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Analysis of Fatalities from Acute Narcotism in a Major Urban Area

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ABSTRACT: The incidence of acute, fatal narcotism in San Francisco was determined to be 3.2% of all deaths (10 882) subject to medical examiner's inquiry in a five-year period. Heroin was responsible for the greatest number of these cases, usually accompanied by alcohol or other abused drugs. The median concentration of the heroin metabolite, morphine, in the blood in fatal cases was 20 μg/dL. Death from propoxyphene, the second most frequently encountered narcotic, was generally determined to be suicidal, while death from heroin was judged to be accidental. The highest rate occurred in black males between the ages of 21 and 30 years. The three most consistent findings were positive identification of the drug in the body (100% of the cases), pulmonary edema (90.4% of the cases), and microscopic liver changes (71.1% of the cases).

KEYWORDS: pathology and biology, narcotics, death

In an earlier report from the Office of the Chief Medical Examiner-Coroner on acute heroin fatalities in San Francisco, Baselt et al [*1*] noted that the mortality rate from heroin overdoses had increased dramatically in that city since 1968 and was one of the highest in the nation. A similar epidemic has been reported in a number of other metropolitan areas of the United States, as well as in the United Kingdom, Europe, and Australia [*2-6*]. In this regard, Siegel et al [*7*] likened the increase in intravenous narcotism to an endemic situation, with an occasional short or prolonged outburst of epidemic proportions.

In this study we examined deaths from acute narcotism in a major U.S. city over a five-year period, involving over 10 000 medical examiner's cases. The demographic as well as the toxicological characteristics of the cases are evaluated.

Since the criteria used for the diagnosis of acute narcotism vary with different medicolegal jurisdictions, those used by the San Francisco Medical Examiner-Coroner's Office are described. The investigational steps involved in determining the cause and mode of death before filing of a death certificate in such cases are emphasized.

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Methods

General

All cases within the jurisdiction of the medical examiner-coroner in San Francisco undergo investigation, including inspection of the body, evaluation of the scene of death, collection of data concerning the decedent and the events surrounding death, autopsy of the body, performance of toxicologic screening tests, and review by the medical examiner. Cases are subject to different degrees of scrutiny thereafter. When the cause of death is not determined by a conventional autopsy, more extensive histological, bacteriological, biochemical, and toxicological studies may be instituted. A toxicological screen for barbiturates, alcohol, and other volatile substances is performed in all cases. Further toxicological examination for the identification and quantification of narcotics and other drugs, such as sedative-hypnotics and tranquilizers, may be done for any of the following reasons:

1. The deceased is the victim of a homicide.
2. The deceased has been involved in a serious accident.
3. Notes found at the scene indicate intent of suicide.
4. Unusual amounts of medication or empty drug containers are found on the premises.
5. Evidence of abuse drugs or paraphernalia used for their induction into the body is found.
6. Past history indicates that the person was a drug abuser.
7. Gross findings such as hypodermic needle marks or scarring of veins are observed at autopsy.
8. Microscopic findings suggestive of intravenous narcotism as a possible cause of death are found at autopsy.
9. There is no apparent cause of death.

Morphine and Codeine

Analytical procedures have been developed for the quantitative, as well as the qualitative, identification of important narcotic substances. The body fluids analyzed for narcotic substances include blood, bile, and urine.

Bile and urine samples are screened for morphine and other common opiates after acid hydrolysis by extraction by using the method of Fujimoto et al [*8*] and subsequent examination of the extract with thin-layer chromatography. Chromatograms are developed in benzene/dioxane/ethanol/ammonia (100:80:10:11) and methanol/ammonia (100:1.5). Visualization is by spraying with iodoplatinate and Dragendorff's reagent [*9*].

Quantification of morphine is by gas chromatography of the trimethylsilyl (TMS) derivatives, using *N*-allylnormorphine as an internal standard [*10*]. Morphine and *N*-allylnormorphine (internal standard) are isolated from samples by solvent extraction with ethyl acetate after pH adjustment to 8.5 $\pm$ 0.1 with a buffer solution. The ethyl acetate phase is back-extracted with dilute hydrochloric acid and the aqueous phase reduced to dryness using heat and vacuum. The residue is reconstituted in *N,O*-bis-(trimethylsilyl)-trifluoroacetamide (BSTFA), which acts both as a silylating agent and solvent.

