JOURNAL OF FORENSIC SCIENCES

J Forensic Sci, May 2011, Vol. 56, No. 3
doi: 10.1111/j.1556-4029.2010.01687.x
Available online at: onlinelibrary.wiley.com

PAPER

PATHOLOGY/BIOLOGY

Stephen J. deRoux,[1] M.D.; Anthony Sgarlato,[2] M.D.; and Elizabeth Marker,[3] Ph.D.

Phencyclidine: A 5-Year Retrospective Review from the New York City Medical Examiner's Office

ABSTRACT: We report here a 5-year retrospective review of autopsy cases from the New York City Medical Examiner's Office that demonstrated phencyclidine (PCP) in the blood. There were a total of 138 cases. There were 52 deaths because of mixed drug intoxication: the blood PCP concentrations in these cases ranged from <1 to 598 ng/mL. There were 80 violent deaths in which PCP was quantified in the blood but was unrelated to the cause of death. There were five nonviolent deaths in which PCP exclusively was detected. In four of these, there were preexisting medical conditions that could also have contributed to death. In these, the highest PCP concentration was 361.3 ng/mL, a concentration lower than seven of the individuals in our violent death category. This suggests that lower concentrations may be fatal with comorbid conditions.

KEYWORDS: forensic science, phencyclidine (PCP), drug intoxication, drug fatalities, drug overdose

Phencyclidine (PCP) 1-(1-phencyclohexyl) piperidine was developed as an anesthetic and analgesic agent. It is structurally related to ketamine and belongs to a class of drugs known as arylcyclohexamines. The hydrochloride salt is readily soluble in water and ethanol. Although it was effective as an anesthetic in humans, it was discarded for this purpose due in part to unacceptable side effects, including thought disturbances. Because of its relatively easy synthesis by amateur chemists in clandestine laboratories and its inexpensive production, PCP became an illicit substance of abuse. It is known by many synonyms; arguably, the best known is "angel dust." It is self-administered by smoking, insufflation, oral ingestion, or intravenously. The most popular method is by smoking, often being mixed with smokable leaves such as tobacco, marijuana, mint, and parsley. The typical high from PCP lasts 4–6 h (1).

We have conducted a 5-year retrospective review of all autopsy cases of the New York City Medical Examiner's Office that demonstrated PCP in the blood. Our primary objective was to determine whether the drug caused or contributed to death in these individuals and whether it was the proximate cause of death, i.e., is there a definitive lethal concentration of PCP in blood?

Materials and Methods

We conducted a computerized database search of the records of the Forensic Toxicology Laboratory of the New York City Office of the Chief Medical Examiner to find all cases from January 1, 2003, to December 31, 2007, in which PCP was detected in blood retrieved at autopsy. All of these case files were reviewed.

Two types of extraction techniques were used for the identification and quantitation of PCP in biological specimens. Chemicals and reagents used for these extractions were ACS grade.

Qualitative Liquid/Liquid Extraction for the Identification of PCP in Blood and Brain by Gas Chromatography

For blood analysis, a 5 mL aliquot of validated negative blood, externally purchased blood control (0.2 mg/L) or unknown sample was quantitatively pipetted into a borosilcate glass extraction tube. One aliquot of certified negative blood was fortified with a preprepared methanolic calibrator (including PCP) to a final concentration of 1 mg/L. For brain analysis, validated negative blood and unknown samples were homogenized (one part tissue, two parts deionized water) and a 5 mL aliquot was quantitatively pipetted into an extraction tube. Controls were prepared similarly to blood in the brain homogenate.

Internal standard (50 μL of 50 mg/L methapyriline) was added to all tubes followed by vortex mixing. To each was added 2 mL of pH 9.8 sodium carbonate buffer followed by mixing. After addition of 10 mL n-butylchloride, the vials were mixed at low speed on a mechanical shaker for 10 min and centrifuged for 10 min at approximately $1650 \times g$. The organic layer was transferred to a clean tube and 5 mL of 0.5 N HCl was added to each followed by shaking and centrifugation as before. The organic layer was aspirated to waste and 2 mL sodium carbonate added to neutralize the acid. Subsequently, 2 mL pH 9.8 carbonate buffer was added followed by 200 μL toluene/hexane/isoamyl alcohol (39:10:1). All tubes were then mixed on a mechanical shaker at high speed for 10 min and centrifuged as above. The solvent layer was removed to an autosampler vial. Five microliters of the calibrator, control, and unknown extracts was injected on an Agilent 6890 gas

[1]New York City Office of Chief Medical Examiner and Department of Forensic Medicine, New York University School of Medicine, New York, NY 10016.
[2]New York City Office of Chief Medical Examiner, New York, NY 10016.
[3]Forensic Toxicology Laboratory, New York City Office of Chief Medical Examiner and Department of Forensic Medicine, New York University School of Medicine, New York, NY 10016.

