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Narcotic-Related Deaths in the District of Columbia: 1971-1979

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ABSTRACT: Two hundred eighty-seven deaths directly related to narcotic abuse occurred in the District of Columbia between July 1971 and December 1979. Factors contributing to death in some cases included lack of opiate tolerance as well as the conjoint abuse of ethanol. Free morphine was identified more often in the blood of victims dying rapidly than in the blood of those with longer post-injection survival. A statistically significant correlation between the number of heroin-related fatalities and the purity of heroin available to the user was observed over the 8½-year study period.

KEYWORDS: toxicology, death, narcotics, heroin

The incidence of sudden death resulting from narcotic drug usage in the District of Columbia continues to be a dynamic and complex problem. Ongoing interest in the epidemiology of this type of death resulted in several previous publications from the District of Columbia Medical Examiner's Office, in collaboration with the Centers for Disease Control [*1-3*].

A number of factors unique to the District of Columbia have facilitated the present study of deaths directly related to narcotic abuse. The jurisdiction is of manageable size, permitting complete examination of all suspected cases. A single experienced police investigative unit provides information regarding the circumstances of death and follow-up studies when indicated. Uniform forensic scientific criteria were adhered to throughout the study period, including circumstantial, pathologic, and toxicologic investigative parameters.

Materials and Methods

Deaths directly related to narcotic use from July 1971 through December 1979 were reviewed. Each case was abstracted and analyzed according to 40 parameters, including demographic, circumstantial, pathologic, and toxicologic information.

Narcotic-related deaths were separated into categories depending on the circumstances of death. Those deaths directly related to the use of narcotic drugs constitute the present study group. Fatalities related to medical complications of narcotic usage and deaths resulting from unnatural causes in persons incidentally found to be narcotic users were excluded from this study.

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Within the cohort of direct narcotic-related deaths, each case was further subclassified as to the particular drug identified at autopsy. The latter included five separate drug categories: specifically, heroin, methadone, the combination of heroin and methadone, Dilaudid®, and unknown drug. In the several cases where a particular narcotic drug was not identified, there was overwhelming circumstantial and pathologic evidence of fatal narcotic abuse, in spite of negative toxicologic studies. In certain of the latter cases extensive postmortem decomposition precluded satisfactory toxicology analysis.

Morphine was identified in postmortem specimens by using the fluorometric procedure of Kupferberg, as modified by Goldbaum [*4*]. In 1979, this procedure was replaced by a homogeneous enzyme immunoassay technique (EMIT®) [*5*].

Fifty-eight of the cases reported here were included in a previous publication [*2*]. Each of these cases was reviewed and abstracted for the present study.

In 1960, the Narcotics Branch of the District of Columbia Metropolitan Police Department began collecting street heroin samples by random purchases that were submitted to and analyzed by the regional laboratory of the Drug Enforcement Administration. A monthly record of average street level heroin purity was obtained over the study years.

Results

All 287 deaths directly related to the abuse of narcotic drugs were reviewed. Of these, 193 were caused by the abuse of heroin, 64 by methadone, 21 by the combination of heroin and methadone, and 4 by Dilaudid; 5 were classified as unknown drug. A graph of the numbers of deaths per drug employed over time is presented in Fig. 1.

Demographic characteristics of the decedents are given in Table 1. The average age was 26.8 years; 93% of the victims were black and 83% were male.

In 32 of the 287 cases, heroin samples were known to have been used by more than one individual. In 27 of these instances only one of these individuals died. Three deaths resulted from the use of the same batch of 12% heroin. In a separate incident, two deaths resulted from the use of a single heroin sample of unknown purity.

In 54 of the 287 cases, there was a documented history of attempted "home" resuscitation by nonprofessionals. The most common methods employed included placing ice cubes on the victim (16 cases), placing the victim in the bathtub or shower (15 cases), and injecting salt water intravenously (11 cases). It is probable that nonprofessional resuscitation was attempted in more cases than was reported.

Marked geographic clustering of deaths within the principal narcotic usage area of the city was noted. One hundred thirty-five (47%) of the victims died in their own homes. No statistically significant seasonal or monthly differences in the incidence of narcotic-related fatalities were observed during the study period.

An estimate was made in each case as to the addiction status, and therefore the degree of opiate tolerance, at the time of death. The victim was either a chronic (tolerant) or a novice (intolerant) user. The latter group consisted of both previous addicts with current abstinence and neophytes. The decedents were so classified based on the presence or absence of (1) the cutaneous stigmata of chronic intravenous narcotism; (2) multiple recent and resolving injection sites found on subcutaneous examination; or (3) a history of continuous and long-term "hard drug" usage.