Aliquots of the silylated extract are introduced into two gas chromatographs equipped with columns containing OV-1 and OV-17 on Chromasorb G. Identification is based on the characteristic retention times on the two columns. Quantification is by comparing the peak height response ratios (morphine/*N*-allylnormorphine) of unknowns to the peak height response ratios of standards carried through the procedure [*11*].

The detection limit for morphine (free base) is 0.5 μg/dL using a 5-mL sample. In addition to morphine and *N*-allylnormorphine, the method detects other opiates, such as diacetylmorphine, 6-monoacetylmorphine, 3-monoacetylmorphine, codeine, dihydroco-

deine and hydromorphine at concentrations of 0.5 to 10 μg/dL, depending on the drug in question.

Other Common Narcotics

Tissue concentrations of propoxyphene, methadone, and meperidine were determined by the gas chromatographic methods of Wolen and Gruber [*12*].

A total of 10 855 medical examiner's cases were received from 1 Jan. 1971 to 30 June 1975. Acute narcotism accounted for 2.34% of these deaths. These cases were reviewed and the data tabulated and analyzed. The one-tailed *t* test of statistical significance was used where applicable.

Interpretation of the significance of toxicological data is made by the toxicologist at the request of the medical examiner. Review of qualitative and quantitative data regarding the substance, together with the history, physical evidence, and pathology of the case, will lead to one of the following six conclusions.

1. Findings indicate death caused by the agent.
2. Findings are compatible with death from the agent.
3. Findings are not indicative of death from the agent, but the agent is contributory with other findings.
4. Findings are compatible with some degree of impairment of cerebral functions.
5. Findings are within the therapeutic range of the drug.
6. Findings have no significance attached.

Results

Toxicological Findings

Six narcotics were involved in the 255 deaths directly attributable to acute narcotism and in the 172 deaths to which they contributed. The data are summarized in Table 1.

The most frequently encountered narcotic was heroin, designated in the cause of death (COD) as morphine-type alkaloid (MTA) because the method used for analysis identifies not heroin, but free and bound morphine. Since monoacetylmorphine, heroin, and related alkaloids were all metabolized to morphine and morphine glucuronide, any morphine-derived component detected is referred to as MTA. There are no cases in this study based on history or evidence that could be designated with confidence as due to morphine itself.

Of the deaths attributed to acute narcotism, MTA was determined to be the primary cause of death in 202 cases, representing 1.86% of all of the cases in the 4.5-year period.

Propoxyphene was the second most frequently identified narcotic in fatal narcotism, and

TABLE 1—*Summary of cases in which narcotics were the primary or contributing cause of death.*

Narcotic	Primary Cause	Contributing Cause	Total
MTA	202	57	259
Propoxyphene	45	41	86
Codeine	3	55	58
Methadone	3	12	15
Meperidine	2	4	6
Hydromorphone	0	3	3
Total	255	172	427

it was the primary COD in 45 cases. The other narcotics, with the exception of hydromorphine, were designated as the primary COD in only eight cases. There were 172 cases in which more than one of the six narcotics, in various combinations, were designated as the COD. The metabolic product of heroin (expressed as MTA) was present either alone or in combination with other drugs in 259 (60.55%) of 427 such cases.

The data from 263 cases in which MTA was identified as the narcotic responsible for or contributing to death were analyzed for the presence or absence of other drugs and the relative contribution of each to the COD. In 80 cases (30.4%) MTA was the only drug identified. In 91 (34%) alcohol was present in concentrations of 0.1% or more, but no other drugs were present. In 61 cases (23.1%) alcohol was present in concentrations less than 0.1%, but no other drugs were detected. In 31 cases (11.7%) other drugs and MTA were presented in combination. These data clearly indicate that in fatal cases MTA is more frequently used with alcohol and other drugs than by itself.

