

Letters to the Editor

Do We Know the Lethal Dose of Cocaine?¹

Dear Sir:

Cocaine use and abuse, and hence related fatalities, have increased significantly over the past several years (for example, Refs 1 and 2). Mittleman and Wetli [3] and Washton and Tatarsky [4], among others, have reported increases in admissions to treatment facilities in the United States for cocaine-related problems. According to the Bureau of Dangerous Drugs, convictions for possession and trafficking in Canada have gone up from 44 in 1972 to 847 in 1980 and 1390 in 1983 [5]. Although only about 3.3% of Canadian adults have used cocaine [5], 6.5% of all American adults aged 26 years or more and 28.3% of young adults have done so [6]. A report by the White House Strategy Council on Drug Abuse estimated that 10 million Americans had taken cocaine within the preceding 12 months [7].

In the literature, surprisingly frequent mention is made of the consumption of what might be termed fantastic amounts of cocaine. For example, Siegel [8] notes the case of a 28-year-old male in the habit of smoking 14 g of freebase a day, whereas death has been known to occur after intravenous administration of as little as 20 mg [9].

Several authors (for example, Refs 9 and 10) have emphasized the variability and unpredictability of the fatal dose of cocaine. A typical statement may be found in a 1984 paper by Nanji and Filipenko [11]: "While the oral lethal dose of cocaine is given as over 1 gram, death occurs with as little as 20 mg given intravenously." Similarly, Stark et al. [12] recommend that "therapeutic doses of cocaine be restricted to less than 200 mg, and that dosage be minimized whenever possible." And almost 30 years ago, Adriani and Campbell [13] summed up the situation as follows: "It is impossible to state the lethal doses of the different anesthetics [including cocaine] because such doses vary with the route of administration, rate of absorption and elimination, and tolerance of the individual to the drugs." Our literature search did not produce evidence of much more precise or sophisticated knowledge on this topic in the 1980s.

This search was coincident with preparation of an annotated bibliography on the adverse effects of nonmedical cocaine use [14]. Coverage includes major English, French, and German papers from 1880 to 1984, among which were discovered 10 animal and 18 human studies specifically discussing the question of cocaine's lethal dose (see Tables 1 and 2).

Information about the safety of various doses of cocaine for man should ideally include the maximum nonlethal dose, the LD₅₀, the LD₁₀₀, and the level at which there is no possibility of toxic effect for all subjects or "NOEL" [15]. Such information is difficult to find, partly because LD₅₀ and LD₁₀₀ studies cannot be done on humans, and partly because sniffing and freebase smoking which are popular among users were not replicated in the animal experiments we discovered.

Aside from these problems, there are numerous methodological issues. LD₅₀ studies have received much recent criticism [15-17]. Many experimental and environmental factors affect the LD₅₀ and it is known not to be a biological constant [18]. Nevertheless, for cocaine, some estimate of the lethal dose would be helpful, as the amounts taken and the number of users are increasing.

Almost all of the ten animal studies disclosed by our search were done in the past ten years employing modern experimental techniques. Only two studies used a route of administration

¹The views expressed in this document are those of the authors and do not necessarily reflect those of the Addiction Research Foundation. The authors wish to thank Dr. H. Kalant for his comments on an earlier draft of this paper.

TABLE 1—*Animal studies on lethal dose of cocaine.*

Reference	Species (N)	Cause of Death or Symptoms Preceding Death	Lethal Dose	Meaning
1925, Tatum et al. [25]	(1) rabbit (15) (2) dog (26) (3) cat (?)	respiratory depression, convulsions, hyperpyrexia	(1) 100 mg/kg (2) 26.7 mg/kg (3) 35 mg/kg	minimal fatal s.c. dose average fatal dose average fatal dose
1932, Downs and Eddy [19]	rat (10)	rapid respiration and convulsions	75 mg/kg	repeated injections every 24 h or 72 h for 5 to 44 days killed all rats
1968, Suzuki et al. [28]	guinea pig (21)	inability to keep posture, increased heart and respiratory rates, cyanosis, cardiac arrest	25–28 mg/kg	submucosal, single injection in palate killed all guinea pigs
1977, Evans et al. [22]	mouse (30+)	respiratory failure	75 mg/kg	intra-peritoneal injection killed 53% of male mice and 74% of female mice
1977, Post [20]	rat (6)	convulsions	60 mg/kg	average of 21 injections before death in all rats
1978, Cuttravas et al. [27]	dog (40)	convulsions, increased heart and respiratory rates, increased body temperature	(1) 13 mg/kg (2) 20 mg/kg	acute intravenous LD ₅₀ acute intravenous LD ₉₉
1978, Epstein and Altshuler [21]	rat (20)	seizures and weight loss	35 mg/kg	repeated intra-peritoneal injections once or twice daily eventually killed all rats receiving this amount (N = 10)
1978, Waters et al. [26]	dog (17)	convulsions, increased body temperature, increased respiratory and heart rates	20–24 mg/kg	mean lethal dose calculated after repeated intravenous injections killed all animals
1982, Bedford et al. [23]	mouse (?)	not stated	95.1 mg/kg	intra-peritoneal LD ₅₀
1982, Fantel and MacPhail [24]	rat (?) mouse (?)	not stated	75 mg/kg	injections intra-peritoneal lethal to all maternal animals on or before Day 14