With these criteria, 238 (83%) of the 287 decedents were deemed to be addicts at some point in life. However, only 142 of the 287 (49%) were chronic users having opiate tolerance at the time of death. Of the remaining 145 individuals not known to be chronic users at the time of death, 85 (31%) had a history of recent or prolonged abstinence and were therefore considered to be intolerant, while in 60 cases (20%) insufficient data were available for accurate classification of the degree of tolerance.

Ethanol, in concentrations in excess of 0.1 g/dL, was identified in 89 (33%) of the 265

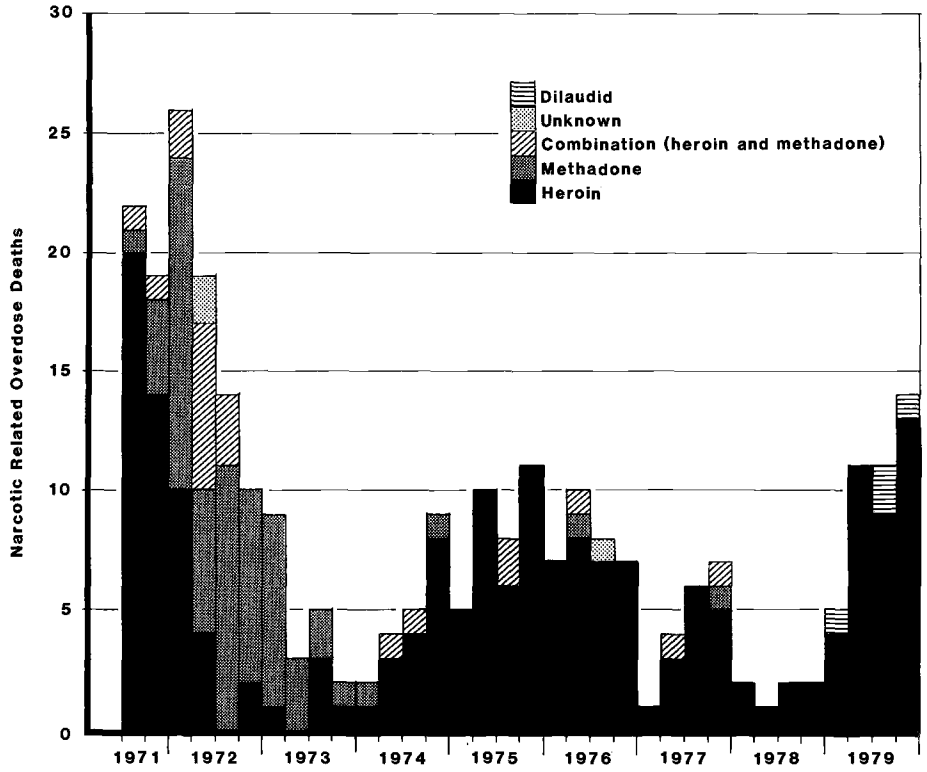

FIG. 1—*Fatalities directly related to the use of narcotics, per drug employed, by quarter from July 1971 through December 1979.*

TABLE 1—*Demographic data pertaining to victims of narcotic-related overdose deaths.*

	Cases		Male		Female	
	n	% of Total	*n*	%	*n*	%
Age						
Mean years	(26.8)	...	(27.0)	...	(25.6)	...
Range	(13-62)	...	(15-62)	...	(13-56)	...
Race						
Black	268	93	226	95	42	84
White	19	7	11	5	8	16
Total	287	100	237	83	50	17
Marital status						
Single	193	67	160	67.5	33	66
Married	78	27	64	27	14	28
Divorced	14	5	12	5	2	4
Unknown	2	1	1	0.4	1	2
Employment status						
Employed	147	51	140	59	7	14
Unemployed	139	48	97	41	42	84
Unknown	1	...	...	...	1	2

study cases in which the victim had not been hospitalized for more than 12 h. Thirty-seven percent (79 of 187 cases) of the deaths from heroin also had evidence of conjoint use of alcohol, whereas fatalities from methadone and from the combination of heroin and methadone exhibited a statistically significant lesser alcohol involvement, 20% (12 of 60 cases) and 17% (3 of 18 cases), respectively.