A similar analysis was made for propoxyphene alone and in various combinations. Seventy-two cases were included. There were only 14 cases (19.4%) in which propoxyphene alone was identified. Alcohol was the only other substance in 23 cases (31.9%); other drugs were present in 17 cases (23.6%). Combinations of propoxyphene, alcohol, and other drugs accounted for 18 cases (25.0%).

The quantification of MTA in various body fluids is of some assistance in establishing the relative time of death following narcotic administration. Following intravenous administration, the concentration of MTA in the blood falls until it is barely identifiable at the end of 3 h. During this time the concentration in the bile rises, reaches a peak, and then declines; the concentration in the urine rises also, following a pattern that is similar to that of the bile but is delayed.

In 261 cases of fatal narcotism, the identification of MTA in blood, bile, and urine was analyzed. In all cases in which all three fluids were available, MTA was identified in at least one. MTA was identified in the blood alone (none detected in bile or urine) in only 5.7% of the cases. It was identified most frequently in the urine (67.0%). It was identified in all three fluids in only one third of the cases.

The ranges and mean values of MTA concentrations in the blood, bile, and urine were tabulated (see Table 2). The cases were grouped on the basis of the presence or absence of alcohol and other drugs. For blood concentration, median values were also determined. The various groups were compared statistically to determine whether the MTA concentration that resulted in death was affected by the presence of other drugs or alcohol or both. Because of large standard errors, any differences observed were frequently not statistically significant.

The highest MTA levels were found in the bile and the lowest in the blood. Where MTA alone was the COD, the mean blood level was 59 μg/dL and the median, 20 μg/dL. Mean blood concentrations of MTA in the other groups ranged from 10 to 88 μg/dL, but in no case did the mean differ significantly from that of the group where MTA alone caused death. Thus, the presence of alcohol or other drugs had no significant effect on the blood MTA levels found.

When compared to the MTA levels in the "MTA alone" group, MTA levels in bile and urine were significantly lower in those cases where death was due to a combination of MTA, alcohol, and other drugs. In addition, levels in bile were significantly less when greater than 0.1% blood alcohol was present. Also, MTA levels in urine were significantly less when the COD was primarily MTA but both alcohol and other drugs were present. The situation may reflect (1) the administration of a lower dose of MTA in cases where other drugs were also taken, (2) a more rapid death, or (3) a delayed death following the last narcotic dose in a chronic user.

The data indicate that mean bile and urine concentrations of MTA in fatal cases average 1 to 4 mg/dL, while concentrations in the blood are about 20 μg/dL.

TABLE 2—*MTA concentrations in blood, bile, and urine in fatal narcotism cases.*

Drugs	MTA Concentration in Body Fluids		
	Blood, μg/dL	Bile, mg/dL	Urine, mg/dL
MTA alone	$n = 44$	$n = 75$	$n = 56$
mean	59	4.15	1.83
median	20		
range	5-1200	0.1-92.2	0.5-9.4
With alcohol <0.1%	$n = 19$	$n = 29$	$n = 22$
mean	37	2.90	1.16
median	40		
range	9-90	0.03-17.8	0.1-10.4
With alcohol >0.1%	$n = 39$	$n = 48$	$n = 33$
mean	25	0.97[a]	1.01
median	20		
range	8-60	0.05-4.8	0.05-22.4
With other drugs (COD mainly MTA)	$n = 18$	$n = 20$	$n = 15$
mean	51	2.80	1.06
median	28		
range	5-190	0.08-8.2	0.1-3.2
With other drugs (COD combination)	$n = 10$	$n = 25$	$n = 25$
mean	22	2.41	1.18
median	20		
range	6-34	0.1-6.3	0.05-5.6
With alcohol <0.1% and other drugs (COD mainly MTA)	$n = 3$	$n = 6$	$n = 6$
mean	88	2.33	0.65[b]
range	15-210	0.48-7.5	0.3-2.0
With alcohol >0.1% and other drugs (COD mainly MTA)	$n = 3$	$n = 5$	$n = 4$
mean	71	1.55	0.74
range	27-150	0.35-4.7	0.1-1.6
With alcohol <0.1% and other drugs (COD combination)	$n = 7$	$n = 6$	$n = 5$
mean	26	0.59[b]	0.69[b]
median	20		
range	7-60	0.1-4.2	0.03-1.3
With alcohol >0.1% and other drugs (COD combination)	$n = 2$	$n = 7$	$n = 6$
mean	10	1.21[a]	0.24[b]
range	10-10	0.1-4.2	0.5-0.7

[a]Significance for $P = 0.05$.
[b]Significance for $P = 0.01$.

