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Methods: Officers follow a 12 step testing sequence and evaluate signs such as pupil sizes and responses, eye movements, heart rate, body temperature, mental timing, and balance. A matrix is then used to compare the subject's signs to those that would be produced by the seven types of drugs. If a pattern match is found, the officer concludes that the subject is under the influence of a drug and specifies the drug type.

Results: Several field and laboratory validation studies have been conducted using these procedures. In general, officers were 70 to 90% accurate in determining intoxication status and drug classification, but poly-drug use and drug rebound effects can sometimes cause problems.

Summary: Ocular and other physiological signs can be used to detect drug intoxication and classify the type of drug taken. Knowledge of the procedures used in the Drug Recognition Program can enable optometrists to serve as consultants to the police and as expert witnesses in cases involving the use of ocular signs that indicate drug use.

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THE DRUG EVALUATION CLASSIFICATION PROGRAM:
USING OCULAR AND OTHER SIGNS TO DETECT INTOXICATION

By

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A thesis submitted to the faculty of the
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BIOGRAPHY

Edward Kosnoski is from Kent, Washington. He received a Bachelor of Arts degree from Washington State University in 1992, and will graduate from Pacific University's College of Optometry in 1998. He plans to practice in the Seattle area upon graduation.

Abstract

Background: A systematic approach to determining drug intoxication has been developed for use by police officers. By considering specific physiological signs, trained officers can detect the effects of seven major drug types.

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Summary: Ocular and other physiological signs can be used to detect drug intoxication and classify the type of drug taken. Knowledge of the procedures used in the Drug Recognition Program can enable optometrists to serve as consultants to the police and as expert witnesses in cases involving the use of ocular signs that indicate drug use.

Introduction

Consider the following scenario. A police officer on patrol observes a car weaving within it's lane, crossing the yellow line, and changing speed for no apparent reason. At this point the officer has probable cause to make a traffic stop and does so. During the stop, the officer observes the suspect's behavior, checks for the presence of drugs[d] or alcohol in the vehicle, and can perform standard field sobriety tests, if appropriate. Then, if the officer is satisfied that the suspect is intoxicated or impaired, constitutional rights are read, an arrest is made, and the person is transported to a police station. At the station, the option of providing or refusing a breath sample that can be used to determine blood alcohol level is presented. If the suspect refuses, penalties can include loss of driving privileges for an extended period. If a breath sample is provided and it indicates a blood alcohol concentration (BAC) above the legal level, usually 0.08 or 0.10 mg/ml depending on state law, the suspect is charged with driving while intoxicated and is taken to jail to await release to a responsible person.

This scenario changes somewhat if the suspect appears to be impaired or intoxicated but has a BAC below the legal limit. At this point there is a strong possibility of intoxication with a drug other than or in addition to alcohol. In Oregon, a person arrested for driving under the influence of intoxicants (DUII) is required to provide a urine specimen that can be analyzed for metabolites of various drugs.[1] Unfortunately, even if the analysis is positive for metabolites of illegal or intoxicating drugs, it might be insufficient evidence to obtain a conviction for DUII.

In theory, and perhaps in practice, a defense attorney could concede that the suspect had taken a drug, for example cocaine, but could also argue that the presence of metabolites in the urine did not mean that the suspect was under the influence of the drug at the time of arrest. This is a viable defense in Oregon because it is currently not illegal to be under the influence of a drug; it is only

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illegal to possess or sell a drug, or to operate a vehicle under its influence.[2] The problem for the police is then to convince a judge or jury that the suspect was indeed under the influence of the drug at the time of arrest. To address this and related problems, the Drug Evaluation Classification Program (DECP) has evolved.[e,3]

The Drug Evaluation Classification Program

In over half of the states and several foreign countries, selected police officers have been trained as Drug Recognition Experts (DREs). Training includes 72 hours of formal classroom education, a certification phase during which officers evaluate a minimum of six drug-intoxicated subjects, assist in the evaluation of six more, and take a comprehensive written examination.[4]

DREs are called upon to examine suspects who are believed to be under the influence of drugs, but who do not have sufficiently high BACs to justify a charge of driving under the influence of alcohol. DREs observe and quantify a variety of physiological and psychological signs to determine if the suspect was under the influence of a drug at the time of arrest and to determine what type of drug(s) the suspect had taken. Testimony by the DRE based on these signs is usually sufficient to establish the suspect's intoxication status, but credibility of the testimony is enhanced when the laboratory tests of urine metabolites correspond to the DRE's determination of what type of drug(s) the suspect had taken.

Overview of Paper

In this paper, the most commonly abused drugs will be described and the methods used by DREs to detect their use will be presented. Emphasis will be placed on ocular signs that aid DREs in making their determinations. Material that is not otherwise referenced is drawn from personal observations of the authors and from the Instructors' Version of the manual used to teach the DRE training course.[3]

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Commonly Abused Drugs

DREs are trained to detect the effects of seven drug categories. A brief summary of the effects produced by these drugs is shown on Tables 1 and 2. Table 3 shows the street names and approximate costs for several of the commonly abused drugs.

Insert Tables 1, 2 and 3 About Here

Central Nervous System Depressants

Typical agents

Alcohol (ETOH) is probably the most commonly abused central nervous system (CNS) depressant. Other drugs in this class include barbiturates (e.g., Phenobarbital); non-barbiturates (e.g., chloral hydrate, Quaalude, and Soma); minor tranquilizers (e.g., benzodiazepines such as Valium, Ativan, Halcion, and Xanax); antidepressants (e.g., Elavil, Sinequan, Prozac, Paxil, and Tofranil), and similar drugs that can act as CNS depressants when taken in large quantities; major tranquilizers (e.g., Thorazine, Haldol, and lithium); and combination drugs.[3]

Most of the drugs in this category can be consumed by mouth as pills, tablets, or liquids. Some can also be taken rectally to avoid destruction in the digestive system and/or rapid break down by the liver. Drugs taken in this way reach the brain via the circulatory system without first passing through the liver as would happen if they were taken orally.

The barbiturates are typically taken either orally or injected intravenously. Because of the large gauge needle required to inject the barbiturate solution and its high alkalinity, injections produce significant areas of skin swelling and possible necrosis up to several centimeters in diameter.

The effects of drugs in this category can be additive. For example, the combination of relatively small quantities of alcohol with other CNS depressants (including prescription drugs taken in

prescribed amounts) might create intoxication and driving impairment which can justify arrest and conviction.

Mode of action

In general, CNS depressants other than the benzodiazepines are non-selective in action and depend on the dose taken as well as the situation and mood of the user.[4] Most of these drugs seem to act with varying potency at different neuronal sites by inhibiting neurotransmission at synapses mediated by gamma-aminobutyric acid (GABA), whereas benzodiazepines specifically potentiate the neural inhibition mediated by GABA.[4]

The half-life of a drug provides an indication of the length of time that a drug is active, specifically how long it takes for 50% of the drug to be eliminated. The half-lifes of CNS depressants vary greatly from as little as two hours for Halcion to as much as 120 hours for Phenobarbital.[4]

As compared to several other abused drugs, CNS depressants have relatively little direct effect on the autonomic nervous system. For this reason, most do not produce significant pupillary miosis or mydriasis. However, these drugs can have deleterious effects on the suspect's smooth pursuit eye movement system.