used by humans (intravenous); the others used subcutaneous, submucosal, or intraperitoneal injections. A summary of the available studies is shown in Table 1.

The 4 studies on rats were done with different methods. The early study by Downs and Eddy [19] gave 5 male and 5 female albino rats intraperitoneal injections of a daily single dose (75 mg/kg of body weight) selected because it was below the "invariably fatal dose of 100 mg/kg." The average number of days to death was 14.2 for males and 34.8 for females. In a similar study, Post [20] gave 60 mg/kg daily to 8 rats; 6 died after an average of 21 injections. However, 2 survived for an average of 80 injections, hence individual differences in susceptibility are very large. In both studies, rats typically died of convulsions.

A larger study by Epstein and Altshuler [21] examined 20 male rats in groups of 4 each. The groups received saline, 20 or 35 mg/kg each day or 20 or 35 mg/kg twice a day. Only 2 seizures occurred in the 20-mg/kg group during 300 days of administration, on Days 162 and 204. Both animals died. All animals receiving 35 mg/kg of cocaine had seizures and died. Those receiving 35 mg/kg once a day had slightly fewer days ($N = 47.0$) before the first seizure than did those which got 2 doses per day ($N = 51.8$). Some tolerance for the effects of cocaine appears to have developed in animals receiving 2 doses.

Two studies of mice also give rather different results. Both used Swiss mice from the same supplier. Evans et al. [22] found that intraperitoneal doses of 75 mg/kg resulted in 53% mortality in males and 74% in females. As in earlier studies, phenobarbital pretreatment reduced the lethality of cocaine. In a similar study, Bedford et al. [23] found that the LD_{50} was 95.1 mg/kg (89.2 – 101.5).

Some indication of the lethality of cocaine in rats has been gained from a study of the teratogenicity of cocaine by Fantel and MacPhail [24] who gave 50, 60, and 75 mg/kg to groups of pregnant Sprague-Dawley rats. Death did not ensue after the 2 lowest doses; however, all rats ($N = 12$) died by Day 14 after receiving 75 mg/kg of cocaine hydrochloride. This is in contrast to Downs and Eddy [19] who found that females lived for an average of 34.2 days at that dose.

Four studies of cocaine lethality have used dogs. Tatum et al. [25] in an early study found that the minimal lethal subcutaneous dose for the dog was between 15 and 40 mg/kg with an average of 27.7 mg/kg. The use of barbital sodium before administering cocaine increased the minimal lethal dose to about 100 mg/kg. Waters et al. [26] gave increasing intravenous doses to dogs ($N = 17$) until death. They found that the mean lethal dose was 22 ± 2 mg/kg. Chlorpromazine pretreatment prevented most of the cardiovascular, respiratory, and biochemical response to cocaine. Catravas et al. [27] used forty dogs in a study of eight different intravenous dose levels. The LD_{50} was 13 mg/kg with 95% confidence limits of 11.9 to 14.2 mg/kg. The LD_{99} of cocaine was 20 mg/kg (16.5 to 24.4 mg/kg). Clearly, these levels are well below those found by Tatum et al. [25] and Waters et al. [26], possibly because of variation in experimental methods and procedures.

Only one study has been done for rabbits [25] and guinea pigs [28]. Tatum et al. [25] found that the minimum lethal dose for rabbits was 100 mg/kg (route of administration not specified). For the guinea pig, a submucosal injection of 25 to 28 mg/kg was fatal for all ($N = 21$). However only 35% died after an injection of the same amount into the femoral vein, and none died when the injection was into the abdominal wall.

Results from animal studies on cocaine are particularly difficult to apply to human situations. The lethality of cocaine varies greatly with the type of administration, but the favorite human method (sniffing) was not used in the animal studies. The LD_{100} for rats has been variously reported to be about 35, 60, or 100 mg/kg. For humans weighing 70 kg, this would be a range of doses from approximately 2.5 to 7 g. The LD_{100} for mice appears to be 75 to 100 mg/kg. For dogs, the LD_{100} has been reported to be 20 mg/kg intravenous or below the minimal lethal subcutaneous dose. In some studies, females were found to be especially sensitive to cocaine, and in others, much less sensitive, depending on species. Most studies show a wide range of lethal dose values with some animals able to tolerate four or five times as

TABLE 2—Cocaine human fatality studies.