Anatomical Findings

In addition to the toxicological results, certain anatomical findings are considered to be important in determining the cause of death. These include recent needle marks, scarred veins, pulmonary edema, or other unusual pathology of the liver and lungs. Data regarding these findings are presented in Table 3. Pulmonary edema was found in about 90% of narcotism deaths; other lung pathology and liver pathology, such as fatty infiltration and hepatitis, were frequently observed. Scarred veins and recent needle marks, often substantiated by histological examination of skin sections, were reported in two thirds of the MTA deaths but only in about 5% of propoxyphene deaths.

TABLE 3—*Pathologic findings in fatal narcotism, % of total cases.*[a]

Pathologic Finding	Drug					
	MTA	Propoxyphene	Codeine	Methadone	Hydromorphine[b]	Meperidine[b]
Scarred veins	172/256 (67.2)	4/71 (5.6)	3/10 (30.0)	4/7 (57.1)	3/3 (100)	0/2 (0)
Recent needle marks	178/254 (70.1)	3/71 (4.2)	1/10 (10.0)	3/7 (42.9)	3/3 (100)	2/2 (100)
Pulmonary edema	227/251 (90.4)	65/70 (92.9)	9/10 (90.0)	7/7 (100)	3/3 (100)	2/2 (100)
			MICROSCOPIC PATHOLOGY			
Lung	125/225 (55.6)	25/67 (37.3)	2/9 (22.2)	4/7 (57.1)	1/2 (50.0)	0/2 (0)
Liver	162/226 (71.7)	44/68 (64.7)	5/8 (62.5)	4/7 (57.1)	1/2 (50.0)	1/2 (50.0)
Skin	117/230 (50.9)	3/67 (4.5)	2/8 (25.0)	1/7 (14.3)	3/3 (100)	0/2 (0)

[a] Results presented as positive cases/total cases (percent).
[b] $n < 5$ cases.

Investigative Findings

Evidence found at the scene of death was also considered in determining the probable cause of death. Such evidence included drugs, drug containers and paraphernalia, and an unusual location of the body. A history of past drug abuse was also considered. Data concerning these findings are summarized in Table 4. Unusual location of the body, which included public restrooms, apartments belonging to friends or strangers, hallways, stairways, closets, automobiles, and entrances to emergency hospitals, was probably a more reliable indication of MTA overdose than the other types of physical evidence at the scene, each of which can be removed before the official investigation begins. Unusual location of the body was mentioned in about 45% of deaths involving MTA but in only 26% of cases involving propoxyphene, reflecting the suicidal nature of the latter as opposed to the accidental nature of the former.

Drugs or drug paraphernalia were found in approximately one third of the MTA cases, and a history of abuse was elicited about as frequently. Where propoxyphene and codeine were involved, the drugs and prescriptions for them were found in more than 50% of the cases.

A decision as to the COD depends on skillful interpretation of the history, observations made at the scene of death, autopsy findings, and toxicological results. The three most consistent findings in the cases considered here were positive identification of the drug in the body (in 100% of the cases), the presence of pulmonary edema (90.4% of the cases), and microscopic liver changes (71.1% of the cases). Microscopic examination of the skin and tissue at the site of injection was less important in confirming recent narcotic administration, but scarred veins and recent needle marks were frequently present. Physical evidence suggesting narcotism at the scene of death was less frequently encountered.