The systems that control smooth pursuits and saccades have common final pathways (extraocular muscles and their related nuclei), but they have separate control centers at the brain stem and cerebellar levels.[5-7] These control centers appear to be differentially sensitive to the effects of certain drugs, such as depressants, with the smooth pursuit centers being the most sensitive. When the smooth pursuit system is compromised, persons lose their ability to track a slowly moving stimulus and smooth pursuits deteriorate into a series of catch-up saccades. Additionally, in eccentric gaze if the smooth pursuit system cannot make small refixation movements to compensate for drifts back toward center, the drifts become so large that they must be compensated for by saccades. The lack of ability to make smooth

pursuits and the drift-saccade refixation motions made in lateral gaze are used by police officers to detect the influence of depressant drugs. Along with a distinct and prolonged endpoint nystagmus, they form the horizontal gaze nystagmus test (HGN).[8,9]

<u>Behavioral and Physiological Effects</u>

When used at therapeutic levels, the body and brain are calmed, slowed, and relaxed by these drugs. At abuse levels, some of the drugs produce an initial state of excitement followed by reduced social inhibitions, slowed reflexes, possible euphoria, and reduced ability to divide attention between multiple tasks. Persons who take these drugs at abuse levels look, act, and feel drunk.

The CNS depressants slow many aspects of the body's physiology which results in decreased core temperature, respiration rate, blood pressure, and heart rate. Death can occur from severe CNS depressant overdose.

<u>Specific effects on eyes and vision</u>

Persons intoxicated with CNS depressants will typically show bilateral ptosis, difficulty converging, exaggerated end-point nystagmus, failure of smooth pursuit, lateral gaze nystagmus, and occasional diplopia. Those who have consumed very high levels might also show vertical gaze nystagmus. Pupil size and action will be normal except if the person has taken an overdose of Quaalude or Soma in which case the pupils might be dilated.[3] Reasons for this dilation are not well understood.

<div align="center"><u>Central Nervous System Stimulants</u></div>

<u>Typical Agents</u>

CNS stimulants can de divided into three sub-categories: cocaine, amphetamines, and other substances.[3] Cocaine is derived from an evergreen plant native to South America and has been used by natives of that area for centuries. In the United States, cocaine supposedly enjoyed a short period of popularity during the early 1900s as one of Coca Cola's ingredients, but has more recently become a major illicit drug world wide. Cocaine can be taken orally,

smoked (when a "rock" of cocaine is smoked in a pipe by heating it with a cigarette lighter, it makes a crackling sound, hence the term "crack cocaine"), snorted into the nose as a powder, or injected intravenously. Especially when injected, there is an initial intense rush of euphoria and excitement. These feelings continue for up to several hours after which there can be a rebound depression of CNS activity.

Partly because the effects of cocaine use are short-lived, some users have progressed on to the amphetamines, especially methamphetamine, because its effects last much longer. In some regions of the United States (e.g., Oregon), methamphetamine abuse has become almost epidemic. Like cocaine, methamphetamine can be taken orally, snorted, injected, or smoked. The form prepared for smoking looks like clear crystals and carries the street name "ice." Effects of methamphetamine are similar to those produced by cocaine except that they are initially somewhat less intense and can last for up to eight hours.

Other abused CNS stimulants include Ritalin and Cylert which are prescribed for attention/hyperactivity problems, and several drugs such as Preludin that are prescribed for weight control.

Mode of action

Cocaine stimulates the CNS in general, presumably by selectively depressing the activity of inhibitory neurons. Organs that are enervated by the sympathetic nervous system show potentated responses to direct stimulation, to norepinephrine, and to epinephrine, because cocaine blocks the uptake of catecholamines at nerve endings that would otherwise terminate neural activity.[10]

Amphetamines and other drugs produce general stimulation of the CNS by potentiating the release of neurotransmitters such as norepinephrine from nerve terminals.[11] These drugs have varying effects on peripheral organs, but many of the effects are consistent with an increase in sympathetic nervous system activity.

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The half-lifes of most CNS stimulants are generally short, usually about one to two hours, although certain drugs may have half-lifes of several hours.

Behavioral and Physiological Effects

CNS stimulants accelerate most of the body's functions including heart rate, blood pressure, and respiration rate. Core body temperature is also increased, and body tremors are often present. Persons high on stimulants tend to be very talkative, euphoric, and restless. Chronic users will often show signs of nasal irritation if the drugs are snorted and flattened teeth caused by bruxism.[3]

Speed of mental processing is usually increased so that estimates of time and distance can be significantly inaccurate. When asked to estimate 30 seconds mentally, "speeders" might actually estimate the passage of 30 seconds in less than 10 seconds. This effect is well known to many stimulant users so when asked by an officer to estimate 30 seconds, they sometimes count what they believe to be 30 seconds three or four times consecutively.

Because of unpleasant sensations that occur as the drug level drops in the body (rebound effects), the temptation to take more of the drug is strong and many users binge on stimulants for several days at a time.

Specific effects on eyes and vision

The muscle tremors evident in persons who are abusing CNS stimulants are also seen in the eyelids, especially when the lids are partially closed. Convergence abilities, smooth pursuits and the ability to hold the eyes in lateral gaze appear relatively normal, but the classic ocular sign of stimulant use is dilated pupils that react slowly to light. This is due to the sympathetic nervous system effects of the drugs.[3] Because of the mydriasis produced by stimulants, it is common for users to wear sunglasses even in dim light situations.

Hallucinogens (Psychedelics)

Typical Agents

Many substances can produce hallucinations. These include natural materials such as mescaline (derived from the peyote cactus), psilocybin (derived from mushrooms), nutmeg, morning glory seeds, Jimson weed, and bufotenine produced by skin glands of certain toads. Perhaps more well known are the synthetic substances including lysergic acid diethylamide (LSD), ecstasy (also known as MDMA - methylenedioxymethamphetamine), and STP (also known as DOM - dimethoxylamphetamine). The latter two substances are known as psychedelic amphetamines and produce effects that combine those of the hallucinogens and CNS stimulants.[3]

Mode of action

LSD and related drugs are active throughout the CNS but their specific effects are not yet well understood. Some research suggests that LSD has agonist effects at presynaptic receptors for a specific inhibitory neurotransmitter 5-HT in the midbrain, and this results in reduced neural firing rates.[12]

No specific effects on the autonomic nervous system have been documented for the natural psychedelics, but "bad trips" and other psychological consequences of intoxication with these drugs can certainly produce abnormal autonomic activity. The psychedelic amphetamines affect the sympathetic nervous system and can cause pupil dilation.[3]

The half-life of most hallucinogens is about three hours.[12]

Behavioral and Physiological Effects

Agents in this category can significantly distort sensory inputs to the brain and produce mental perceptions not related to reality. Another common effect is synesthesia which involves a combining of sensory perceptions, e.g., sounds can take on chromatic characteristics or aromas.