Reference	Country	Number of Deaths	Sex of Victim(s)	Age of Victim(s) (in Years)	Cause of Death or Symptoms Preceding Death	Lethal Dose and Route (When Given) or Postmortem Blood Concentration (PMBC)
1893, Mattison [29]	U.S.A.	2	m	56,36	general paralysis	... approximately 0.78–0.98 g per os
1895, Garland [35]	U.K.	1	f	"young"	epileptiform convulsions	approximately 1.9 g/day
1902, Bose [36]	India	2	m	20,45	diarrhea, convulsions, anorexia, feeble heart	...
1913, Bose [30]	India	3	1m,2f	23,28,20	unconsciousness	...
1924, Mayer [37]	U.S.A.	26	convulsions and respiratory arrest	0.5 to 20% concentration in solution, sometimes with epinephrine, by parenteral injection
1972, Suarez et al. [32]	South America and U.S.A.	2	m	17,22	...	(1) 4 g (ruptured condom taken internally) (2) (8 burst packets taken internally)
1974, Price [38]	...	1	m	2 or 3 g per os; PMBC 0.8 mg/100 mL
1977, Lundberg et al. [39]	U.S.A.	3	1m,2f	33,16,28	pulmonary edema	0.5 g intravenously; PMBC 0.82 mg/100 mL, PMBC 0.75 mg/100 mL
1978, DiMaio and Gariotti [40]	U.S.A.	4	1m,3f	19,19, 28,26	convulsions, loss of consciousness, widespread brain damage, pulmonary edema	intravenous injection; PMBCs: 0.11, 0.37, 0.36, and 0.75 mg/dL
1978, Finkle and McCloskey [42]	U.S.A. and Canada	26	m : f = 3 : 1	16–35	seizures, respiratory arrest, coma, cardiac arrest	70% of cases had PMBCs of 9.0 µg/mL or less; 17.3%, more than 10 µg/mL; some, 4 µg/mL

1979, Wetli and Wright [43]	U.S.A.	24	17m,7f	15-36	respiratory depression and generalized seizures	average PMBC 0.60 mg/dL for 18 of 24 cases; others not known; routes intravenous injection, snorting or per os 100 g
1980, Aramayo and Sánchez	South America	1	acute intoxication	
1980, Bednarczyk et al. [47]	U.S.A. and South America	2	1f,1m	21,29	(1) convulsions (2) loss of consciousness, convulsions, cardiac and respiratory arrest 35 h after consuming 110 balloons filled with cocaine	(1) PMBC 5.0 mg/kg (intravenous injection) (2) PMBC 1.6 mg/kg
1981, Alired and Ewer [37]	U.S.A.	1	m	36	pulmonary edema	intravenous freebase
1981, Fishbain and Wetli [33]	U.S.A. and South America	1	m	...	respiratory arrest	one "cot" of cocaine intracorporeal smuggling)
1981, Nakamura and Noguchi [44]	U.S.A.	18	8m,6f (4 unsure)	20-40	pulmonary edema, hemorrhage, generalized visceral congestion	PMBC 0.21 mg/L
1984, Mittleman and Wetli [5]	U.S.A.	36	m:f = 1.4:1	15-52	respiratory depression	average PMBC 6.2 mg/L
1984, Nanji and Filipenko [11]	Canada	1	f	23	cerebral edema, coma, asystole, ventricular arrhythmias	...

much cocaine as others. Various tranquilizers and barbiturates can mitigate the adverse effects of cocaine and prevent death.

Lethal dose studies in humans consist mainly of clinical reports of death from accidental or purposive ingestion of large amounts of cocaine, mostly by heavy users. Unfortunately, however, such reports do not necessarily elucidate the question of how much cocaine it took to cause death. The victims have often ingested other drugs at about the same time as they took cocaine. Also, if the amount consumed is not known (as in most cases) it has to be inferred, either from the postmortem blood concentration or from circumstantial evidence. None of the papers reviewed contains any discussion of the relationship between postmortem blood concentrations and lethal dose.²

Seventeen studies report death from cocaine use with some indication of the dose taken or the postmortem level of cocaine in the blood. These studies can be grouped into several types. The first type give no reported postmortem blood level for cocaine and only nonquantitative information about the dose taken. This includes the early studies by Mattison [29] and Bose [30] and the more recent study of death in a freebase smoker by Allred and Ewer [31].