Demographic Characteristics

The demographic features of age, sex, and race in MTA- and propoxyphene-related deaths are summarized in Table 5. The largest number of cases of acute narcotism from MTA were found among white males, but because of the racial distribution of the population in San Francisco, the highest rate (that is, persons dying from a specific cause per 1000 of a specific group) was in black males. The mean age at death from MTA ranged from 29 to 33 in the four population groups, with the majority of deaths occurring between the ages of 21 and 30. Only a small percentage of deaths from MTA occurred in the over-40 group. The extremes of age at death were 15 and 65. Interestingly, while Orientals compose almost 11% of the population, there were no deaths of individuals under 35 in that racial group. The mean age of six Orientals dying from MTA overdose was 57.8, and three of those were males over the age of 62. This finding suggests the habit of opium smoking rather than intravenous narcotism. While the mean age of white males and females dying from MTA poisoning was about 30 years, the mean age of those dying from propoxyphene intoxication was nearly a decade higher.

Two thirds of the propoxyphene deaths occurred in persons over 31 years of age. In contrast to deaths from MTA where the male/female ratio was 3.03, there was no difference between the white male and female rates for propoxyphene deaths. The number of blacks dying from propoxyphene was too small to permit a valid comparison.

Mode of Death

In this medicolegal jurisdiction the decision as to whether death is due to accident, suicide, homicide, or natural causes is usually made by the medical examiner, although it is decided by a coroner's jury in some cases. The majority of deaths occurring from MTA and methadone overdose were designated as accidental (Table 6). Death generally was

TABLE 4—*Physical evidence suggestive of acute narcotism, % of total cases.*

	Drug					
	MTA	Propoxyphene	Codeine	Methadone	Hydromorphone	Meperidine
Number of cases	245	73	10	6	3	2
History of abuse, %	30.2	8.2	20.0	50.0	66.7	0
Paraphernalia, %	37.4	2.7	0	0	100	50.0
Drugs, %	28.6	46.6	50.0	0	66.7	100
Unusual location, %	44.9	26.0	20.0	33.3	0	50.0
Prescriptions, %	6.5	52.1	60.0	0	33.3	50.0

TABLE 5—*Demographic factors in fatal MTA and propoxyphene poisoning.*

	MTA				Propoxyphene			
	White		Black		White		Black	
	Male	Female	Male	Female	Male	Female	Male	Female
Number of cases	130	37	55	26	35	30	5	2
Mean age	30	30	33	29	38	41	31	30
Age 10–20, %	11.1	22.9	5.3	20.0	5.7	6.7	20.0	...
Age 21–30, %	49.6	45.7	39.3	40.0	25.7	26.7	20.0	50
Age 31–40, %	26.7	17.1	33.9	16.0	34.3	20.0	40.0	50
Age > 40, %	12.6	14.3	21.4	24.0	34.3	46.7	20.0	...
Rate per 100 000	62.2	15.2	113.9	51.1	16.0	12.3	10.4	3.9

TABLE 6—*Accidents, suicides, and undetermined modes of death for selected cases of death from acute narcotism.*

Narcotic	Accidents	Suicides	Undetermined[a]
MTA	246	5	7
Propoxyphene	26	42	5
Codeine	4	5	1
Methadone	5	0	3
Meperidine	0	1	1
Hydromorphone	3	0	0

[a]Including suspicion of homicide.

designated as suicidal following the taking of propoxyphene, codeine, and meperidine. In some cases, a decision was not reached as to whether the death was unequivocally from suicidal, homicidal, or natural causes.

Significant concentrations of MTA were found in biologic fluids in 48 cases of violent deaths. The cause of death in these cases was generally a bullet wound, stab wound, or head injury. MTA was also present in 35 other cases where death was due primarily to diseases associated with the use of narcotics. These diseases included pneumonia, hepatitis, cirrhosis of the liver, septicemia, bacterial endocarditis, vasculitis, and multiple pulmonary infarcts.

