Phencyclidine Blood Concentrations in DRE Cases*

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Abstract

Phencyclidine (PCP) concentration was measured in blood obtained from 259 individuals over a two-year period subsequent to Drug Recognition Expert (DRE) evaluation by the Maryland State Police. The purpose of this study was to evaluate the accuracy of the DRE in the identification of PCP-related impairment using the presence of PCP in blood to confirm drug use and to test for a correlation between PCP concentrations in blood and impairment as indicated by DRE evaluation. Of the 259 cases evaluated, 124 were identified as positive for PCP based on DRE evaluation, 130 were positive for PCP based on toxicological analysis, and 56 of the 124 were identified as positive for PCP only by DRE and subsequently confirmed to contain only PCP. The mean PCP concentration for those cases in which only PCP was identified by both DRE and toxicology was 51 ng/mL (standard deviation, 26 ng/mL) with a range of values of 12–118 ng/mL. Although no correlation was determined between PCP concentration and behavior, it is clear that, even at concentrations as low as 12 ng/mL, PCP-induced behavioral effects are measurable by DRE evaluation. This study also revealed that despite a low false-positive rate (3%) of detection of PCP use by the DRE, the false-negative rate of 8% supports the conclusion that the toxicological analysis of blood specimens for PCP provides the necessary, objective corroboration of the DRE's opinion concerning impairment.

Introduction

Phencyclidine (PCP) remains a significant drug of abuse in today's society. However, unlike marijuana and cocaine, the abuse of PCP appears to be localized in certain regions of the country such as New York City, Los Angeles, Detroit, Chicago, and the Baltimore-Washington, D.C. corridor (1). The most common route of administration of PCP is smoking, usually on a tobacco, marijuana, or parsley cigarette, although it is less frequently administered by mouth, insufflation, and intravenous injection. PCP, first synthesized in 1926, was released in England in 1960 as a dissociative anesthetic. Because of profound adverse effects upon emerging from the anesthetic (e.g., hallucinations, delirium ["emergence delirium"], disorientation, extreme agitation, muscle rigidity, and seizures), human use was discontinued. Veterinary use of the drug continued until 1979 when PCP was added to the list of Schedule II controlled drugs (2–4). PCP is currently one of five abused drug groups tested in the Health and Human Services Workplace Drug Testing Program and is the only drug for which there is no possible medical explanation for a positive result.

In addition to its anesthetic properties and hallucinogenic effects, PCP also acts as an analgesic, a central nervous system (CNS) depressant, and a stimulant and is generally classified as a dissociative anesthetic (i.e., produces analgesia and amnesia without respiratory depression resulting in a state in which the patient appears dissociated from his environment but not necessarily asleep [2]). After high doses or chronic use, an acute psychosis that resembles schizophrenia may develop (2,4–6). The psychosis may be persistent, but it is unclear if that is a result of physiological and neurological changes secondary to PCP use, the persistent presence of PCP in the body after chronic use, or simply an unmasking of underlying pathology present before PCP use (3). This constellation of effects makes it difficult to predict or anticipate an individual user's response to PCP. Another difficulty associated with PCP use is the apparent lack of a direct correlation between blood concentrations and behavioral effects as is seen with ethanol (7–9); this lack of correlation may be a result of slow release of PCP from high lipid-containing and adipose tissues and through a gastric recirculation process (ion trapping of PCP in the stomach with subsequent reabsorption in the small intestine) (5,9).

Throughout this century, numerous studies enabling the forensic toxicologist to correlate blood ethanol concentration with behavior have been performed. At a given blood ethanol concentration, certain behaviors or a range of behaviors can be expected without direct observation of an individual providing supporting evidence of impairment. These studies have

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permitted legislative bodies and regulatory agencies to enact laws and regulations prohibiting individuals from performing certain activities at or above a specified blood ethanol concentration (e.g., DUI/DWI laws). Unfortunately, an analogous situation does not exist with other drugs of abuse. The need to recognize the role of drugs of abuse in the impaired driver led to the formation of the Drug Evaluation and Classification (DEC) program. This program is based on a cooperative effort between three disciplines: law enforcement, in the person of the Drug Recognition Expert (DRE) who documents impairment; toxicology, in the person of the toxicologist who provides analytical support and expert opinion concerning the effects of drugs on human performance; and prosecution. The initial step in this program involves the evaluation of a series of physiological and psychomotor tests and observations performed by trained individuals referred to as DREs. The evaluation performed by the DRE forms the basis of an opinion concerning impairment resulting from the use of one or more drugs from seven drug groups (CNS depressants, CNS stimulants, hallucinogens, phencyclidine, narcotic analgesics, inhalants, and cannabis). The DEC system used is a standardized, systematic method of examining a person suspected of impaired driving to determine if the suspect is impaired, if the impairment is due to drug use or is medically related, and the broad category of drugs suspected of causing the impairment (10). The opinion concerning the presence of drugs should be corroborated and, as a consequence, blood or urine or both specimens are collected for toxicological analysis. The toxicological analysis provides the scientific and objective support for the subjective report of drug-associated impairment.