Another type of study involved "body packers" or smugglers who die when large amounts of cocaine are absorbed after plastic-wrapped bags of cocaine rupture inside the gastrointestinal tract. The study by Suarez et al. [32] reported two deaths in "body packers" where 4 g or more of cocaine were absorbed. Fishbain and Wetli [33] also described a "body packer" death, but did not describe the amount of cocaine taken. Another death in a "body packer" reported by Fishbain [34] had a postmortem blood level of 0.21 mg/L. Studies of "body packers" are not very helpful in establishing lethal doses as deaths result from absorption of cocaine through the gastrointestinal tract, a form of administration rarely experienced by cocaine users. Also, huge amounts are absorbed quickly so that several times the real lethal dose may enter the blood stream.

Only 11 studies report doses or postmortem blood levels of cocaine in persons other than "body packers." Several early studies report medicinal use of cocaine but give no information on postmortem blood levels. For example, Garland [35] reported on a death from convulsions in a patient who had taken 2 drachms orally of a 10% solution of hydrochloride of cocaine for a toothache. (In this context, referring to a solution, "drachm" is a unit of volume, not weight. One fluid drachm = 1 teaspoonful, or about 4 mL. Hence, 2 drachms = 8 mL. If the solution was 10% w/v, the amount of cocaine would be 0.8 g, or 800 mg. Garland actually said 12 to 15 grains, which = 780 to 975 mg). Bose [36] described two cases of death from cocaine use in India, but only in one case was the amount taken noted (30 grains a day) (1 grain = 0.0648 g). Also, Mayer [37] reviewed some 26 deaths as a result of the use of cocaine (alone and with procaine) as local anesthetics. The cocaine was used in various concentrations (0.5 to 20% solutions) and sometimes with epinephrine; usually death resulted from an accident or an unusually large dose. Unfortunately, Mayer [37] does not report details of individual cases, and the average amounts of cocaine leading to death are not stated.

Modern studies of deaths from illicit cocaine frequently give an estimate of doses or postmortem blood levels, but the levels found vary greatly. For example, Price [38] reported on a death in a cocaine addict who took 2 to 3 g orally. The postmortem blood level was 8 ppm (8 $\mu\text{g}/\text{mL}$) of cocaine. Lundberg et al. [39] reported three cases of death caused solely by cocaine intravenous injections (no other drugs found). In one case, death ensued from an injection of 0.5 g of street cocaine with an unknown potency. The doses taken in the other two cases were unknown but the postmortem blood concentrations were close at 0.82 and 0.75 mg/100 mL. However, DiMaio and Garriott [40] reported four deaths all from intravenous cocaine use, and blood levels of cocaine at autopsy ranged from 0.11 to 75 mg/100 g. All of

²In a personal communication to the authors (1985), Dr. B. S. Finkle has noted that "it is virtually impossible to infer with any accuracy dose ingestion on the basis of a postmortem blood concentration."

those who died appeared to be heavy users of intravenous drugs. Finally, Bednarczyk et al. [41] described a case of death after a cocaine injection, in which the postmortem blood level was 5.0 mg/kg.

Only 3 studies [42–44] have reported on more than a few deaths from cocaine, providing data on series numbering 26, 24, and 18 cases, respectively, and including postmortem and toxicological results.

Finkle and McCloskey [42] described 111 cocaine-related deaths of which 26 were due solely to cocaine. Surprisingly, 7.7% of cases were a result of sniffing cocaine, casting doubt on past assumptions that this method is relatively safe. More recently, Stark et al. [12], Lichtenfeld et al. [45], and Myers and Earnest [46] have explicitly refuted the notion that nasal inhalation is a safe way to take cocaine. The latter team report fatalities after inhalation, ingestion, and injection, confirming the observation of Lichtenfeld et al. [45] that “[n]o particular route of administration seems to play a dominant role in a fatal outcome.” The variation in cocaine blood levels was considerable (1 to 25 $\mu\text{g}/\text{mL}$), but in some cases, the level was 4 $\mu\text{g}/\text{mL}$ or below.

Wetli and Wright [43] reported on 24 deaths from cocaine. Blood levels were measured for 13 cases. The highest blood-cocaine levels were found for those who took the drug orally (0.92 mg/dL), some of whom were “body packers.” Snorters had the next highest levels (0.44 mg/dL) followed by intravenous injectors (0.30 mg/dL). They also reported 4 deaths with levels under 0.10 mg/dL—a rather low level.

A similar study by Nakamura and Noguchi [44] examined 18 deaths of which cocaine was the sole cause. Almost all cases involved injection, but there were a few cases of sniffing. The average blood-cocaine level was 0.28 mg/dL which is very close to that found by Wetli and Wright [43]. Again, the range of values was very great (0.08 to 80 mg/dL).