The ability to perform skilled tasks is significantly diminished for persons under the influence of hallucinogens because

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they produce distractions that prevent concentration on the task. With the psychedelic amphetamines, blood pressure, pulse rate, and body temperature are typically elevated. Body muscle tremors and uncontrolled movements might also be evident.[3]

Specific effects on eyes and vision

Aside from synesthesia, the psychedelic amphetamines can produce pupillary dilation via presumed effects on the autonomic nervous system.[12] Convergence, smooth pursuit, and lateral gaze will appear normal for persons who have taken drugs in this classification.

Dissociative Anesthetics (PCP and Analogs)

Typical Agents

Substances in this category include phenylcyclohexyl piperidine, usually called phencyclidine or PCP, and an analog called ketamine. Ketamine is currently used as a veterinary anesthetic under the trade names Ketalar, Vetalar, and Ketaject.[3] This has made some veterinarian offices the subject of thieves looking for ketamine.

PCP and its analogs have been termed dissociative anesthetics because they seem to dissociate the brain from pain sensations. They originally showed promise as anesthetics that could leave patients conscious but not responsive to pain during surgery. Unfortunately, a number of "bad trips" during and after surgery caused the manufacturer to remove the drug from human clinical use in the late 1970s.

PCP is most commonly used by mixing it with marijuana or tobacco and smoking it. Because PCP burns at very high temperatures, some users prefer to smoke it with mentholated tobacco or chew mint leaves while smoking. Besides smoking, PCP can be taken orally, snorted, or injected. Depending on the amount taken and mode of administration, effects of PCP last from several hours to a day or two.

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Mode of action

The specific modes of action for the dissociative anesthetics have not yet been completely defined.[12] It is believed that PCP inhibits the uptake of the neurotransmitter dopamine and this suggests that the drug might temporarily mimic the mental illness schizophrenia.[13] In both PCP users and schizophrenics, cognitive deficits and abnormal dopamine levels in certain brain nuclei can be detected.

No specific autonomic nervous system effects are known to occur as a result of PCP use, but the effects on smooth pursuit eye movements are similar to those produced by depressants.

Behavioral and Physiological Effects

Because PCP renders a person insensitive to pain, individuals under its influence can be quite dangerous. Anecdotal stories abound regarding persons high on PCP having multiple limbs broken or being shot several times and still continuing to fight police officers. It is an understatement to note that the police treat suspected PCP users with a considerable degree of caution.

Psychologically, PCP seems to produce a combination of CNS stimulation and depressions along with hallucinations. Physiologically, there is increased muscle tone along with elevated pulse rate, blood pressure, and body temperature.[3] Because of this increased temperature, it is not uncommon for PCP users to remove their clothing or to break through windows because of their resemblance to the surface of water.

Specific effects on eyes and vision

Because PCP mimics some aspects of CNS depressants, persons who have taken PCP will experience convergence problems, exaggerated end-point nystagmus, failure of smooth pursuit, lateral gaze nystagmus, and possibly diplopia. Typically, the onset of lateral gaze nystagmus will occur at less than 30 degrees and there will be up-beating vertical gaze nystagmus. Pupil size and action will typically be normal.[3]

Narcotic Analgesics (Opioids)

Typical Agents

These drugs are either natural alkaloids derived from opium or synthetic substances. They are used to reduce the brain's perception of pain and should not be confused with dissociative anesthetics which seem to actually block the pain information.

Natural alkaloids include powdered opium, morphine, codeine, and heroin which is the most commonly abused of the these substances. Natural alkaloids formulated as prescription drugs include Dilaudid, Hycodan, and Percodan. Synthetics include Demerol, a family of substances known as fentalyls, and Methadone which is used to treat heroin addicts. Methadone is a synthetic narcotic analgesic that does not provide the intense high associated with heroin. Its effects last long enough so that it can be taken once a day to prevent or reduce the withdrawal symptoms.[3]

Drugs in this class can be taken orally, smoked, or injected. Intravenous injection is a common mode of heroin use and the resultant needle tracks on the arms, legs, and other sites indicate the extent of drug use.

Mode of Action

Depending on the site of administration, narcotic analgesics produce their effects by inhibiting pain neurons in the spinal cord and elsewhere within the CNS.[12] Analgesia results from inhibition of the release of neurotransmitters by primary afferent neurons.[12]

The half-lifes of natural alkaloids, such as morphine and heroin, may be as short as one-half to two hours, whereas synthetics such as Methadone can have half-lifes as long as 40 hours.[12]

Behavioral and Physiological Effects

Use of narcotic analgesics produces a dreamy, euphoric state with a reduction in pain sensation. A major problem for persons who abuse this class of drugs is that tolerance builds rapidly and increasingly large amounts must be taken to achieve the pleasant

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effects - or prevent the extremely unpleasant effects of withdrawal. Because withdrawal is so unpleasant, some abusers reach a point at which they take just enough of the drug to prevent withdrawal and can function at a relatively normal level, or they become involved in a Methadone maintenance program.

Persons under the influence of narcotic analgesics usually have droopy eyelids and appear to drop off to sleep frequently. However, they are not truly asleep because they respond to verbal commands and other sensory stimuli. This condition is referred to as being "on the nod." Other body processes are slowed; blood pressure, heart rate, and core temperature are all decreased from normal values.[3]

Typically, the pleasant effects of these agents last up to six hours, but influenza-like rebound effects including chills, elevated body temperature, and cramps can occur one to two days after drug use.

Specific effects on eyes and vision

The hallmark visual effects of narcotic analgesics are constricted pupils (often less than 3.0 mm in the dark) and droopy eye lids. Typically, no abnormal horizontal or vertical nystagmus is noted, and the ability to converge is not affected. During the rebound phase as the drug effects wear off, excessive pupillary hippus will sometimes be noted; during withdrawal from prolonged drug use, pupillary dilation is also possible.[3]

Inhalants

Typical Agents

Substances that are inhaled include chemicals such as acetone, toluene, benzene, gasoline, and kerosene. These and other substances are obtained from dry cleaning fluids, model airplane cement, paints, fingernail polish remover, etc.[3]

Persons also inhale propellants from aerosol containers that contain cooking sprays, paints, insecticides, hairs spray, etc. The propellants include various hydrocarbons, all of which are relatively

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toxic. Abuse of inhalants seems to be more common among children in poor areas because of the easy accessibility of aerosol containers. Beyond the effects of the propellants, the materials, such as paint pigments also contained in the aerosol sprays are capable of causing significant damage to the body.

Another class of abused inhalants are the anesthetic gases. These include ether, chloroform, nitrous oxide, and amyl nitrate. Nitrous oxide is an especially interesting inhalant because it is used as a propellant in several brands of aerosol whipped cream containers and is available over-the-counter in small cartridges termed "whippets" for home or commercial whipped cream production.

Inhalants are often taken by soaking rags with the materials and/or concentrating the gases in bags or balloons. Typically, the onset of effects produced by inhalants is immediate with the high lasting from less than five minutes to several hours or more.

Mode of Action

In general, inhalants produce depression of the CNS, resulting in disinhibition. The exact neural effects are uncertain for most substances because of their complex chemistries.[12] No specific autonomic nervous system effects are known to occur as a result of inhalant use, but the effects on smooth pursuit eye movements are similar to those produced by depressants.