The purpose of this study was to evaluate the accuracy of the DRE in the identification of PCP-related impairment using the presence of PCP in blood as confirmation of drug use and to test for a correlation between PCP concentrations in blood and impairment as indicated by DRE evaluation. PCP was chosen for this DRE/toxicology comparison because PCP is easily quantitated using gas chromatography (GC) with nitrogen phosphorus detection (NPD) or mass spectrometry (MS) without requiring derivatization or complicated extraction techniques, is stable in blood (11), and its half-life in the blood is greater than 12 h (12), which suggests that collection of a blood specimen within several hours of observed impairment will not substantially affect the quantitative value. Moreover, a review of data obtained in Maryland over a 2-year period revealed a large number of cases in which only PCP was identified by the DRE. Although all blood samples submitted subsequent to DRE evaluation were screened for common drugs of abuse, PCP concentrations in blood are reported only for those cases meeting the following criteria: PCP was the only drug identified by the DRE and PCP was the only drug subsequently confirmed by toxicological analysis.

Experimental

Case selection

Traffic stops were made by Maryland State Police officers based on observed driving behavior. Field sobriety tests and other observations were made by the arresting officer to establish probable cause that the driver was intoxicated or under the influence of alcohol or drugs or both. An evidential breath test was administered by an approved breath test operator. If the results of the tests were inconsistent with the degree of impaired behavior observed and measured by the arresting officer or evidence suggestive of drug use was found, a drug recognition evaluation was requested. This examination was performed by a certified DRE according to the protocol established by the National Highway Traffic Safety Administration. The DRE evaluation consists of a breath-alcohol test; an interview of the arresting officer; a preliminary examination of the suspect, examination of the eyes for horizontal and vertical nystagmus and lack of convergence, divided attention tasks (e.g., walk and turn, one leg stand, Romberg balance test, and finger to nose), vital sign examination, examination of pupil size under various lighting conditions (near darkness, indirect light, and direct light), examination of muscle tone, examination for injection sites; and interview of suspect concerning drug use based on DRE opinion of which drug category or categories may be present (10). The examination concludes with the formation of an opinion concerning drug and/or alcohol use by the suspect and collection of specimens for toxicological analysis to substantiate the DRE's opinion; the blood was forwarded to American Medical Laboratories (Chantilly, VA) for analysis.

Drug testing

All blood specimens were screened for common drugs of abuse (amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine metabolite, opiates, and PCP) by radioimmunoassay (Diagnostic Products, DPC, Los Angeles). All presumptive positive results were confirmed and quantitated by GC–MS.

PCP analysis

Screening. Blood specimens were screened for the presence of PCP using the DPC Coat-A-Count radioimmunoassay. The assay was performed according to manufacturer specifications using a cutoff of 10 ng/mL of PCP.

Confirmation and quantitation. The presence of PCP in blood samples screening positive was confirmed and quantitated using GC–MS operated in the selected ion monitoring mode. Five ions were monitored: *m/z* 186, 200, and 242 for PCP and *m/z* 205 and 247 for PCP-d_5 (internal standard). An Hewlett-Packard (Palo Alto, CA) 5890 GC coupled to a 5970 mass selective detector was used. The injector temperature was set at 200°C and the oven was operated with the following temperature program: 100°C (1 min) ramping to 280°C at 20°C/min. A 12.5-m cross-linked methyl silicone fused-capillary column was used with helium as the carrier gas flowing at 1 mL/min. Samples were considered positive if quantitative values were greater than 5 ng/mL.

Extraction. PCP-d_5 (0.2 µg) and bicarbonate buffer (1 mL, pH 10.5) were added to 2 mL standard, control, or blood specimens. After vortex mixing, the contents of each tube was poured onto a Chem-Elut solid-phase extraction tube (Varian Products, Harbor City, CA). The column was washed with three

6-mL portions of hexane/isoamyl alcohol (99:1). These washings were extracted with 1 mL of 0.1N H_2SO_4. The acid layer was alkalinized with 500 µL of concentrated NH_4OH and extracted with 100 µL of chloroform. One microliter of the chloroform layer was injected onto the GC–MS.