The human studies of cocaine-related fatalities give only a general impression of lethal doses. Virtually no direct information on doses is available in the studies of death from illicit cocaine. Indications from some studies of medicinal cocaine show that death can ensue from 0.8 to 1 g. We have, at best, postmortem blood levels of cocaine in most studies of illicit use, although a dose of only 0.5 g has been fatal in at least one case.

None of the papers examined shows how to translate postmortem values into actual doses taken. Monkeys receiving 15 mg/kg intraperitoneally (or about 1 g for a 70-kg human) had concentrations of 0.7 to 1.0 $\mu\text{g}/\text{mL}$, well below those usually found to be fatal in humans [42]. Finkle and McCloskey [42] have stated that the highest levels achieved by addicts taking a “large” intravenous street dose of cocaine are 4 to 7 g/mL. Suppose the “large” street dose is 500 mg, or about two times what users usually take. Finkle and McCloskey [42] found some users dying with postmortem cocaine levels at 4 $\mu\text{g}/\text{mL}$ or less. However, the highest level found after a large street dose cannot be equated with the residual level found in a postmortem sample, from which an unknown proportion has been removed enzymatically both pre- and post-mortem. Factors of tolerance, sensitization, metabolism, and many other circumstances may also be important in the individual case, and they are difficult to specify in advance.

Many authors mention the astonishing rapidity of death following overdose. For example, DiPalma [9] writes: “It is important to remember that the patient who is going to die of cocaine poisoning usually does so in a matter of minutes.” Jonsson et al. [1] speak of “sudden deaths” and “rapidly fatal course of massive cocaine poisoning.” McKinney and Benowitz [2] remark that “[c]ocaine related deaths were sudden . . . Patients did well if they arrived at the emergency room alive.” Thus, although it may not yet be possible to state the lethal dose of cocaine with certainty for all users (this indeed may never be possible), it is evident from our review that death from this drug is rapid, unpredictable, and likely to occur more frequently as numbers of users, experimentation with different routes of administration, and quantities consumed increase. Virtually no dose, however small, can be guaranteed safe for 100% of cases.

Much work remains to be done on cocaine-related deaths, but the available evidence suggests the following tentative conclusions:

1. The LD₁₀₀ of cocaine for rats varies between 35 and 100 mg/kg. For mice it varies from 75 to 100 mg/kg. For dogs it varies between 16.5 and 24.4 mg/kg.
2. The minimum lethal dose varies considerably from one species to another and with type of administration used. In animals, submucosal injections are more likely to be fatal than intravenous or intraperitoneal.
3. Several studies show that drugs such as barbital and chlorpromazine can reduce the lethality of cocaine.
4. Results are inconsistent concerning sex and lethality. Some studies show females more prone to the lethal effects of cocaine and some show the opposite, depending on the species.
5. Many studies of cocaine-related deaths in humans do not give information on dose levels or postmortem blood cocaine levels. Few studies of illicit cocaine use have both.
6. Studies of deaths among "body packers" are unhelpful in assessing lethal doses as such large amounts are absorbed involuntarily when the plastic containers break.
7. The range of postmortem cocaine-blood levels is very great (1 to 25 µg/mL) in one study of human deaths caused solely by cocaine.
8. Some users died with blood levels less than those achieved from a "large" street dose.
9. Death from cocaine, when it does occur, is usually rapid, sometimes unpredictable, and is likely to be reported with greater frequency if popular usage increases.

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Discussion of "Patterned Injury and Its Evidentiary Value"

Dear Sir:

The case report "Patterned Injury and Its Evidentiary Value" by Valerie J. Rao, M.D. (Vol. 31, No. 2, April 1986, pp. 768-772) raises several interesting points. Dr. Rao describes her method of preserving the evidence as one in which she made a tracing on clear acetate sheeting with a marking pen and describes it as "exact." Surely, that is an exaggeration. The area of the injury, as seen in the accompanying photograph, is not flat, and it would be very difficult, if not impossible, to cause acetate sheeting to conform to the rounded area. This would necessitate some shifting of the acetate as the tracing, by hand, progressed and this leaves the "exactness" of the method much in question. Surely a properly produced photomacrograph of the area would be much more exact.

Dr. Rao also discusses a procedure followed to determine whether a patterned shoe sole was responsible for the observed injury. Her procedure and her conclusions are somewhat questionable. She infers that her problem is akin to toolmark examination which in a sense it is, or more generally, to any type of impression comparison. When one approaches such a comparison, two basic principles should be kept in mind. One is that the method of comparison should be as direct as possible. That is, no intermediate step should be involved unless it is unavoidable. When comparing a two-dimensional impression to a suspect three-dimensional object, it is necessary to introduce an intermediate step because one has great difficulty visualizing the impression one would expect to find on a plane surface when impressed by an irregular three-dimensional object. To solve this dilemma, one makes another impression with the suspect instrument (being careful not to cause any change to it). This brings up the second basic principle that the comparison will only be reasonably valid if one can adhere fairly closely to the circumstances that pertain to the evidence impression. Without going into detail, these would include such things as using the suspect instrument, same amount of force, same direction of force, and so forth. In this case, it would be assumed that the suspect footwear would be used. Why the mirror image cast? What purpose did it serve? What was indented on the shoe is now raised, what was a raised area on the shoe is now indented. The shoe was on hand, why not use it?