Behavioral and Physiological Effects

The effects of inhalants vary somewhat depending on what substance was inhaled, but they typically involve euphoria, feelings of grandiosity, bizarre thoughts, and distorted perceptions of time and space.

Many of the abused inhalants can have severe long- and short-term effects on the body by producing damage to the brain, respiratory system, liver, kidneys, and bone marrow. Physiological addiction to inhalants is rare, but psychological habituation is possible.

Because of the many substances that can be abused, it is difficult to make generalizations about their effects on blood pressure or body temperature. However, pulse rate is typically increased.[3]

Specific effects on eyes and vision

Like the CNS depressants and PCP, inhalants can produce exaggerated end-point nystagmus, failure of smooth pursuit, lateral gaze nystagmus, and, for high doses, vertical nystagmus. Persons intoxicated with these agents can have difficulty with convergence and may have normal or slightly dilated pupils.

Because many of the inhalants are irritating to the eyes, conjunctival injection and watering will often be seen for a period of time after use.

Cannabis

Typical Agents

Marijuana and related products are derived from the various species of the cannabis plant. The primary active ingredient is delta-9 tetrahydrocannabinol (THC), but there are many other substances in the plant that also have physiological and/or psychological activity.

Typically the leaves and flowers of the plant are dried and smoked or eaten, but they can also be crushed and boiled to create hashish which can then be pressed to produce hashish oil.[3]

Synthetic THC is also available as Marinol or Nabilone and can be prescribed for cancer or AIDS patients to reduce vomiting and stimulate the appetite.

Mode of Action

The exact neural effects of cannabinoids are uncertain, but they probably act at multiple receptor sites within the CNS. In addition, physiological effects and patterns of action vary depending on the agent used.[12]

<u>Behavioral and Physiological Effects</u>

Cannabis is most commonly taken by smoking the plant leaves and flowers, or by adding hashish to other substances. Cannabis produces a pleasant, euphoric state in which visual and other stimuli can become more vivid. In the intoxicated state, short-term memory and attentional processes are impaired. Depending on the mode of ingestion, the effects of cannabis occur within a few minutes and can last several hours.

If cannabis has been smoked recently, it is common to find bits of plant material in the mouth, the back of the tongue might have a greenish coating, and the taste buds can be raised.

<u>Specific effects on eyes and vision</u>

Cannabis will typically not produce exaggerated end-point nystagmus, failure of smooth pursuit, lateral gaze nystagmus, or vertical nystagmus. However, persons who have ingested cannabis can show significantly injected conjunctival vessels, problems with convergence, possibly dilated pupils, tremors when the lids are partially closed, and pupils that exhibit rebound dilation.[3]

Rebound dilation can be demonstrated by dark adapting the suspect for at least 90 sec and then illuminating the eye directly with a pen light. Normally the pupil will constrict and stay constricted as long as the light is on. But, with rebound dilation, the pupil will initially constrict, then it will open slightly, constrict again, open more, constrict again, etc. for several cycles. Rebound dilation can be differentiated from hippus because with rebound dilation the pupil gets larger on each cycle whereas with hippus the oscillation is around a constant pupil diameter.

Twelve Step DRE Evaluation Process

Suspects are evaluated by DREs using a standardized 12 step procedure.[3] This standardization assures that no important sign will be missed, allows comparison of results from one police jurisdiction to another, and assists the DRE in establishing credibility in court.

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Step 1. Determination of Blood Alcohol Concentration

The initial test involves determining whether the suspect has a BAC above the legal limit for driving in the particular jurisdiction where the arrest was made. Typical BAC limits range from 0.08 to 0.10 mg/ml. If the suspect is over the legal limit, the DRE evaluation procedure normally stops because a legally sufficient reason for the suspect's impaired driving has been established.

Step 2. Interview of the Arresting Officer

If the suspect's BAC is below the jurisdiction's legal limit or is not consistent with the observed impairment, the DRE then interviews the arresting officer to obtain information about driving, statements the suspect has made, whether any paraphernalia or actual drugs were found, etc.

Step 3. Preliminary Evaluation

Preliminary evaluation of the suspect by the DRE consists of a brief interview, assessment of speech ability and content, and a determination of whether the suspect might be sick or injured. Questions include those related to diabetes, epilepsy, any other medical or visual problems, whether the suspect is under the current care of a physician or dentist, and whether any prescription medications are being taken. If medical problems are detected, the DRE will refer the suspect for the proper care.

During this phase of the evaluation, the suspect's pulse rate is measured. Pulse rate will then be measured two more times during the DRE's testing to assess any trends, i.e., is the drug effect becoming stronger or wearing off during the course of testing. DRE protocols set normal pulse rate limits of between 60 and 90 beats per minute.

Step 4. Eye Movement Evaluation

The suspect is next asked to remove any eyeglasses, and the DRE assesses the ability of the eyes to track an object (e.g., finger, penlight, or other suitable stimulus) held at approximately 15 inches from the suspect's face and moved horizontally. If the suspect

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cannot track or if some other problem is noted (e.g., the suspect has an artificial eye), this part of the test battery is usually omitted and a notation is made on the DRE's evaluation form.

Assuming that the suspect can track with both eyes moving symmetrically, the HGN testing sequence is started. The first step involves assessing the ability to track an object moving about 120 degrees from left to right in four seconds and then back the other way. If the pursuit system is intact, tracking will be smooth and accurate. If the system is compromised, tracking will be inaccurate with eyes frequently falling behind or jumping ahead of the target as saccades are used to compensate for differences between eye and target positions. This will be perceived by the DRE as lack of smooth pursuit. (Figure 1)

Insert Figure 1 Showing HGN Testing About Here

The second test in the HGN battery involves assessing eye movements at the full extent of lateral gaze. Most normal persons will have a few "jerks" when the eyes are moved to this position, but intoxicated persons will have distinct jerking that persists for at least four seconds.

The third test begins with the eyes in the straight ahead position and fixated on the DRE's stimulus which is positioned about 15 inches from the suspect's nose. The target is moved slowly to the side and the eyes are observed for evidence of nystagmus. If it is detected, the angle of onset is noted.

There is a tendency for the eyes to drift back toward the primary position from a lateral gaze position, but in non-intoxicated persons this drift is compensated for by tiny refixation movements that might involve the smooth pursuit system. In intoxicated persons, the drift becomes considerably larger before the system initiates a compensatory movement. This is seen as a nystagmus

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with the slow phase drift toward the nose and a temporal fast phase jerk.

The extent of lateral gaze required to produce nystagmus is generally related to the degree of intoxication. DREs regard nystagmus with an onset of between zero and 30 degrees as having an immediate onset which indicates significant intoxication. Onsets from 30 to 45 degrees can also indicate intoxication.

If the suspect is intoxicated with alcohol alone, a 40 to 45 degree angle of onset typically indicates a BAC of between 0.05 and 0.10. When other drugs are present, an accurate BAC cannot be determined from the onset angle, but smaller angles usually indicate greater intoxication.

Scoring of the HGN battery gives one point per eye for failure of smooth pursuit, distinct and sustained end-point nystagmus, and nystagmus with an onset of 45 degrees temporal gaze or less. Of the six possible points, four or more are considered to be indicative of intoxication.