Results and Discussion

A total of 259 cases were included in this study reflecting the total number of DRE cases submitted by the Maryland State Police to American Medical Laboratories between 1993 and 1995. Of the 259 specimens, 124 were identified as positive for PCP, either alone or in combination with other drugs, based on the DRE evaluation. Of the 259 specimens, 130 were positive for PCP, either alone or in combination with other drugs, based on toxicological analysis of the submitted blood specimen. These results are summarized in Table I.

Table I. Distribution of Reviewed Cases by DRE Evaluation and Blood Toxicology

		Blood toxicology		Total DRE
		Positive	Negative	
DRE evaluation	positive	120	4	124
	negative	10	125	
	Total toxicology	130		259

Table II. Summary of PCP Blood Concentrations for DRE/Toxicology Positive PCP Cases* (n = 55)

	Mean	Standard deviation	Median	Range
PCP blood concentration (ng/mL)	51	26	44	12–118

* n = 55 blood quantitations;
n = 56 cases positive only for PCP by both DRE and toxicological evaluation.

Within the cohort of cases reported by the DRE as containing PCP either alone or in combination with other drugs (124 cases), 56 were reported as containing only PCP by the DRE and confirmed to contain only PCP based upon toxicological analysis. As compared with toxicology results, the DRE had a false-negative rate of approximately 8%; 10 of 130 suspects were not identified as using PCP based on DRE evaluation but were found to be positive for PCP. The DREs had a false-positive rate of approximately 3%; 4 of 129 suspects were identified as using PCP based on DRE evaluation, but PCP was not found in the blood by toxicological analysis. In other words, approximately 8% of PCP-positive suspects were missed by the DRE, and approximately 3% of the suspects identified as having used PCP were not corroborated by toxicology findings. The efficiency of the DRE evaluation to detect PCP use was 95% (245/259).

A quantitation was performed on 55 of the 56 cases in which only PCP was present, and a summary of these results is reported in Table II. The mean blood-PCP concentration was 51 ng/mL with a standard deviation of 26 ng/mL. The median PCP concentration was 44 ng/mL with a range of values from 12 to 118 ng/mL. The distribution of PCP concentrations is detailed in Figure 1. The range of concentrations detected in these drivers is well within the ranges reported in other DUI cases and medical examiner cases in which PCP intoxication was not the primary cause of death. These concentrations are typically less than 100 ng/mL with behavioral effects noted as low as 10 ng/mL (7–9,12,13). Blood concentrations greater than 300 ng/mL have been associated with fatal PCP intoxications (12,13).

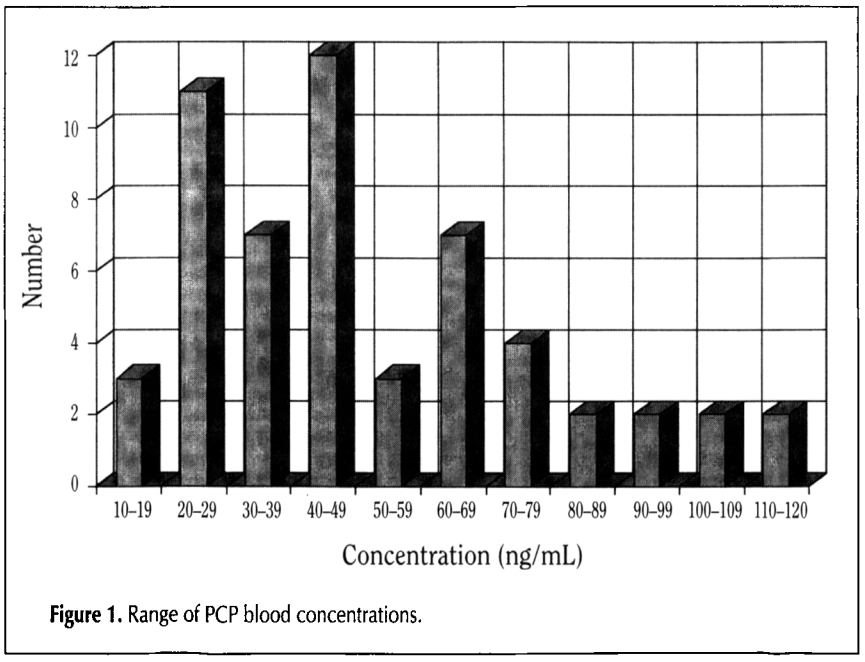

Figure 1. Range of PCP blood concentrations.