I would also question the use of Play-doh[®] as a casting medium. If one chooses to make a cast, a material with excellent fidelity of size should be chosen. An evaluation of Play-doh indicates that it does not meet the criteria by a long shot. There are many good impression materials readily available through dental supply outlets (and probably at the University of Miami School of Medicine as well). One would hope that persons engaged in criminalistics would familiarize themselves with these materials and use them when required.

Finally, I would comment on the conclusions drawn. I'm not sure, from a reading of the

article, what the opinion is. Dr. Rao states, "It is my opinion that the edges of the circular areas resulted in the imprint." I assume she is not implying that the specific shoe in her possession made the mark. All that is available are a few indistinct class characteristics. The most that can be said is that a shoe with a sole pattern of that design "could have" made the mark—nothing more. Even the size could not be judged because the offshore manufacturers of this type of footwear often use the basic pattern for molds of different sizes.

Finally, not knowing the investigative setup in Florida (although I too watch *Miami Vice*), I pose the question, why is a medical examiner doing this work? Is there not a criminalist available who specializes in impression comparisons?

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Ontario Police College
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Author's Response

Dear Sir:

In Dade County in addition to the "usual" methods of preserving evidence for trials, that is, photography, body diagrams, and an autopsy protocol, some of us medical examiners do the "unusual." Acetate paper, which is thicker than usual paper and can be contoured around body surfaces, is used for documentation of stab wounds, both size and location, also for patterned injury. It is as "exact" as it can be, seeing that it is a tracing much like one would use tracing paper in outlining a map.

The reason for the use of Play-doh® is to demonstrate the potential use of an "adequate" medium to preserve evidence. If one is in a small community with limited resources and one has evidence which to the jury can be vividly demonstrated as in my case report, I strongly feel that no matter how unconventional the material available for use is, it should be employed in obtaining this evidence. We in our office have made use of the excellent crime laboratory we have in our jurisdiction—The Metropolitan Dade Police Crime Laboratory. This facility is staffed by a team of toolmark examiners and they utilize state-of-the-art casting material.

The Medical Examiner's Office in Dade County is unique in that each one of the medical examiner's has his or her own areas of interest, that is, facial reconstruction, drugs, sex, and santeria. My area of special interest is patterned injury. These interests are in addition to the "usual" expected duties of the medical examiner, and this is why I had the unique opportunity to work up and publish this case report.

On this particular case when I was asked in court if the shoe found on the crime scene caused the injury to the deceased, all I could testify to was that it was consistent with the injury pattern. It had class characteristics but no individual characteristics that were noted using the methodology I did. The jury had the opportunity of observing and handling the shoe and the Play-doh cast if they needed to. The judge several months after the trial met me in court and recalled the case. His comment to me was interesting—"your testimony and work on that case was better than Quincy."

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Civil Rightists Attack Psychosurgery

Sir:

Can conflict arise between the proponents of civil rights, a broad sociolegal issue, and proponents of psychosurgery, a neurosurgical operation? Such a conflict did occur, to the detriment of a small group of severely ill patients with extreme depression, compulsive disorder, and pain.

Early Psychosurgery

For patients with severe and intractable mental illness, Egaz Moniz, in the 1930s, proposed treatment by paired incisions in the white matter of the frontal lobes, therefore called a leucotomy or lobotomy, which yielded good clinical improvement [1,2], although with complications [3]. Moniz received world acclaim and the Nobel Prize. In this country, in the 1940s, fueled by wartime urgency, Freeman, joined by Watts, embarked on treatment of gravely ill schizophrenics in state hospitals, using a crude lobotomy [4,5]. Despite excessive use of this primitive procedure, many showed favorable results [6-8]. Improved technique, mainly with sick schizophrenics, but also some other psychotic patients, yielded an 83% improvement rate [9]. Further surgical modifications both maintained the success rate and markedly diminished complications [10,11].

However, in the 1950s, with increasing use of psychotropic medication, use of psychosurgery decreased.