Suspects are also checked for the occurrence of vertical nystagmus by moving a stimulus to the upper limit of gaze and holding it there for four seconds. Finally, the ability of the suspect to converge is evaluated by moving a stimulus in a 12 inch diameter circle 15 inches from the nose to establish tracking and then moving it to the bridge of the nose. Using DRE protocols, the suspect has "lack of convergence" if both eyes cannot track the stimulus to the nose.

Step 5. Divided Attention/Psychophysical Tasks

Even non-intoxicated persons have a limited ability to process sensory information and focus attention on multiple tasks simultaneously. For intoxicated persons, this is more of a problem because abnormal vestibular signals and slowed reaction times produce balance difficulties which must be compensated for. Altered mental states (e.g., hallucinations) also consume attentional resources.

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Four specific tests are performed by the DRE as part of this battery. The first involves having the suspect stand with feet together, tip the head back slightly, close the eyes, and mentally estimate the passage of 30 seconds. Significant sway and/or inaccurate time estimates are indicators of drug intoxication. Suspects who have taken stimulants and whose "mental clocks" are running too fast will often estimate 30 seconds to take less than 10 seconds. Those who have taken depressants or narcotics will have slowed mental clock speeds, and those who have taken hallucinogens can lose track of the task and never reach 30 seconds. DRE protocol limits for normal responses are 30 seconds plus or minus five seconds.

Next, the person is placed with one foot ahead of the other and given relatively complex instructions to walk nine paces heel to toe, make a turn as indicated by the officer, and walk back nine paces. Inability to follow instructions, sway, and mis-steps count against the suspect. Then the suspect is asked to stand for 30 seconds with one foot at a time elevated. The fourth task involves having the suspect touch the nose with the tip of the finger on command. Inability to do these tasks correctly suggests impairment.

Step 6. Vital Signs

During this phase of testing, DREs measure pulse rate for the second time. They also determine blood pressure and body temperature (sublingually) using standard clinical techniques. DRE protocol limits for normal blood pressure are 120 to 140 mmHg systolic and 70 to 90 mmHg diastolic. Body temperature protocol limits are 98.6° F plus or minus 1.0° F.

Step 7. Dark-Room Evaluation

During this phase of the examination, the suspect is taken into a room that can be darkened. Pupil sizes are measured in normal room light and after 90 seconds of total darkness (using the minimum light required to see the pupils). They are also measured with indirect light supplied by a penlight held tangent to the front of

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the eye and with the penlight directed straight into the eye. To make these measurements, DREs use a pupillometer which consists of a three inch by five inch card on which are printed black circles with diameters ranging from 1.0 to 9.0 mm in 0.5 mm steps. This card is held along the suspect's face next to the eye and comparisons are made between the pupils and the black circles on the card. (Figure 2, 3 and 4)

Insert Figure 2 Showing Use of the Pupillometer About Here

Reaction of the pupil to a bright light is also determined with special emphasis on rate of constriction, degree of hippus, and length of time the pupil stays constricted with the light on. In the case of intoxication with marijuana, the pupil may initially constrict when the light is turned on and then open slightly, close, open more, close, etc. in what is called "rebound dilation." Other drugs cause mydriasis, miosis, or sluggish reactions to light. Using DRE protocols, abnormal pupil sizes are defined as those under 3.0 mm or over 6.5 mm for any of the measurement conditions.

Insert Figures 3 and 4 Showing Pupils of Suspects on Stimulants and Narcotics About Here

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In the dark-room, the inside of the nose is evaluated for evidence of drugs, irritation, lack of nose hairs, or other evidence of drug use. The mouth is also examined for damage caused by smoking drugs like crack cocaine that burn at high temperatures, drug residue (especially from marijuana), and a green coating on the tongue which are also consistent with smoking marijuana.

Insert Figure 5 Showing Tongue About Here

Step 8. Muscle Tone

Tone is evaluated by palpating the left bicep and forearm down to the wrist. Tone will be increased by stimulant drugs and reduced by depressants or narcotics.

Step 9 Injection Sites

Many persons use drugs by injecting them into the veins ("shooting"), under the skin ("skin popping"), or into the muscles ("muscling"). The fastest drug rush can be obtained by intravenous injection, but certain drugs are very toxic to the veins so some users prefer to skin pop or muscle them and thus experience a less intense but more prolonged high. Chronic users have numerous scars, puncture marks, or bruises on their arms, legs, neck, etc. (Figure 6) Some who have destroyed the easily accessible veins or who want to cover up their drug use will inject into the veins under the tongue, into the vascular beds under the finger nails, or into other areas not readily observed by an officer.

Insert Figure 6 Showing Track Marks About Here

Step 10. Suspect's Statements

When presented with evidence from the DRE's examination, suspects are offered an opportunity to discuss with the DRE what drugs had been taken and essentially confess to their use. Some will do this in hopes of favorable treatment from the DRE or the judicial system. Statements made during this phase of the evaluation can be used in court because the suspect was previously advised of their constitutional right not to answer any questions.

Step 11. Expert Opinion of the DRE

Based on the signs presented by the suspect and any other information that has been obtained, the DRE prepares a report containing information on the testing procedures used, the results, and an opinion regarding whether or not the suspect was under the influence of a drug at the time of testing. Further, the DRE

identifies the category of drug(s) affecting the suspect and makes a statement about the ability of the suspect to safely operate a vehicle.

<u>Step 12. Toxicological Examination</u>

In states such as Oregon, Implied Consent Laws provide that suspects must furnish a breath, blood, or urine specimen if arrested for driving while intoxicated or impaired. Failure to provide these specimens can result in a lengthy suspension of driving privileges.

If a specimen is obtained, it is sent to a toxicology lab for analysis of drug metabolites. Results of these analyses can then be used in court along with the DRE's report.

Poly-Drug Use and Drug Rebound Effects

If all suspects had taken only one class of drug, and if the DRE could be sure when the drug was taken, evaluation would be relatively simple. Unfortunately, many or most drug users take multiple drugs with unknown purities and often are not sure when they took them. Poly-drug use can produce a conflicting set of signs that must be carefully evaluated.[3] Poly-drug effects can be "null" meaning that the drugs taken do affect the sign being evaluated, "overlapping" meaning that one of the drugs affects the sign but others do not, "additive" meaning that several of the drugs produce the same effect on the sign, or "antagonistic" meaning that some of the drugs produce one effect and others produce the opposite effect. Antagonistic effects are often most problematic because if they are equal and opposite, the sign can appear normal.

An example of antagonistic effects occurs with suspects who have taken "speedballs" which are typically mixtures of heroin and cocaine. Because the effects of cocaine are quick and intense, the initial reaction of the pupil is dilation, but after an hour or so the cocaine and heroin are in balance and the pupil might appear normal. Finally, after several hours, the heroin is the predominant drug and the pupils constrict.

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A further complication for DRE evaluations arises because the body attempts to resist the artificially altered state caused by drug use and tries return to a condition of homeostasis. Typically, this involves activation of the sympathetic or parasympathetic components of the autonomic nervous system depending on what type of drug was taken. Because the autonomic activity can last longer than the drug effects, it is common for rebound effects to occur that are opposite to the effects produced by the drug itself.