Received 1 Dec. 2009; and in revised form 1 Mar. 2010; accepted 13 Mar. 2010.

chromatograph (Agilent Technologies, Wilmington, DE) equipped with a HP-17 megabore column (50% phenyl 50% methylsiloxane) and nitrogen phosphorus detector. The injection port, operated in the pulsed splitless mode, was set to 275°C; detector temperature was 325°C. The initial oven temperature was 190°C ramped at 10°/min to 230°C, held for 2 min, ramped again at 15°/min to a final temperature of 280°C with a 20 min hold.

Samples whose resulting chromatography displayed a response for PCP (relative retention time 0.60) were submitted for analysis and identification by gas chromatography/mass spectrometry (GC/MS) coupled to an Agilent 5973 or 6890 MSD. While not identical, the GC conditions for separation were similar to those for the nitrogen phosphorus detector GC analysis. The MSD was operated in electron ionization scan mode.

Solid-Phase Extraction Procedure for the Quantitation of PCP in Blood and Brain by Gas Chromatography/Mass Spectrometry in the Single Ion Monitoring Mode

Once PCP was identified as present, the subject specimen was extracted for GC/MS (Single Ion Monitoring Mode [SIM]) analysis. One milliliter of autopsy blood, autopsy brain homogenate, validated negative blood, or validated negative brain homogenate was aliquoted into appropriately labeled extraction tubes. A five-point calibration curve (1, 5, 10, 50, and 100 ng/mL) and a positive control (2 ng/mL) were prepared in the validated negative blood or validated negative brain homogenate. Ten microliters of internal standard, PCP deuterated at five carbons on the benzene ring (PCP-D_5) (Cerriliant), was added to each extraction tube to a final concentration of 0.5 mL. This was followed by 3.0 mL pH 4.5 sodium acetate buffer. The tubes were vortex mixed and centrifuged for 20 min at *c.* 1650 × *g*, and the supernatant was poured into a Polychrom Clin II solid-phase extraction column (Cerex; Speware Corporation, Baldwin Park, CA). Positive pressure was applied to achieve a flow rate of 1–2 mL/min. The columns were washed with 1 mL pH 9.0 potassium carbonate buffer and 1 mL deionized water and dried under positive pressure for 15 min at *c.* 25 psi. PCP was eluted with 2.0 mL ethyl acetate containing 2% ammonium hydroxide. The eluent was evaporated to dryness at 40°C, reconstituted with 75 µL ethyl acetate, and sent for MS analysis.

Separation for SIM MS was performed on an Agilent HP6890 GC in pulsed splitless mode with a 15 m RTX50 capillary column (50% phenyl-50% methyl polysiloxane) (Restek Corporation, Bellefonte, PA) with the following parameters: injection port temperature 260°C, oven 140°C for 1 min, ramp 15°/min to 295°C, hold for 3.6 min, and transfer line temperature 280°C. The GC was coupled to a HP5973 mass selective detector set to monitor the following ions: 200, 242, and 243 m/z for parent PCP and 205, 247, and 248 m/z for the PCP-D_5 internal standard.

For results to be accepted, all ion ratios from controls and unknowns had to be within ±20% of those of the calibrators, and quantitative quality control results had to be within ±20% of the target concentration. The PCP concentration in an unknown sample was determined by comparing the ratio of the quantitative ions (200, 205 m/z) to a nonlinear curve determined from the corresponding quantitative ion ratios of the calibration curve.

