (TIAFT). He also participates in committees including the Committee on Alcohol and Other Drugs of the Highway Traffic Safety Division of the National Safety Council and the Joint AAFS/SOFT Drugs and Driving Committee. Dr. Corbett is certified as a Diplomat in Forensic Toxicology by the American Board of Forensic Toxicology (D-ABFT).

Laurel Farrell, M.S.

Ms. Laurel J. Farrell received her B.A. in Chemistry from the University of Northern Colorado in 1979. Ms. Farrell then worked for the Colorado Department of Public Health and Environment for over twenty-one years serving in a variety of capacities in the drug and alcohol analytical laboratories. For the last half of her employment she served as the staff authority in the toxicology laboratory routinely providing expert testimony in Colorado courts and in US District Court on the effects of alcohol and other drugs on human performance. For the last two and half years, Ms. Farrell has been assigned to the Colorado Bureau of Investigation's Denver Laboratory. She is a member of several professional organizations. As an active member of the Society of Forensic Toxicologists, she has just finished seven years as an officer/director serving as President in 2002. She is a Fellow of the American Academy of Forensic Sciences and served as Chair of the Joint AAFS/SOFT Drugs and Driving Committee from 2000-2002 and as a member on this committee from 1995 to the present. Over that time period, Ms. Farrell has assisted in coordinating a number of continuing education workshops in the area of drug impaired driving and has recently served a guest editor for two volumes of Forensic Science Review focusing on the Effects of Drugs on Human Performance and Behavior. She is also an elected member of the National Safety Council's Committee on Alcohol and Other Drugs and the International Council on Alcohol, Drugs, and Traffic Safety.
Marilyn Huestis, Ph.D.

**Dr. Marilyn A. Huestis** is the Acting Chief, Chemistry and Drug Metabolism Section (CDM), Clinical Pharmacology and Therapeutics Research Branch, Intramural Research Program (IRP), National Institute on Drug Abuse (NIDA), NIH. Dr. Huestis conducts controlled drug administration studies and directs the core chemistry laboratory of the IRP, NIDA. She has worked in the fields of clinical and emergency toxicology, therapeutic drug monitoring, urine drug testing, and forensic toxicology, which have provided a unique background and the knowledge and experience necessary for drug abuse research. Her research focuses on the pharmacodynamics and pharmacokinetics of drugs of abuse. Special areas of interest include cannabinoids, alternate matrices for drug analysis, correlations of blood levels of drugs with performance effects, medication development projects including the buprenorphine as a pharmacotherapeutic agent in opioid dependence, and in utero drug exposure. Pregnant opiate addicts receiving buprenorphine or methadone as part of their treatment program have provided a unique opportunity to study the disposition of drugs in the mother and fetus, and the relationship between drug concentrations in a wide variety of biological specimens and maternal and neonatal outcome measures. Dr. Huestis hopes to develop a better understanding of drug abuse in women and the consequent drug exposure of neonates and children. Dr. Huestis is the principal investigator of several phase I clinical studies evaluating the effects of the cannabinoid receptor antagonist, SR 141716 in cannabis users. Dr. Huestis received a bachelor’s degree in biochemistry from Mount Holyoke, a master’s degree in clinical chemistry from the University of New Mexico, and a doctoral degree in toxicology from the University of Maryland in Baltimore. Dr. Huestis has been working in the fields of forensic and analytical toxicology, and clinical chemistry for more than thirty years and is recognized nationally and internationally for her contributions to the field. She has published extensively in these fields and serves on the Editorial Board of the Journal of Analytical Toxicology. She is an Adjunct Associate Professor in the Toxicology program of the University of Maryland at Baltimore and directs graduate and post-graduate student research. Dr. Huestis is currently President of the International Association of Forensic Toxicologists, past president of the Society of Forensic Toxicologists (SOFT) and past Chair of the Toxicology Section of the American Academy of Forensic Sciences. Dr. Huestis is also a member of the International Cannabinoid Research Society, American Association for Clinical Chemistry, the International Association of Therapeutic Drug Monitoring and Clinical Toxicology, the California Association of Toxicologists, Society of Hair Testing, and the United States Anti-Doping Agency Research Advisory Board.

Wayne Jeffrey, M.S.

**Mr. Wayne K. Jeffery** received his B.Sc (Pharmacy) degree in 1968 and M.Sc. (Pharmaceutical Chemistry) degree in 1971, from the University of Alberta, Edmonton, Alberta, Canada. He has been the Toxicology Section Head, Royal Canadian Mounted Police, Forensic Laboratory, Vancouver, since 1976. Mr. Jeffery is a member of 7 professional associations, including the Alberta Pharmaceutical Association and the Canadian Pharmaceutical Association. He has been a member of the Canadian Society of
Forensic Sciences, Drugs and Driving Committee since 1986 and has been chairman since 1994. He is the co-coordinator of the DRE/SFST Program in British Columbia and is the DRE coordinator for Canada. Mr. Jeffery has 19 scientific publications dealing with all aspects of Forensic Alcohol and Toxicology including 3 chapters in published books. He has given training on drug identification and identifying the drug user to Police forces in Asia, Caribbean, Central and South America and Europe; and is a lecturer on the following Police courses: Drug Identification, Drug Undercover Investigative Techniques, Clandestine laboratory Investigations and Chemical Safety and Drug Awareness Training.

Jan Raemakers, Ph.D.

Dr Jan Ramaekers obtained his Ph.D. in psychopharmacology from Maastricht University, on behavioral toxicity of medicinal drugs. Dr Ramaekers spent 8 years of research at the Institute for Human Psychopharmacology at Maastricht University. During these years he conducted a large number of experimental studies on the effects of medicinal drugs, such as antidepressants, antipsychotics, anxiolytics, anticonvulsants and antihistamines on cognition, psychomotor function and actual driving performance of healthy volunteers and patients. In 1995, the Institute for Human Psychopharmacology received the Widmark Award (International Counsel of Alcohol, Drugs and Traffic Safety), “for numerous contributions to the advancement of the cause of alcohol, drugs and traffic safety and sustained contributions to the support in this field”. In 1998, Dr Ramaekers accepted a position as Assistant Professor at the Faculty of Psychology at Maastricht University. He has been a co-organizer of courses in the field of Human Psychopharmacology, Biological Psychology and Traffic & Aviation Psychology. Dr Ramaekers is currently involved in research on the effects of illicit drugs, i.e. marijuana and MDMA, on driving. He is a member of the British Association of Psychopharmacology (BAP), the Collegium Internationale Neuro-Psychopharmacologicum (CINP) and the International Counsel of Alcohol, Drugs and Traffic Safety (ICADTS).