A good example is the depression and "down" effects that occur after cocaine or methamphetamine wears off. To return to euphoria, the suspect needs to take more of the drug, but if none is available (e.g., the person is in police custody), several of the signs that DREs evaluate can be consistent with the use of a CNS depressant or a narcotic analgesic. To avoid being confused by drug rebound effects, DREs consider the half-life of the drug in the body (short for drugs like cocaine), how long the person has been in custody (i.e., how long since the person could have last taken a drug), trends in the three pulse rate measures which could indicate changing drug levels in the body, and other information about what drug was taken.

Validity of DRE Evaluations

DRE opinions are not yet accepted as evidence in all states, partially because case law (i.e., cases in which judges have set precedent by allowing DRE opinions to be used as expert testimony) has not been established. When DRE opinions have been introduced as evidence, defense attorneys have raised questions concerning the validity of DRE testing procedures and conclusions.

DRE program validity can be demonstrated in several ways. First, the majority of signs that DREs evaluate are also evaluated in medical examinations of drug overdose patients. Miosis produced by narcotics and mydriasis produced by stimulants, along with the effects of drugs on heart rate, blood pressure, and body temperature are well known and documented in the medical literature. In the

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absence of confounding conditions, the presence of these signs can be valid indicators of drug use.

Second, there are laboratory and field studies that demonstrate the validity of DRE evaluations. In these studies, the DRE's determination of what drugs the suspect had taken were compared to the "gold standard" laboratory blood or urine test findings. The degree of agreement between the DRE's determinations and the laboratory test results indicates the degree of DRE test validity.

LAPD Field Study

Results of a field study on the validity of DRE testing were published in a 1986 National Highway Transportation Safety Administration (NHTSA) report.[14] In this study, subjects had been arrested for DUII and were evaluated by selected senior DREs from the Los Angeles Police Department. If the DRE determined that the subject was under the influence of a drug other than alcohol, a classification of not intoxicated, opiate, sedative, stimulant, or marijuana was made. A blood sample was then requested; 173 suspects provided samples. If the DRE determined that no drug was active in the subject's system (i.e., the suspect was not intoxicated), no blood was drawn.

Laboratory tests indicated that 47 of the suspects had taken a single drug and 125 had taken multiple drugs. PCP was the most commonly detected drug, followed by alcohol and marijuana.

When the DREs' determinations were compared to laboratory test data, the determinations were entirely correct for 49% of the suspects and partially correct for an additional 38%. Totally correct means that the DRE correctly named all of the drugs the suspect had taken. Partially correct means that the DRE correctly named only some of the drugs.

The DRE evaluations were entirely incorrect for 13% of the suspects; most of these errors occurred for suspects who had taken

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multiple drugs. Accuracy rates ranged from 92% for correct detection of PCP to 33% for cocaine.

In perspective, this early study demonstrated that a reasonable degree of test validity (87% correct detection of at least one drug) could be obtained using the DRE procedures with suspects already under arrest for DUII. However, the study provides no information on false negative rates (persons who were actually intoxicated but who were determined to be not intoxicated) since no blood was drawn from these suspects.

Johns Hopkins Study

A laboratory validation study of DRE procedures was conducted by Bigelow, et al. in 1985 at the Johns Hopkins University School of Medicine.[15] In this study, four DREs evaluated subjects who had been administered one of the following: marijuana (12 puffs of 1.3% or 2.8% THC cigarettes), Valium (15 or 30 mg), secobarbital (300 mg), or d-Amphetamine (15 or 30 mg). Placebos were also given. Drugs were administered to 80 subjects in separate sessions using a balanced Latin square design so that each subject received each drug. The study was conducted using double masked conditions.

Prior to testing, the DREs were informed that no drug combinations, alcohol, PCP, or LSD had been given, but were not told about what other drugs might have been used. Their task was to place the subject into one of 5 categories: not intoxicated, opiate (none was actually given), sedative (Valium or secobarbital), stimulant (d-Amphetamine), or marijuana.

The DREs rated the placebo cases as not intoxicated with 95% accuracy, but they also rated 45% of cases in which drugs had actually been given as not intoxicated. The percentage of false negatives varied from 82.5% for subjects who had taken 15 mg of d-Amphetamine to 5% for those who had taken 300 mg of secobarbital. These results suggest either that the officers set their criteria levels for determining that a drug had been taken excessively high,

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or that some of the drug levels used in the study were too low to affect the subjects in a detectable manner.

When the DREs determined that a drug had been given, they divided their responses between the stimulant, depressant, and marijuana categories. (The opiate classification was selected only one time out of 320 trials; no opiates were administered in this study.) The classifications were correct for 92% of the cases.

Results of this study indicate that when officers feel confident enough to classify a subject as intoxicated, and when only a single drug selected from a limited population of drugs has been given, the ability of the DRE to make an accurate classification is quite good. The DRE conclusions were reasonably valid under these conditions.

Arizona Study

A more recent study was conducted using State of Arizona records covering a 53 month period from 1989-93.[16] Suspects were arrested for DUII (presumably based on failure of the standard field sobriety tests), had an evaluation done by a certified DRE, and provided a biological specimen (urine). Sixty-eight subjects had no drug detectable in the urine, 153 had evidence of only one drug, and 253 had evidence of multiple drug use found in the urine.

For the 68 subjects with no drugs detected in the urine, DREs concluded that 62% of them were under the influence of a drug and that 38% were not. This high false positive rate might be accounted for by the fact that the laboratory was unable to detect or classify certain "designer" or unusual drugs, that the subjects had other problems which simulated the effects of drug use, or that the DREs simply made errors.

The DREs concluded that 190 of the suspects had taken only one type of drug. Of these suspects, the drug classifications were correct (based on DRE criteria) 76% of the time. The DREs were correct in determining the specific classification 72% of the time, and in 3% of the cases the DRE specified the drug class correctly but

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erroneously believed that another drug was also present. For 24% of the suspects, the DRE incorrectly classified the drug present or concluded that a drug was present that could not be demonstrated in the urine.

There were 268 suspects for whom the DREs found evidence of multiple drug use. At least one of the drugs was classified correctly for 87% of these suspects. However, the DREs were correct in specifying all of the multiple drugs in the systems for only 18% of the suspects. This is not surprising because of the complex drug interactions that can occur.

Overall, the results of this study indicate that the DREs made a correct classification of at least one drug the suspect had taken or made a correct determination that no drug was present in about 85% of the cases.

Summary of Validation Studies

In evaluating the results of these studies, it is important to remember that parts of the 12 step evaluation process involve examination of any drugs or paraphernalia in possession of the subject. In addition, DREs attempt to solicit confessions from suspects and use this information in making their determinations. Although confessions can often be misleading, they are more commonly accurate for depressant or narcotic users who are often familiar with the criminal justice system. These confessions can increase the accuracy of DRE evaluations. For example, in the Arizona field study, 93% of the suspects who tested positive for narcotics admitted using these drugs, 85% of depressant users admitted use, and 59% of marijuana users confessed. It should not be assumed, however, that just because a confession is obtained, the DRE would not have reached the same conclusion based only on the suspect's signs.