Results

Over the time frame of the study, PCP was detected in 138 blood samples. In 131 cases (95%), the blood was obtained from the heart and in seven (5%) from the femoral vein. The blood PCP concentrations are from the heart except where specified (case 4). Of the 138 cases, there were 52 deaths because of mixed drug intoxication (not including marijuana). The blood PCP concentrations ranged from <1 to 598 ng/mL. The case with the lowest concentration was accompanied by cocaine 0.05 mg/L, methadone 0.71 mg/L, and amitriptyline 0.12 mg/L in the blood. The case with the highest concentration also contained ethanol 0.08 g% and morphine 0.15 mg/L in the blood. Of the 52 deaths, one was deemed to be a suicide. He was 29 years old and had called his sister to say he would take his own life before being found dead on a grassy area. His blood PCP was 41.0 ng/mL. In addition, he had amitriptyline 0.89 mg/L and nortriptyline 0.78 mg/L in his blood. There were 80 violent deaths in which PCP was present in the blood. There were 30 firearm injuries, 10 vehicular accidents, nine sharp force injuries, nine falls from a height, five drownings, four hangings, four blunt force assaults, three thermal injuries, and one each of electrocution, crushing injury, fall down stairs, smoke inhalation, suicidal asphyxiation, and foreign body asphyxiation. The lowest PCP concentration (<1.0 ng/mL) was in an 18-year-old man gunshot wound homicide victim. The highest concentration (581.4 ng/mL) was in a 23-year-old man who was witnessed to dive into a pond and not come up. Of these 80 individuals, 13 (16.25%) had PCP only, 20 had PCP accompanied by marijuana, and 47 (58.75%) had PCP in combination with multiple other substances.

In addition to the 13 violent deaths in which PCP only was detected, there were five nonviolent cases in which only PCP was detected in the blood. In four of the five, there were associated underlying medical conditions that could also have contributed to death.

Case 1 was a 25-year-old man who was found unresponsive at home and pronounced dead on arrival at the emergency room. At autopsy, he was found to be morbidly obese (body mass index 54) with associated cardiac hypertrophy (heart weight 790 gm). His blood PCP concentration was 53.4 ng/mL.

Case 2 was a 50-year-old man with a history of diabetes mellitus who collapsed in front of coworkers and was pronounced dead on arrival at the emergency room. At autopsy, he was found to be obese (body mass index 40). He had marked coronary artery atherosclerosis, cardiac hypertrophy (heart weight 500 gm), and nephrosclerosis. His blood PCP concentration was 75.0 ng/mL.

Case 3 was a 38-year-old man with a history of drug abuse who was found unresponsive inside his automobile. He was pronounced dead on arrival at the emergency room. At autopsy, he was found to be obese (body mass index 32) with associated cardiac hypertrophy (heart weight 480 gm). His blood PCP concentration was 361.0 ng/mL.

Case 4 was a 37-year-old man with a history of substance abuse who was found unresponsive outdoors. He was transported to the emergency room where he was pronounced dead. At autopsy, he was found to have cardiac hypertrophy (heart weight 450 gm) with marked nephrosclerosis. His femoral blood PCP concentration was 361.3 ng/mL.

Case 5 was a 30-year-old man who was admitted to the emergency room in an agitated state after "doing PCP." His urine toxicology screen was positive for cannabinoids. It was negative for barbiturates, benzodiazepines, cocaine, methadone, and opiates. His serum was negative for ethanol, acetaminophen, and salicylates. No tests were performed for PCP. In the emergency room, he was sedated with Ativan, Haldol, and Benadryl. One hour after arriving at the emergency room, he had a cardiopulmonary arrest and was resuscitated. He never regained consciousness and was pronounced dead by neurological criteria and removed from ventilatory support 5 days later. At autopsy, his blood PCP concentration was

70.0 ng/mL. His only other significant autopsy finding was anoxic encephalopathy.

There was one case that did not fit neatly into any category. He was 27 years old and was found frozen in the ice of a lake. The PCP concentration in decomposition fluid was 129.9 ng/mL, which also contained 0.05 g% of ethanol. His cause and manner of death were undetermined.

Discussion

Phencyclidine was first synthesized as a dissociative anesthetic in 1956 and recommended for clinical trials in humans in 1957. In a paper published in 1958, Greifenstein and DeVault (2) reported that it could produce a profound state of analgesia in primates. In seven human subjects, it was found to cause a consistent and significant rise in both systolic and diastolic blood pressure. Pulse rate did not change as consistently as blood pressure. There was no bradycardia, tachycardia, or arrhythmia. Respiration was not affected. It was subsequently used in 64 patients for various surgical procedures and found to be unsatisfactory for surgical procedures in 13: several patients were unmanageable postoperatively, they exhibited "severe excitation with manifestation of a state of near mania," and three exhibited minimal convulsive movements. Postoperatively, the patients were slightly disoriented as if intoxicated and exhibited an associated euphoria. Patients also experienced amnesia, and some had severe hallucinatory disturbances. They concluded that PCP acted primarily on the central nervous system either by stimulation or by depression. They found the effects in humans to be highly dose dependent. In anesthetic doses (0.25 mg/kg of body weight intravenously), there was a moderate hypertensive response with tachycardia and no significant change in respiratory function. With higher doses (0.5–1.0 mg/kg body weight intravenously), there was severe agitation, muscle rigidity, and seizures, without respiratory depression.