Discussion

Clinical Toxicology

The clinical toxicology of propoxyphene has not been well established. While the acute toxic effects of this compound in animals are decreased with a narcotic antagonist, treatment of intoxications with these antagonists in humans is not reported as effective. Minimal lethal doses for humans range from 500 to 1000 mg. Concentrations of propoxyphene in the blood were reviewed for a series of fatal cases by Baselt et al [*13*]. Their data indicate concentrations of propoxyphene in the blood usually in excess of 4 mg/dL. There is some reason to believe that the metabolite norproxyphene, which has a considerably longer half-life than the parent drug, contributes to the toxic, though not the analgesic, effect [*14*].

Infusion of heroin in normal male former drug users at the rate of 10 mg/h produced signs and symptoms of acute narcotism that in two instances required reversal with nalorphine after 40 mg had administered [*15*]. It was estimated that, assuming the metabolites of heroin are formed at the same rate as those of morphine, a man should be able to tolerate an infusion rate of 6 mg/h over a prolonged period. The protective value of narcotic tolerance has been demonstrated in studies among addicts using heroin, addicts on methadone maintenance, and those who have undergone withdrawal and later returned to narcotic abuse.

Early studies by Way et al [*16*] on morphine metabolism and absorption indicated that the drug was rapidly metabolized in vivo and in vitro. Deacetylation of heroin to 6-monoacetylmorphine (MAM) and morphine occurred most rapidly and to the greatest extent in the liver. Eighty percent of a subcutaneous dose was absorbed within 15 min. After intravenous administration, there was rapid disappearance indicating a biological half-life of about 2.5 min. In animals, the disappearance of circulating heroin is accompanied by the rapid appearance of MAM in the brain, followed shortly by the appearance of morphine. Metabolites of heroin that penetrate the central nervous system of animals do so largely as MAM. During the peak effects of heroin, it is probable that both MAM and morphine are

eliciting pharmacological actions. MAM was not detected in any of the narcotic fatalities in this study.

The absorption and distribution of morphine in the body after parenteral administration of morphine or heroin have been studied by a number of investigators. Berkowitz et al [*17*] demonstrated that, following intravenous injection, 93% of the morphine disappeared from the serum within 5 min. The early serum levels (2 min) averaged 29 μg/dL in patients under 50 and 49 μg/dL in older patients. The decline in morphine serum levels paralleled the decline in morphine analgesia and coincided with the appearance of morphine glucuronide in the serum. Robinson and Williams [*18*] examined autopsy specimens from eight known or suspected addicts. Their analysis of tissues indicated that the morphine in the bile and liver usually exceeded free morphine in the kidney and that morphine may or may not be found in the blood. (Morphine occurs both "free" and "bound," the latter as protein-bound morphine in the tissues.) Concentrations of MTA in the bile as high as 33 mg/dL were observed. High levels of both free and bound morphine were found in the lung occasionally.

Garriott and Sturner [*19*] reported on morphine concentrations in the blood of 22 individuals who died of acute heroin overdose. On the basis of microscopic changes in the lung, they estimated the duration of survival following the last drug injection and grouped their subjects according to this estimated survival time. In the short survival group (less than 3 h), concentrations of free morphine in the blood ranged from 10 to 93 μg/dL. In the intermediate group (3- to 24-h survival), free morphine was found in the blood in concentrations of 3 to 10 μg/dL. In a single case, morphine was still detected in the blood (6 μg/dL) after three days' survival. They hypothesized that abrupt renal shutdown, in cases of circulatory shock accompanied by cardiorespiratory arrest, might prevent the drug from reaching the urinary bladder or bile. This may explain the 5.7% of our cases in which MTA was found only in blood. They also suggested that morphine might be found in the bile for at least several days after administration and that the high concentrations of morphine frequently found in bile after "narcotic overdose" were a result of previous injections and not related to the lethal episode. However, when survival time is prolonged, both bile and urine may show high concentrations of the drug. They did not routinely assay morphine in the liver or kidneys. However, in the few cases in which it was done, concentrations were usually greater than 100 μg/100 g. Other authors have concluded that the concentration in these organs is not a reliable indication of a fatal acute reaction to heroin.