The degree to which suspect admissions, mannerisms, and communications are used by the DRE to determine which drugs might have been taken is indicated by a study in which DREs examined

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subjects dosed with various levels of alcohol, cocaine, or marijuana.[17] The DREs were not allowed to question subjects about recent drug use or attempt to solicit admissions of drug use. Other aspects of the 12 step process were also omitted. In this situation, the DRE's predictions of drug type were accurate only 44% of the time. Although other factors might account for this seemingly low percentage, it could also indicate that the higher accuracy levels reported in prior studies resulted in part from confessions, observations of behavior, conversations with the suspects, or information that was lost because the entire 12 step process was not used.

Summary

Except for simple curiosity about drug abuse, why should the DRE program be of interest to optometrists? The reasons are multiple. First, driving while under the influence of drugs poses a threat to everyone. If a set of valid procedures can be found to aid in convicting persons driving under the influence, all of society should be interested.

On a more specific level, many or most optometrists have probably examined patients under the influence of drugs without knowing it. Abused drugs can affect many aspects of the test findings and intoxication can lead to incorrect diagnoses being made for the patient. Based on a knowledge of DRE test procedures, an optometrist should be able to conduct a few simple tests and detect drug intoxication in many cases. The optometrist can then act accordingly by re-interpreting vision test findings, counseling with the patient, or making a police report if driving or other dangerous activities are involved.

Optometrists also have an opportunity to participate directly in their state's drug evaluation program. Many of the DRE procedures evaluate ocular signs and this is a field of obvious optometric expertise. Participation in DRE training programs can sometimes be arranged for optometrists, and ride-alongs with DRE officers can

provide an opportunity to observe DRE procedures first hand. For those who want to become even more involved, teaching and expert witness opportunities exist. This is a program that benefits society and it warrants optometric support.

Footnotes

a. Pacific University College of Optometry, Forest Grove OR, 97116

b. Oregon State Police, Albany OR. Oregon State DECP Coordinator and Certified DRE Instructor

c. Oregon State Police, Portland OR. Certified DRE Instructor

d. The term "drug" can be used in several contexts. From a medical perspective, a drug is typically thought of as a substance used to diagnose, prevent, or treat disease. Substances as airplane glue or paint would not be considered drugs using this definition. However, from a traffic law enforcement perspective, a drug is often defined as any material which impairs the ability to operate a motor vehicle. Many in the medical community would prefer that the term "substance" be used instead of "drug" in this context, especially since the abused materials include glue, paint, insecticides, etc. This would help to make clear the obvious differences between the beneficial effects of "drugs" and the deleterious abuse of "substances." However, because of the orientation of this paper, the law enforcement definition of "drug" will be used.

e. In some states the program is referred to as the Drug Recognition Expert Program and officers are referred to as Drug Recognition Experts. In other states, case law does not allow the word "expert" to be used and the program is referred to as the Drug Evaluation Classification Program.

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Table 1. General information on abused drugs from DRE course manual.[3]

	Depressants	Stimulants	Hallucinogens	Phencyclidine	Narcotic Analgesics	Inhalants	Cannabis
General Indicators of Drug Use	Uncoordinated; disoriented; sluggish; thick, slurred speech; drunk-like behavior; gait ataxia; drowsiness, droopy eye lids; fumbling. (Methaqualone increases the pulse and produces body tremors)	Restlessness; Body tremors; excitement; euphoria; talkative; exaggerated reflexes; anxiety: bruxism; redness in nasal area and runny nose if drug snorted; loss of appetite; insomnia; increased alertness: dry mouth; irritability	Dazed appearance; body tremors; synesthesia; hallucinations; paranoia; lack of coordination; nausea; disorientation; speech problems; perspiration; poor time and distance perception; disorientation; flashbacks. (LSD can produce piloerection)	Perspiration, warm to the touch; blank stare; early onset of HGN; speech problems; incomplete verbal responses; repetitive speech; increased pain threshold; cyclic behavior; confusion; agitation; hallucinations; possible violence and combativeness; chemical odor; unusual gait	Droopy eyelids; nodding of the head; drowsiness; depressed reflexes; low, raspy, slow speech; dry mouth; facial itching; euphoria; fresh and old injection sites; nausea. (Tolerant users can exhibit very little psychomotor impairment.)	Residue of substance (e.g., paint) around nose and mouth; odor of substance; possible nausea; slurred speech; disorientation; confusion; blood shot, watery eyes; lack of muscle control; flushed fact; non-communicative; intense headache	Marked injection of conjunctiva; odor of marijuana; marijuana debris in mouth; body tremors; eyelid tremors; relaxed inhibitions; increased appetite; impaired perception of time and distance; disorientation; possible paranoia
Duration of Effects (Times vary depending on specific drug taken and method of administration)	Barbiturates: 1 to 16 hours Tranquilizers: 4 to 8 hours Methaqualone: 4 to 8 hours	Cocaine: 5 to 90 minutes Amphetamine: 4 to 8 hours Meth-amphetamine: 12 hours	Duration varies widely	Onset of effects in 1 to 5 minutes; peak effects in 15 to 30 minutes; effects evident for 4 to 6 hours	Heroin: 4 to 6 hours Methadone: up to 24 hours Others: variable durations	Volatile solvents: 6 to 8 hours Anesthetic gases and aerosols: very short durations	Effects are evident for 2 to 3 hours Impairment can last up to 24 hours without awareness of effects
Method of Administration	Typically oral, occasionally injected	Insufflation (snorting), smoking, injection, oral	Oral, insufflation, smoking, injection, transdermal	Smoking, oral, insufflation, injection, eye drops	Injection, oral smoking, insufflation	Insufflation	Smoking, oral

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| Signs of Overdose | Shallow breathing; cold, clammy skin; dilated pupils; rapid, weak pulse, coma | Agitation, increased body temperature; hallucinations, convulsions | Long, intense trip | Long, intense trip | Slow, shallow breathing; clammy skin; coma; convulsions | Coma | Fatigue; paranoia |

Table 2. Signs typically produced by drugs in categories shown. Reproduced with minor modifications from DRE course manual.[3]

	Depressants	Stimulants	Hallucinogens	Phencyclidine	Narcotic Analgesics	Inhalants	Cannabis
HGN	Present	None	None	Present	None	Present	None
Vertical Nystagmus	Present with high doses for the individual	None	None	Present	None	Present with high doses for the individual	None
Lack of Convergence	Present	None	None	Present	None	Present	Present
Pupil Size	Normal except for Soma and Quaaludes which usually dilate the pupils	Dilated	Dilated	Normal	Constricted	Normal but can be dilated in some individuals	Dilated but can be normal in some individuals
Pupil Reaction to Light	Slow	Slow	Normal except for certain psychedelic amphetamines which cause slow reactions	Normal	Little or no reaction	Slow	Normal
Pulse Rate	Down except might be increased by Quaaludes or alcohol	Up	Up	Up	Down	Up	Up
Blood Pressure	Down	Up	Up	Up	Down	Down with anesthetic gases and up with volatile solvents	Up
Body Temperature	Normal	Up	Up	Up	Down	Up, Down, or Normal	Normal

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Table 3 Commonly abused drugs with street names and approximate prices in Portland OR, Summer 1997.