The following year, Meyer and Greifenstein (3) reported on 102 patients who were administered PCP: 80 received the drug intravenously and the others orally. They noted that the drug occasionally resulted in symptoms similar to those observed in individuals subjected to sensory deprivation. There was impairment of pain, touch, proprioception, and discriminative aspects of sensation. The effects of the drug were again found to be dose dependent, with motor function being unimpaired until high doses at which time ataxia and nystagmus resulted. At the point of sensory deprivation, there was anxiety, depression, and fear with impaired thinking and concentration. At higher doses, patients experienced delusions and hallucinations. Their findings suggested that the drug acted primarily on the sensory cortex, brain-stem, and thalamus. In the same year, Chen et al. (4) published their findings in animal experiments and confirmed Greifenstein's findings in humans that PCP acts primarily on the central nervous system either by stimulation or by depression. Signs and symptoms were dose dependent. At anesthetic dosages, respiration and blood pressure were not markedly suppressed. However, at highly toxic doses, it caused transient respiratory depression, hypotension, and bradycardia. In more recent animal experiments, Milosevic et al. (5) found that PCP was neurotoxic, resulting in apoptosis. The mechanism of early neuronal cell death may be related to extracellular dopamine accumulation (dopamine is potentially neurotoxic) given that PCP is known to block dopamine reuptake. This may explain some of the cognitive dysfunction, memory deficiency, and psychotic episodes that may be observed in PCP users.

Therefore, while PCP proved generally effective as an anesthetic and in fact superior to many anesthetics because it did not depress respiration, it produced unacceptable side effects including agitation, violent behavior, paranoid delusions, disorientation, delirium, and hallucinations. As a result of these side effects, human clinical investigation was abandoned in 1965. It was subsequently utilized as an animal tranquilizer in veterinary medicine but was replaced by the structurally similar ketamine in 1978. However, it is relatively easily synthesized and is inexpensive to produce and has become an illicit substance of abuse. At low doses, defined by Young et al. (1) as 1–5 mg, PCP produces disorganization of thought processes, distortion of body image, somatic and visual hallucinations, blurred vision, slurred speech, agitation, ataxia, tremors, anxiety, and euphoria as well as tachycardia, increased blood pressure, and loss of response to painful stimuli (1,6–9). At moderate doses, defined by Young et al. (1) as 5–10 mg, PCP causes excitation, dysarthria, ataxia, convulsive movements, stupor, and coma, as well as hypertension without respiratory depression (1,7–9). At high doses (20 mg or more), one may expect to see hyperthermia, muscle rigidity, rhabdomyolysis, depression of reflexes, opisthotonic posturing, generalized seizure activity, prolonged coma, and death. At these high doses, it acts as a respiratory depressant and may also cause arrhythmias and transient hypotension (1,6,8–10). Respiratory depression appears to be the most significant sequel of PCP poisoning (8).

Bailey et al. (11) reported that for casual users of PCP, the plasma concentration ranged from <10 to 812 ng/mL. Several authors have reported on nonfatal PCP intoxication. Most of these authors did not report the serum concentrations of PCP (6,12,13). In 26 cases reported by Pearce (14), the blood concentration ranged from 6.7 to 240 ng/mL. In addition to the PCP, four were also under the influence of alcohol and one was intoxicated with barbiturates and methaqualone. Reynolds (15) reported on a 22-year-old man who survived a suicide attempt utilizing PCP, and his blood PCP concentration was 530 ng/mL.

In our series, there were 80 violent deaths (58%) in which PCP was quantified in the blood but was unrelated to the cause of death. This is in keeping with other publications where most of the PCP positive fatalities were violent in nature (10,11,16,17). The highest PCP concentration in our cohort was 581.4 ng/mL (a 23-year-old man who drowned in a pond). In published studies, the concentrations of incidental blood PCP detection in nondrug-related (mostly violent) deaths ranged from <20 to 11,000 ng/mL (10,11,15–19). In a 1983 publication, Budd and Liu (19) found PCP in blood and other body fluids and tissues in 30 violent deaths and stated that "in most of the cases PCP use led to bizarre and/or irrational behavior that resulted in death of the individual rather than PCP overdose proving directly fatal."