In addition to the principal narcotic drug, other drugs of abuse were identified in some cases. In 1973 a discrete "mini-epidemic" of methamphetamine abuse was noted over a four-month period, a phenomenon that was promptly curtailed by strict drug control measures [*6*]. Phenmetrazine, a drug of abuse seemingly unique to the District of Columbia [*7*], was identified with greatest frequency between 1973 and 1977. The percentage of cases per year is shown graphically in Fig. 2.

In 187 of the 193 heroin deaths, blood morphine analysis was performed. In the remaining cases, the victim had been hospitalized for more than 24 h. Free blood morphine in concentrations ranging from 0.005 to 0.2 μg/dL was found in 114 (61%) of these cases, the positive group. In 73 instances no free blood morphine was identified, the negative group. Of the 114 positive cases, 64 (56%) were known to have died or collapsed rapidly, whereas only 18 (25%) of the 73 negative cases were known to have died rapidly. An individual was considered to have died or collapsed rapidly if (a) the decedent was witnessed to have collapsed within 15 min after injection or if (b) the decedent was found with a needle still in the arm, or if a syringe was clutched in the hand or recovered directly alongside the body.

The relationship between the purity of street-level heroin and the number of heroin deaths per quarter during the study period is depicted in Fig. 3. As a measure of the degree of correlation between these two variables, a Pearson correlation coefficient of $r = +0.41$ was calculated, where a value of 0.0 would indicate no correlation and a value of 1.0 would indicate a perfect correlation. This is a statistically significant correlation, $P < 0.01$.

Discussion

During the 8½ years of the study period, a widely oscillating incidence of narcotic-related overdose deaths was observed. The changes involved both the type of drugs identified and the number of deaths per time period. Methadone use, prevalent in the early 1970s, declined sharply after 1973 when take-home privileges were eliminated at local narcotic treatment clinics. Heroin use followed a more variable course and was seen to increase dramatically in 1979, a pattern that continues to date. Dilaudid, a drug not encountered before 1979, was responsible for four deaths. Dilaudid was also identified in association with heroin in three additional cases.

The epidemiology of narcotic-related fatalities has been the subject of many previous studies [*1–3,8–20*]. In spite of these investigations, the exact pharmacophysiologic mechanism of death resulting from narcotic use is not yet fully understood. Early studies were hampered by relatively insensitive toxicologic methods. Consequently, a lack of toxicologic confirmation may have been one factor that promoted the invoking of various mechanisms of death other than pharmacologic overdose, including idiosyncratic response [*12*], hypersensitivity [*10*], quinine cardiotoxicity [*21*], immunologic reaction [*19*], and others. Recent studies employing more sensitive methods have quantitated small amounts of morphine in body tissue samples [*14,22*].

Previous reports suggested that the level of free blood morphine might correlate with survival time after injection. Our data confirm this theory in part. Garriott and Sturner [*14*] separated 22 heroin deaths into groups, according to length of post-injection survival, based on the pulmonary histopathologic changes first described by Siegel and co-workers [*23*]. Although we attempted this type of separation, in our experience pulmonary histopathologic changes are not consistently reproducible. We therefore separated our cases according to the toxicologic parameters determined by Garriott and Sturner, namely "short-interval" (sur-