Drug Class	Drug Name	Generic Name	Street Name	Typical Cost per Street Dose	Form of Drug
Depressant	Soma	carisoprodol	Soma	$5 per tablet	Tablets
Depressant	Xanax	alprazolam	Xanax	$5 per tablet	Tablets
Depressant	Valium	diazepam	Valium	$5 per tablet	Tablets, injectable
Depressant	Flexuril	cyclobenza-prine HCl	Flexuril	$5 per tablet	Tablets
Depressant	Prozac	fluoxetine-hydrochloride	Prozac	$5 per tablet	Tablets
Depressant	Alcohol	ethyl alcohol	Booze, sauce, juice, brew	Varies	Liquid
Stimulant	Meth-amphetamine	meth-amphetamine	Crank, speed, crystal, crystal meth, ice	Dependant upon purity; relatively inexpensive	Moist paste-like material; colors range from clear to yellow-brown; also translucent crystals
Stimulant	Cocaine	cocaine	Snow, blow, crack, rock, coke	More expensive; dependant upon purity; $50 + per gram	White powder; rock-type material
Stimulant	Ritalin	methyl-phenidate hydrochloride	Ritalin	$5 per tablet	Tablet
Stimulant	Desoxn	meth-amphetamine hydrochloride	Desoxn	$5 per tablet	Tablet

Hallucinogen	Peyote	psilocybin	Peyote, buttons	Unknown	Cactus buttons
Hallucinogen	Mushrooms	mescaline	Mushies, caps, cubinzas, shrooms	$30+ per gram	Dried mushrooms, tea
Hallucinogen	Nutmeg		Nutmeg	Unknown	Brown powder; solid lump
Hallucinogen	Jimson weed		Jimson weed, datura	Unknown	Dried parts of plant
Hallucinogen	Morning glory seeds		Morning glory seeds	Unknown	Seeds, tea
Hallucinogen	Bufotenine		Bufotenine	Unknown	Toad skin
Hallucinogen	LSD	lysergic acid diethylamide	Doses, tabs, paper, fries	$5 per dose	Paper squares, drops
Psychedelic amphetamine (Halluc- inogen)	Ecstasy	methylene dioxymeth- amphetamine	Ecstasy, X, MDMA	$20+ per dose	Tablets
Psychedelic amphatamine (Halluc- inogen)	STP	dimethoxyl- amphetamine	STP, DOM	Unknown	Unknown
Dissociative anaethetic	PCP	phencyclidine	PCP, angel dust	Unknown	Power, or liquid
Dissociative anesthetic	Ketamine		Special K	Unknown	Power, or liquid
Narcotic Analgesic	Vicodin	hydrocodone Bitartrate	Vicodin	$5 per tablet	Tablet
Narcotic Analgesic	Percoset	oxycodone and acetominophen	Percoset	$5 per tablet	Tablet
Narcotic Analgesic	Percobarb		Percobarb	$5 per tablet	Tablet

Narcotic Analgesic	Hycodon		Hycodon	$5 per tablet	Tablet
Narcotic Analgesic	Methadone	methadone hydrochloride	Methadone	Unknown	Pink liquid
Narcotic Analgesic	Heroin		Smack, China white, Mexican tar Speedball (heroin and cocaine) Spaceball (heroin and meth)	$10-15 per hit	Various forms including white powder, and small blobs that look like tar
Narcotic Analgesic	Fentanyl	fentanyl and meperidine	Tango and Cash, synthetic heroin	Unknown	
Cannabis	Marijuana	delta-9 tetra-hydro-cannabinol	Mary jane, Ganja, Spleef, Joint, Weed, Grass, Bud	$10+ per gram	Dried leaves, stems buds, flowers
Cannabis	Marinol	delta-9 tetra-hydro-cannabinol	Marinol	Unknown	Tablet

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Figure Legends

1. Administration of the HGN test.
2. Use of the DRE Pupillometer to measure the diameter of a suspect's pupils.
3. Dilated pupils similar to those that would be seen in a suspect intoxicated with a stimulant such as methamphetamine.
4. Constricted pupils similar to those that would be seen in a suspect intoxicated with a narcotic such as heroin.

Figure 5. Green coating on the rear portion of the tongue similar to that which might be seen with some suspects who have recently smoked marijuana.

6. Needle tracks on the arm of a long-term heroin user.

Figure 1.   Administration of the HGN test.

Figure 2. Use of the DRE Pupillometer to measure the diameter of a suspect's pupils.
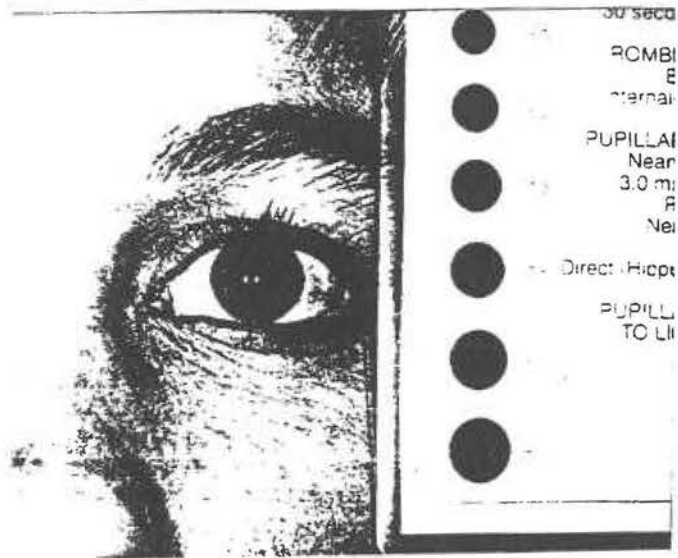
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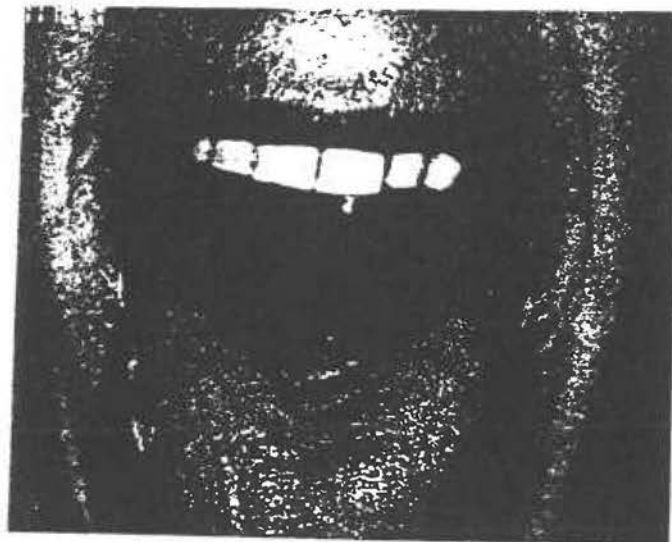
Figure 6. Needle tracks on the arm of a heroin user.