Several authors have reported on accidental (10,11,17,18,20) and suicidal (18) PCP fatal intoxications in combination with other substances. In the accidental overdoses, the blood PCP concentration ranged from 8 to 1800 ng/mL, and in the suicide reported by Cravey et al. (18), it was 12,000 ng/mL. In our series, 52 deaths (38%) were because of combined drug intoxication including PCP. The highest blood PCP concentration in this group was 598.0 ng/mL. He was a 37-year-old chronic substance abuser who was found unresponsive at a train station and resuscitation was begun. However, he was pronounced dead on arrival at the hospital. In addition to PCP, his blood contained ethanol 0.08% and morphine 0.15 mg/L.

In 1972, Reed et al. (21) described a fatal case involving PCP only in a 19-year-old boy found dead in bed. His blood PCP concentration was 300 ng/mL. However, he was also found to have lobar pneumonia. A year later, Kessler et al. (22) reported on a 17-year-old who presented with seizures who failed to regain

consciousness and died 4 days after admission. Blood toxicology 3 h after admission showed PCP 7000 ng/mL and phenobarbital 0.21 mg/L (they do not say if the phenobarbital was given for the seizures). Subsequently, several authors have reported accidental (7,10,18,23) and suicidal (7,16–18) PCP fatal intoxications in the absence of other substances. In the accidental cases, the blood PCP ranged from 300 to 4000 ng/mL. In the suicide cases, the blood PCP ranged from 19,000 to 25,000 ng/mL. In a 1978 publication, Burns et al. (24) suggested that blood PCP concentrations >1000 ng/mL were associated with a comatose state and concentrations of 2000–2500 ng/mL are probably uniformly fatal. We had five nonviolent cases (3.6%) in which blood PCP exclusively was quantified. In four of them, there was significant associated underlying pathology that may have caused or contributed to death. All four had cardiac hypertrophy, and three of them were obese. In these four cases, the highest PCP concentration was 361.3 ng/mL. In the fifth case, there were no significant comorbid conditions to attribute to death. However, blood PCP concentrations were not measured on admission to the hospital, and by the time he was removed from the ventilator 5 days later, his postmortem blood PCP was 70.0 ng/mL. In publications by Caplan et al. (17), Poklis et al. (10), and Pestaner and Southall (23), 2.7%, 8%, and 15.5% of their reported cases, respectively, died from fatal PCP poisoning. Of note, in our 80 cases of violent deaths, seven individuals had blood PCP concentrations in excess of the highest concentration (361.3 ng/mL) seen in the five patients who had PCP only. Poklis et al. (10) showed that victims of overdose had concentrations similar or even lower than cases in which PCP was an incidental finding and stated that "blood PCP concentration of those dying from overdose and those dying from other causes overlap." They suggested "the development of tolerance among chronic users" and "the inherent low lethality of the drug itself" as reasons for this.

Our study showed that most (71.1%) individuals who had PCP in their blood at the time of autopsy had other substances in their blood as well: 52 polypharmacy drug fatalities and 47 violent deaths with associated combined drug intoxication. We had only five individuals whose deaths were not because of violence or combined drug intoxication. However, four of the five had associated underlying medical conditions that could have resulted in their deaths. Their blood PCP concentrations were well below fatal concentrations reported by other authors. This raises a question not previously considered in our literature search: is PCP lethal at lower concentrations than previously reported if there are associated preexisting medical conditions such as obesity, hypertension, and atherosclerotic cardiovascular disease? Our fifth PCP-only related death was in the hospital for 5 days and did not have a concentration measured on admission; therefore, no conclusions can be made from his postmortem blood concentration of 70 ng/mL. Based on our 138 cases, we are not able to determine a fatal blood concentration of PCP because it is frequently used in combination with other chemical agents. In addition, PCP is lipophilic and as a consequence is rapidly removed from the blood and stored in lipoid-rich sites such as brain and adipose tissue. Thus, patients may have higher concentrations of PCP in brain tissue and exhibit clinical manifestations even as the blood concentration declines (11). Of interest, in 44 (31.9%) of our 138 cases, brain PCP concentrations were determined. Twenty-six (59%) of the 44 had brain PCP concentrations greater than the blood concentration and 18 (41%) had brain PCP concentrations less than blood PCP. Of the five cases of nonviolent PCP only in the blood, brain PCP was measured in only one, case 2, and was 54.0 ng/g (less than the blood PCP concentration). In aggregate, it appears that at relatively low concentrations, PCP may be fatal or contribute to death in individuals with preexisting medical conditions. If, indeed any of our four subjects with comorbid conditions died as a result of PCP's effects, then this confirms other authors' assertions that there is an overlap with blood concentrations of individuals who die from PCP toxicity and those who die from nondrug-related causes who are incidentally found to have PCP in their blood.