The term "overdose" as indicated by Greene et al [*20*] has served to indicate the lack of understanding of the true mechanism of death in fatalities correctly related to narcotic abuse. We propose, based on our observations of these cases and on historical, pathological, and toxicological findings, that any of the following three morbid clinical states can lead to death:

(1) an abrupt fatality immediately following the administration of the drug with minimal response in the lung,

(2) a centrally induced respiratory depression, and

(3) an effect on the alveolar capillaries of the lungs resulting in pulmonary edema and fatality in subjects surviving the initial depression.

Because of the long residence time of morphine in the bile and urine, death may not be a result of acute narcotism although MTA is found in the bile and urine. It is clearly important to exclude other causes of death in such cases. Besides the findings of our study, other authors have presented data showing that ethyl alcohol and other drugs are frequently present in heroin fatalities. In the 22 cases examined by Garriott and Sturner [*19*], ethyl alcohol and other drugs were present in 27%. Other authors have indicated drug and alcohol involvement in up to 50% of acute heroin fatalities [*21,22*]. In the study by Greene et al [*20*], methadone was more frequently involved in fatalities than in our study. In 39 of their 109

acute opiate "overdose deaths," death was due to this drug alone and 21 deaths were attributed to the combination of heroin and methadone. In our study, methadone was infrequently encountered as a cause of death, either alone or in combination with other drugs. Quinine, which has been reported as a frequent accompaniment of injectable heroin on the East Coast, is rarely detected in San Francisco.

Demography

There were certain differences in the demography of subjects in the present study as compared to others reported in the literature. Part of the difference may result from the fact that this study included deaths from all narcotics whereas the majority of other studies have dealt solely with heroin-induced fatalities.

Froede and Stahl [*23*] examined the problem of fatal narcotism among military personnel, reviewing 174 cases from 1918 to 1970. These were retrieved from 1.3 million surgical autopsy cases in the files of the Armed Forces Institute of Pathology. Two thirds of the deaths in this study group occurred in the 18 to 25 age group. There was only one case over age 50. The incidence of fatal narcotism was reported as highest among the nonwhite races, but the rate based on racial distribution was not given. Male deaths greatly exceeded female deaths, but the data were drawn from the predominantly male military community. In this study, accidental overdose deaths accounted for only 29.3% of the cases and homicide by narcotics for 3.4%.

In the first statistical analysis of data contained in the clinical record of 1036 patients admitted to the U.S. Public Health Service Hospital in Lexington, KY, more than half of the patients were in the 20 to 29 age group, although no age group was excluded [*24*].

Richter et al [*25*] cited heroin addiction as a major public health hazard and leading cause of death in the 15 to 35 age group. More than 10% of adults admitted to the medical services of a large municipal hospital in Harlem were there for treatment of heroin addiction.

The Washington, DC, addicts studied by Greene et al [*20*] were principally young, black, inner-city males with a history of chronic opiate abuse. The mean age of those dying from heroin was 27.1 years and from other opiates, 24.7 years. In these addicts, morphine was found in 84% of those dying suddenly.

Summary and Conclusion

1. Six narcotics have been identified in 352 deaths from acute narcotism in San Francisco among 10 882 cases.

2. Heroin, identified as MTA, was responsible for the greatest number of these cases.

3. MTA is not often found alone in fatal narcotism. Other narcotics, abused drugs, or alcohol is present in nearly 70% of the cases.

4. Most deaths from MTA are accidental, while those from propoxyphene, the second most frequently encountered narcotic, are generally suicides.

5. Narcotics can be identified in the bile, blood, or urine in all cases of fatal narcotism.

6. Concentrations of MTA are highest in the bile and urine, ranging from 1 to 4 mg/dL in most cases.

7. Blood concentrations of MTA range from 5 to 1200 μg/dL with a mean value of 59 and a median of 20 μg/dL.

8. Mean blood levels of MTA are not significantly changed in the presence of alcohol or other drugs.

9. A number of factors must be considered in arriving at the clinical diagnosis of death from acute narcotism. In our experience, toxicological analyses are more meaningful than pathologic examination, although the diagnosis is partially one of exclusion of other causes.

10. Acute narcotism from methadone occurs relatively infrequently, and this drug is not commonly found in persons dying from acute heroin intoxication in San Francisco.

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