Modern Procedure

With the development of stereotactic neurosurgical technique, precise placement of lesions in the brain became possible. For many psychosurgery patients, these are lesions 1 cm in diameter placed in the white matter of the rostral part of both cingulate gyri, hence, stereotactic cingulotomy. Patients for whom all previous treatment had failed, suffering intractable depression, recurrent suicide efforts, disabling anxiety, obsessive-compulsive disorders, and physical pain, were treated by cingulotomy. With this or a similar modern technique, using controls, blind assessors, quantification methods, and five-year follow-up, 75% of the patients improved markedly [12-14]. Creativity was not impaired, but instead improved in an architect and an artist [15,16]; an accountant, a lawyer, and a physician returned to their occupations [17,18].

Enter Antipsychiatrists

A small but articulate group of psychiatrists, many associated with Thomas Szasz, believe that mental illness does not exist, but that what appears to be mental illness is a problem in living, based on the social dysequilibrium that is caused by social and economic authority [19]. One of Szasz' followers, Breggin, founded an institute for "examining the impact of psychiatry upon personal freedom, political liberty and spiritual conception of man" [20,21]. Breggin said that "psychiatry has always been political, in that it's involved cooperation between the government and medicine for the control of large numbers of human beings," and that "psychiatry, with drugs and shock and state mental hospitals, had always been aimed more at the poor than at any other group." Breggin said that "psychiatrists, like most authoritarian men, expect women to be simple-minded." Breggin recruited Congressmen Louis Stokes of Ohio and Ron Gallegher of New Jersey for the board of this institute; its first act was its attack on psychosurgery. Its first document was Breggin's lengthy statement on psychosurgery in the Congressional Record of 24 Feb. 1972 [22].

Enter Civil Rights

Congressman Stokes claimed that it was well known that psychosurgery had the potential to become a tool for the societal and political repression of minority groups, political dissenters, and the poor. Further, he testified, psychosurgery has no therapeutic value and that there have been no successful psychosurgical operations, so that "the victims of psychosurgery are relegated to the class of subcitizens" [22]. A 1973 Michigan case [23] of *Kaimowitz* proved, he said, that these operations are experimental surgery. He concluded that, in the present context of racial and social mistrust, the practice of this surgery should be banned by Federal law [22].

Attorney Kaimowitz then testified on some doctors' honesty: "I fear," he said, "that surgeons would not reveal their experiments with psychosurgery until they were successful; alternatively, they would hide them under the guise of treatment" [22]. An articulate psychologist compared a demented individual who threatens to blow up a building and receives a diagnosis of mental disorder, with high public officials who order aerial and naval bombardment, "but somehow manage to escape such mental diagnosis or treatment" [24]. A lay writer warned that "Psychosurgery might even become part of the police armamentarium, along with mace, the club and the service revolver" [25].

With the support of Congressman Dellums of California, Congressman Gallegher inserted Breggin's statement in the Congressional Record, because "we share a concern that we're moving toward a therapeutic state and that we are in danger of having psychiatry usurp more of our political liberties" [20].

Commission's Research

A Congressional Commission appointed multidisciplinary teams to study psychosurgery, including the above material, both retrospectively and anterospectively [22]. The Freeman lobotomy, abandoned in the 1950s, was briefly mentioned. In a combined total of 600 patients, 1 was black, 2 were oriental, and 6 were hispanic, most were of middle class, 60% were women; all of these features matched the nonoperated controls.

Results showed a success rate of 78%, with particularly good results in depression and physical pain. Intelligence testing showed not worsening, but improvement, after the surgery. Complications were virtually absent. These previously very ill patients became able to work and to retain their marriages. Patients said they would give their informed consent again, under their preoperative circumstances. The Commission found that "these studies rebut the assumption that all psychosurgery is unsafe and ineffective," finding also that psychosurgery is not being used for social control.

Recommendations and Aftermath

However, this Commission found that psychosurgical procedures could not be considered fully "accepted medical practice" [26, 27], despite its research and previous studies, and that it was more nearly experimental than accepted practice. It therefore recommended that in each hospital performing psychosurgery, an Institutional Review Board [28] be constituted, and a National Psychosurgery Advisory Board similarly regulate [29] this treatment.

In late 1978, the Department of Health, Education and Welfare proposed that a group of psychiatrists, neurologists, neurosurgeons, and psychologists should voluntarily organize a Joint Committee on Psychosurgery to serve as a public advisory board. Response from these professionals was "minimal and unsatisfactory"; neither such a Joint Committee nor a National Psychosurgery Advisory Board has yet been formed [30, 31].

The Attack's Victims

Before this attack on psychosurgery was mounted, each year in this country about 700 patients received psychosurgical treatment for medical disorders causing otherwise hopeless depression, physical pain, or chronic obsessive anguish. After this unjustified attack by anti-psychiatrists and civil rights activists, only a fraction of this number—200 or less—have received psychosurgical treatment [32–34], with about 75% showing good improvement. The attack was a success. Its victims are those dead by suicide from untreated depression, or condemned to life in perpetual misery.