References

1. Young T, Lawson GW, Gacono CB. Clinical aspects of phencyclidine (PCP). Int J Addict 1987;22(1):1–15.
2. Greifenstein FE, DeVault M. A study of a 1-aryl cyclo hexyl amine for anesthesia. Anesth Analg 1958;37(5):283–94.
3. Meyer JS, Greifenstein F, DeVault M. A new drug causing symptoms of sensory deprivation. J Nervous Mental Disease 1959;129:54–61.
4. Chen G, Ensor CR, Russell D, Bohner B. The pharmacology of 1-(1-phenylcyclohexyl) piperidine-HCL. J Pharmacol Exp Ther 1959;127: 241–50.
5. Milosevic J, Veskov R, Vasilev V, Rakic L, Ruzdijic S. Apoptosis induction by phencyclidine in the brain of rats of different ages. Addict Biol 2000;5:157–65.
6. Liden CB, Lovejoy FH, Costello CE. Phencyclidine (Sernylan) poisoning. J Pediatr 1973;83(5):844–5.
7. Burns RS, Lerner SE, Corrado R, James SH, Schnoll SH. Phencyclidine—states of acute intoxication and fatalities. West J Med 1975; 123(5):345–9.
8. Liden CB, Lovejoy FH, Costello CE. Phencyclidine: nine cases of poisoning. JAMA 1975;234(5):513–6.
9. Perry DC. PCP revisited. Clin Toxicol 1976;9(2):339–48.
10. Poklis A, Graham M, Maginn D, Branch CA, Gantner GE. Phencyclidine and violent deaths in St. Louis, Missouri: a survey of medical examiner's cases from 1977 through 1986. Am J Drug Alcohol Abuse 1990;16(3 & 4):265–74.
11. Bailey DN, Shaw RF, Guba JJ. Phencyclidine abuse: plasma levels and clinical findings in casual users and in phencyclidine-related deaths. J Anal Toxicol 1978;2:233–7.
12. Burns RS, Lerner SE. Perspectives: acute phencyclidine intoxication. Clin Toxicol 1976;9(4):477–501.
13. Marshman JA, Ramsay MP, Sellers EM. Quantitation of phencyclidine in biological fluids and application to human overdose. Toxicol Appl Pharmacol 1976;35:129–36.
14. Pearce DS. Detection and quantitation of phencyclidine in blood by use of [$^2H_5$] phencyclidine as select ion monitoring applied to non-fatal cases of phencyclidine intoxication. Clin Chem 1976;22(10):1623–6.
15. Reynolds PC. Clinical and forensic experiences with phencyclidine. Clin Toxicol 1976;9(4):547–52.
16. Noguchi TT, Nakamura GR. Phencyclidine-related deaths in Los Angeles county, 1976. J Forensic Sci 1978;23:503–7.
17. Caplan YH, Orloff KG, Thompson BC, Fisher RS. Detection of phencyclidine in medical examiner's cases. J Anal Toxicol 1979;3:47–52.
18. Cravey RH, Reed D, Ragle JL. Phencyclidine-related deaths: a report of nine fatal cases. J Anal Toxicol 1979;3:199–201.
19. Budd RD, Liu Y. Phencyclidine concentrations in postmortem body fluids and tissues. J Toxicol Clin Toxicol 1983;19(8):843–50.
20. Stein GY, Fradin Z, Ori Y, Singer P, Korobko Y, Zeidman A. Phencyclidine-induced multi-organ failure. Isr Med Assoc J 2005;7:535–7.
21. Reed D, Cravey RH, Sedgwick PR. A fatal case involving phencyclidine. Bull Int Assoc Forensic Toxicol 1972;9(1):7.
22. Kessler GF, Demers LM, Berlin C, Brennan RW. Phencyclidine and fatal status epilepticus. NEJM 1974;291:979.
23. Pestaner JP, Southall PE. Sudden death during arrest and phencyclidine intoxication. Am J Forensic Med Pathol 2003;24(2):119–22.
24. Burns RS, Lerner SE. Causes of phencyclidine-related deaths. Clin Toxicol 1978;12(4):463–81.

Additional information and reprint requests:
Stephen deRoux, M.D.
Office of Chief Medical Examiner of the City of New York
520 First Avenue
New York, NY 10016
E-mail: sderoux@ocme.nyc.gov