DRE clues for the presence of PCP

The DRE is trained to observe behavior and collect physiologic data in an effort to determine if observed impairment is secondary to drug effects. As a consequence of numerous evaluations, the DRE begins to look for a combination of behaviors and physiological indicators that may suggest that an individual is under the influence of a particular drug or one of the members of a particular class of drugs. In the case of PCP, the DRE may report that an individual is under the influence of that drug if most or all of the following clues are observed: horizontal and vertical nystagmus; lack of convergence; elevated pulse rate, blood pressure, and core body temperature; fixed, blank stare; repetitive and/or incoherent speech that may be slurred; general confusion; agitation; and delusions (4,9,10).

Discrepant results between DRE and toxicology

Fourteen of the 259 cases surveyed showed a discrepancy between the DRE

Table III. Discrepant Results Between DRE and Toxicology (n = 14)

Specimen ID #	DRE*	Toxicology†
93-16	cannabinoids	PCP‡, cocaine
92-4	PCP	benzoylecgonine
94-42	PCP, cannabinoids	no drugs detected
94-59	narcotic analgesics	PCP, methadone
94-79	cannabinoids	PCP
94-80	narcotic analgesics, CNS stimulants	PCP, benzoylecgonine
94-108	cannabinoids, CNS depressants	PCP, cocaine, benzoylecgonine
94-114	cannabinoids	PCP
94-115	PCP	carbamazepine
95-1	CNS depressants, narcotic analgesics	PCP, methadone, benzoylecgonine, clonazepam
95-2	cannabinoids, CNS depressants	PCP
95-3	hallucinogens	PCP
95-12	PCP, cannabinoids, CNS depressants	THCCOOH, secobarbital
95-15	cannabinoids, CNS depressants	PCP

* Drug class identified by DRE.
† Drugs identified by toxicological analysis.
‡ Abbreviations: PCP, phencyclidine; CNS, central nervous system; THCCOOH, 11-nor-9-carboxy-Δ^9-tetrahydrocannabinol.

opinion concerning what drug class was responsible for the noted impairment of the subject and the subsequent toxicological results from that subject's blood sample. Of these cases, 10 were found to be positive for PCP although the presence of PCP was not noted by the DRE and 4 were found to be negative for PCP although the DRE suspected the use of PCP in his evaluation of the subject. These discrepant results are detailed in Table III. There is no apparent pattern between the DRE opinion of which drug class or classes are present and the blood toxicology results (e.g., such a pattern might be the opinion that hallucinogens are present when, in reality, PCP is present or the opinion that CNS stimulants are present when, in reality, only PCP is present). In some cases, CNS depressants was indicated when, in fact, only PCP was present, and in other cases, only PCP was indicated when, in fact, no hallucinogen, stimulant, or PCP was found. Blood-PCP concentrations ranged from 12 to 64 ng/mL in those cases in which the DRE did not identify PCP but PCP was detected by toxicological analysis. This suggests that the presence of PCP was not missed by the DRE simply because the PCP concentration was very low, but it reinforces the fact that the behavioral effects of PCP are not related to blood concentrations.

Conclusion

The results of this study suggest that, in the majority of cases in which impairment was observed and recorded by the DRE consistent with the expected effects of PCP, the presence of PCP was confirmed by toxicological analysis. It is important to note, however, that there are certain conclusions that cannot be drawn from this study. The most important point is that this study does not establish a correlation between PCP blood concentration and impairment; it is questionable whether such a correlation can be established. It is also important to note that this study does not suggest that there is no psychomotor impairment associated with blood PCP concentrations below 12 ng/mL (the lowest concentration noted in this cohort of subjects), but it simply shows that a PCP concentration of 12 ng/mL was associated with observable impairment as measured by a DRE evaluation. It should also be noted that observed impairment as noted by a DRE in combination with a confirmed blood level of PCP provides a reasonable scientific certainty that the impairment is related to the use and presence of PCP. The same type of impairment noted by a DRE and a confirmed urine level of PCP provides only a reasonable probability that the observed impairment is drug related because PCP can remain in the urine for a number of days after use.

In summary, the results of this study suggest that the information obtained about a suspect's ability to safely operate a motor vehicle from a DRE evaluation cannot substitute for the toxicological analysis of the suspect's blood and/or urine for the presence of impairing substances. However, the confirmation alone of the presence of such drugs in blood and/or urine may not be sufficient to establish impairment to the degree that the suspect is incapable of safely operating a motor vehicle. The cooperative effort of the DRE and the toxicology laboratory is essential for the correct evaluation of impairment secondary to drug use. In the particular case of impairment resulting from PCP use, the collection of a blood sample subsequent to the DRE's opinion that the suspect has used PCP provides the best mechanism for corroborating the use and presence of that drug.

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