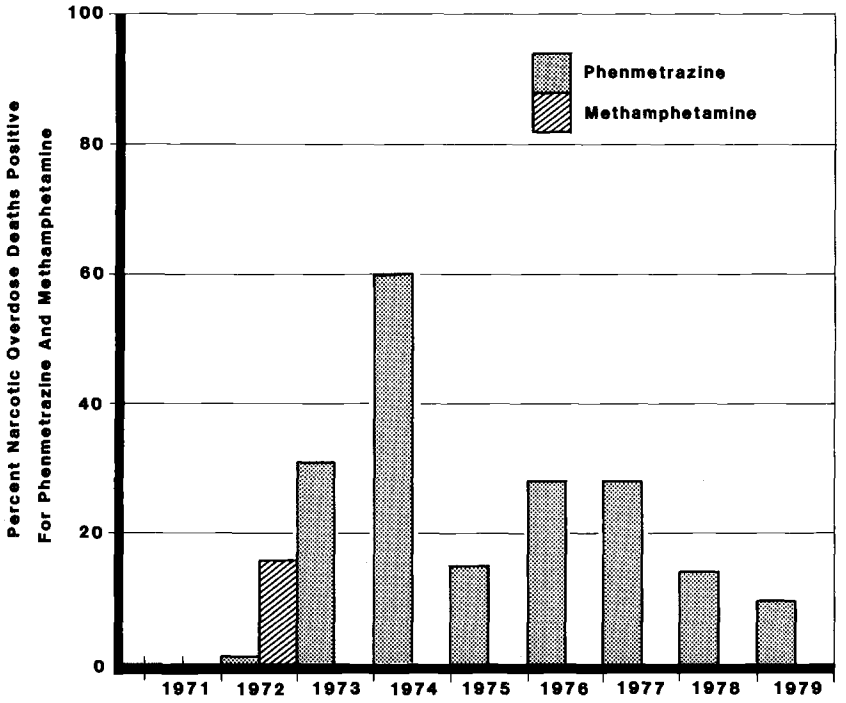

FIG. 2—*Percentage of narcotic-related fatalities involving phenmetrazine and methamphetamine, by year.*

vival less than 3 h, free blood morphine 10 to 93 μg/dL) and "intermediate-survival" (3- to 24-h survival, free blood morphine 3 to 10 μg/dL). With these criteria, no statistically significant difference between these two groups was found in terms of post-injection survival. There was, however, a significant difference between the entire positive group when compared to the negative group.

In a previous study, we postulated that levels of free blood morphine above 0.3 μg/mL might identify those victims who had died rapidly [*2*]. In an effort to reexamine this hypothesis we separated our cases into three groups, namely, blood morphine greater than 0.03 μg/mL but less than 0.3 μg/mL, blood morphine greater than 0.3 μg/mL, and blood morphine not detected. No statistically significant differences were found in the current study among these three groups relative to post-injection survival.

However, when the group with no free blood morphine was compared to the entire group with a positive free morphine level, we found that 64 of the 114 positive group (56%) died rapidly, while only 18 of the 73 negative group (25%) died rapidly. Stated in another way, of the 82 cases known to have died rapidly, 64 (78%) were found to have a positive level of free blood morphine. While in previous studies the absolute level of free morphine categorized as absent, low, or high correlated with survival time, in the present study only the presence or absence of free blood morphine was correlated with post-injection survival time.

In an earlier publication, we suggested that lack of opiate tolerance may potentiate opiate-related "overdose" fatalities [*2*]. Supportive data were published by Baselt and co-workers [*18*], who found elevated bile morphine levels in a group of "incidental addicts" dying from traumatic causes and lower levels in two overdose groups. They suggested that these findings might indicate the presence of tolerance in the trauma group and lack of tolerance in the overdose group. In 1971, a report from New York City [*24*] found 46% of 149 heroin over-

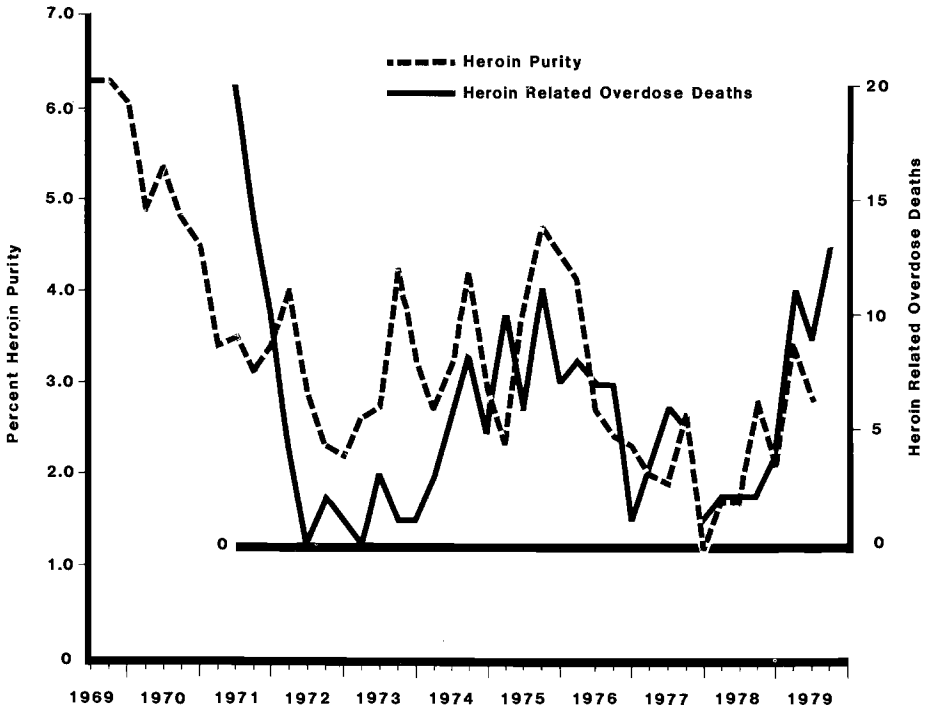

FIG. 3—*Relationship between street heroin purity and the number of fatalities directly related to heroin abuse by quarter from July 1971 through December 1979.*

dose victims to be only casual heroin users (and therefore intolerant), and an additional 26% had returned to heroin use after a period of abstinence. Gardner [*9*] reported that 26 of 47 (55%) opiate users in the United Kingdom had died shortly after a period of abstinence.