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Identification Strength Scale

Dear Sir:

At the annual meeting of the American Academy of Forensic Sciences in February of this year, the Odontology Section held a discussion session dealing with the problem concerning the wide variation in terminology used in establishing the strength of dental identifications. Although the intent of the session was not to rectify the situation as it exists at this time, the only conclusion that can be drawn is that everyone present agreed to disagree on a friendly and professional level. Additionally, the forensic odontology literature presents nonforensic professionals with a confusing variety of differences in terminology being used by this profession [1-3].

Because of the inability of the forensic science community to agree on a standard set of terms concerning the degree of positivity of an identification, I am proposing the following *Identification Strength Scale*. This six-point scale incorporates some commonly used terms in an effort to establish the parameters defined by the numeric values listed. The terms selected are intended merely as guidance, and in no way is it implied that other terms, not included in this discussion, are in any way less valid than those chosen as exemplars. Each member of the forensic science community need only insert his or her common terminology at the appropriate levels within the scale.

The scale is as follows:

5. Positive, Within reasonable dental (scientific) certainty, Absolute certainty.
4. Very probable, Highly probable, Highly consistent, Most likely.
3. Probable, Consistent with.
2. Presumptive by exclusion.
1. Can't exclude.
0. Mismatch, Unidentified.

This is not an altogether new concept, as the military has been using a clearly delineated list of identification terms for some time through the Armed Forces Institute of Pathology [4]. I have expanded this concept in an effort to approach a universal quantification concerning the positivity of an identification. Such an attempt at quantification runs along a somewhat similar line as what the American Board of Forensic Odontology has done with their bite mark scoring sheet [5].

Perhaps this is not the final solution to this complex problem. However, it is hoped that even if this scale is modified in the future at least the momentum will carry the seed of this idea to fruition.

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Sacco and Vanzetti Case

Dear Sir:

For Sacco and Vanzetti, death had no sting for it lacked finality. The controversial memories of their trial and executions are with us still and will, doubtlessly, be with us an infinitum. For myself, I had thought, ingenuously it now appears, that my two-part article in the *Journal of Forensic Sciences* (April and July 1986) would set the record straight at least with regard to the firearms evidence in the Sacco and Vanzetti case. But comes now Arthur J. McBay, Ph.D., to unknot further the record with a soupçon of telling new information.

Dr. McBay has written me and we have conversed about certain previously undisclosed and all-important scientific data in which Dr. McBay has personal knowledge concerning traces of blood on the mortal bullet (Bullet III) recovered from the body of payroll guard Berardelli by Dr. McGrath at the autopsy.

Dr. McBay's information sheds further light on the spurious nature of the charge that the mortal bullet was a figment of police creativity. I discuss this matter at p. 1067 of Part II of my article, but without the benefit of Dr. McBay's firsthand insights.

It appears that in 1961 Dr. McBay was Supervisor of the Massachusetts Department of Public Safety Laboratory. He was present when Dr. William C. Boyd of the Boston University School of Medicine conducted his ill-designed tests on the evidence bullets purportedly to discover any traces of blood on them. If blood there was, then the bullets had at least a glimmer of authenticity. But the ill-designed tests (a saline solution washing for each bullet) gave necessarily ill-fated results—a "weakly positive" benzidine reaction.

Dr. McBay, however, now informs me—in a first public disclosure—that before Boyd tested the bullets in 1961, but on the same day, Dr. McBay lightly rubbed filter paper on the base of the mortal bullet and upon testing it with the benzidine reagent, he detected a "distinct" positive reaction. Dr. McBay also rubbed filter paper on the other surfaces of the mortal bullet (the cupronickel jacket), but received no reaction in testing it with benzidine. Since copper and nickel are chemical catalysts which would interfere with a benzidine test, it is passing strange that Dr. McBay got no reaction from the filter paper rubbed on the circumference of the bullet. Lamentably, Dr. McBay made no contemporaneous notes of his testing.

No cymbals will clang and no horns will blow over Dr. McBay's disclosure, but it is another small piece of meaningful evidence in support of the view that police hanky-panky in

the substitution of the mortal bullet to obtain the conviction of Sacco and Vanzetti is, presumptively at least, a makeweight.

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Discussion of "A Review of *Fatal Vision*"

Reference: Book Review by Emanuel Tanay, M.D.
A Review of *Fatal Vision*
The Slanted Vision
The Jeffery MacDonald Tragedy
JFS, Vol. 31, No. 3, July 1986, pp. 1163-1165

Dear Sir:

Did Dr. Tanay read the same book we did?

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