Louria [*25*], discussing the concept of tolerance, suggested two situations where lack of opiate tolerance might be critical. An addict who has abstained from drug use, whether because of incarceration, hospitalization, or free choice, may return to the drug community and inject himself with the same amount of heroin previously used. This disregard for his loss of tolerance may result in overdosage. Alternatively, a neophyte may inject an amount of heroin similar to that taken by his drug-tolerant cohorts but too potent for him. Supporting this concept, we identified 32 separate instances where more than one individual used the same heroin. In 25 of these 32 cases only one individual died, suggesting that lack of tolerance in that person may have been a factor causing death.

A third situation, one from which the addict has no protection, is one in which very pure heroin is used without prior warning. In 7 of the 193 deaths, high-quality heroin ranging in purity from 12 to 65% was identified. In 3 of these 7 cases the same batch of high-quality heroin was responsible for death. These findings point to a lack of tolerance for the amount of heroin injected.

According to the definitions presented earlier, 238 of the 287 decedents in this study had opiate tolerance at some point in life, whereas only 142 were considered to be tolerant at death. Of the 145 not known to be chronic users at death, 85 had a history of recent or prolonged abstinence. It is probable that more victims were intolerant but the case reports lacked the requisite history. Although our definitions are, by necessity, somewhat arbitrary, they are the best measurement parameters available and were applied equally in all cases. It

is probable that lack of opiate tolerance was a factor in causing the deaths of those individuals not using narcotic drugs regularly prior to the terminal injection.

A relationship between the purity of street-level heroin and heroin deaths was suggested by Huber and co-workers [*16*] in a study of 19 heroin overdose deaths in Atlanta, Georgia, in 1971. In 1974, we reported a statistically significant positive correlation ($r = +0.83$, $P < 0.01$) between heroin purity and the 58 heroin deaths occurring during the 18-month period between July 1971 and December 1973 [*2*]. In Atlanta, a significant, positive correlation ($r = +0.665$, $P < 0.001$) was determined in a similar study of 35 heroin deaths between January 1971 and December 1973 [*17*]. A statistically insignificant correlation ($r = +0.13$) was calculated during a five-year study in San Antonio, Texas, involving 100 heroin deaths [*20*]. The present study constitutes the largest number of heroin deaths (193) analyzed over the longest time interval (8½ years); a positive correlation of 0.41, $P < 0.01$ was observed between heroin purity and heroin fatalities.

These findings continue to point to an association between the level of heroin purity available to the user and the number of narcotic-related fatalities incident to use of that heroin. An alternative explanation noted by Alexander [*17*] as well as Huber et al [*16*] suggests that increased heroin purity reflects a period of increased heroin availability. More users and more frequent use resulting from increased availability may provide more opportunity for heroin-related fatalities to occur regardless of the mechanism of death, which may be unrelated to heroin potency.

Other causative factors may be involved in some deaths. The identification of other central nervous system depressants, ethanol in particular, in association with narcotics has been repeatedly observed [*10,12,14,18,20,22,26*]. In the present study significant levels of ethanol were found in one third of the cases. The relationship of alcohol to narcotic deaths is also a dynamic problem, with increasing numbers of alcohol-related cases being seen in the final study years. It is probable that ethanol potentiates the central nervous system depressant effects of opiates. In addition, individuals under the influence of alcohol might well become "careless" and inject more heroin than planned.

Another factor cited in the literature is the role of quinine [*10,21*]. Nearly all heroin available in the District of Columbia (and on the East Coast, in general) is cut with quinine. Of the 214 cases attributed to heroin abuse in this study, either singly or in combination with other narcotic drugs, quinine was identified in 170 cases. Heroin available in certain other regions of the country is not cut with quinine [*14,22*]. The fact that heroin-related fatalities in all geographic areas are similar in terms of signs, symptoms, pathology, and toxicology (with the exception of the presence of quinine) would argue against quinine's being a significant causative agent in these deaths.

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