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Toxicological Findings in Victims of Traumatic Deaths

The interpretation of the results of toxicological analyses in cases involving acute poisoning or narcotic-related deaths has been the subject of numerous excellent reviews [*1-10*].

Toxicological findings in cases of violent death, although not usually of primary relevance to the determination of the cause of death, have nevertheless received special attention owing to their importance in elucidating the social and psychological circumstances of such deaths. It is here that the effect on human behavior of the interactions of alcohol, tranquilizers, and narcotics plays a vital role.

This paper presents the toxicological findings in specimens obtained from victims dying under established or suspected violent circumstances in New York City during the last two years.

Additional information on the concentration and distribution of drugs in tissue was collected in 1974 and is presented in this paper as an attempt to establish for forensic specimens the concentration of drugs in tissues commonly associated with routine street and illicit usage. These would appear to be as important as are therapeutic concentrations for the purpose of delineating appropriate base values to correctly interpret the results of toxicological analyses in those cases where the presence of drugs is deemed to be the primary causative factor in the death rather than a secondary finding. Certain of our findings concerning the incidence of drugs in violent death were compared with those obtained from recent papers on narcotic abuse among homicide victims in Detroit [*11*].

Material and Methods

The sample selection was limited to the material submitted to the toxicological laboratory at the Medical Examiner's Office of New York City, which received 5913 and 5669 samples in 1973 and 1974, respectively. Toxicological analysis was requested in 3099 cases of traumatic death in 1973 (52.4% of total cases) and 2938 cases in 1974 (51.7% of total).

Because the requisition for such toxicological analysis depends on the criteria of the pathologist, our data cannot be taken to represent the total of all traumatic death in New York City. Most of the data contained herein are not expressed as absolute values or as frequencies of total toxicological findings but instead represent percentage values based on the total number of an indicated type of trauma category.[2] It should be noted

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[2]In 1974 the Toxicological Laboratories obtained analytic data on the following numbers of traumatic death categories: fire related—165; asphyxia by hanging, suffocation with plastic bag, physical obstruction—80; fall from height—475; "floaters"—131; vehicular accidents, drivers—171, passengers—84, pedestrians—254; shooting, stabbing, strangulation, beating—1579.

that the cases used in this study were selected from among those involving circumstances of apparent traumatic death, but without knowledge of the final disposition by the pathologist as to the cause of death. A somewhat more precise but less general survey in 1000 Medical Examiner's cases was performed by the joint efforts of New York University and Columbia University [*12*].

To compare the toxicological findings relating to narcotic drugs and alcohol in 207 victims of homicide in Detroit in 1973 [*11*] with a similar type population in New York City, we selected 689 cases involving death under circumstances of shooting, stabbing, strangulation, and beating (the most common forms of homicide). Cases with indications of suicide, with the ages of the deceased above 35 years, or with incomplete information as to age, race, and sex were not included.

The method of analysis employed in our laboratories is as follows for the different categories of drugs: alcohols—gas chromatography [*13*]; carbon monoxide (CO)—spectrophotometry [*14*] or diffusion into $PdCl_2$ [*15*] with concurrent determination of total hemoglobin through iron content; acid drugs—ultraviolet (UV) spectrophotometry [*16*], thin-layer chromatography (TLC) [*17-19*] and gas liquid chromatography [*20*]; basic drugs—enzyme immunoassay (EMIT®), radioimmunoassay (RIA), and fluorometry [*21*], UV spectrophotometry [*22,23*], gas chromatography [*24*], and TLC [*17-19,25*].

In some cases a complete examination could not be performed owing to the absence of one or more specimens among those regularly sent to the laboratory: blood, bile, brain, urine, or liver.

Results and Discussion

Incidence of Drugs in Traumatic Death

The toxicological findings in specimens obtained from cadavers of persons who died under various circumstances all suggestive of traumatic death are presented in Tables 1-4. Carbon monoxide data are reported in Table 1 only; however, toxicological analysis for CO is performed routinely on victims of vehicular accidents owing to the influence it may have on driving performance [*26,27*]. In Tables 1 to 4 the following designations are given to the indicated terms: narcotics comprises the most common narcotic drugs and

TABLE 1—*Toxicological findings in deaths involved in fire hazards,*[a] %.

Toxicological Results	1973	1974
Negative	21.80	20.99
Ethyl alcohol	...	8.64
Carbon monoxide saturation between 0 and 9%	24.80	37.03
Carbon monoxide saturation between 10 and 39%	32.50	24.07
Carbon monoxide saturation greater than 40%	42.70	38.90
Narcotics	1.80	1.23
Hypnotics	0.60	1.85
Analgesics	...	1.85
Tranquilizers	0.60	2.47

[a] Includes victims of combustion gases poisoning, extensive burns, traumatic injury by falling objects, fall from height, explosion, and electrocution effects which may act as a primary or secondary cause of death. Some few cases are of people who died before the fire hazard could not be excluded.

their metabolites; hypnotics refers to barbiturate derivatives and methaqualone; analgesics include salicylic acid derivatives, phenacetin, and other widely used pain relievers such as propoxyphene and lidocaine; tranquilizers include benzodiazepines, phenothiazine derivatives, and such antidepressants as amitriptyline and imipramine.

Toxicological findings on decedents from fire-related deaths are presented in Table 1, with data obtained from the years 1973 and 1974. Carbon monoxide poisoning (blood saturation above 40%) was, as expected, a very frequent explanation for the occurrence of death, but in most of the cases other physical injuries were the cause of death. In these cases the percentage of saturation of carbon monoxide in blood may be considered a contributing factor to death or it may only reflect the relative hazard potential of CO that existed in the circumstances of the fire. A very low or negative level of CO may indicate that death occurred prior to or at the immediate onset of the fire; it may also point to the possible involvement of other combustion gases in the cause of death. The lower incidence of toxicologically significant CO findings (saturation greater than 10%) for the year 1974 as compared to 1973 may be due to better control techniques, such as the use of other absolute analytical methods [*15*] to confirm both extremely high and threshold values obtained by a simpler, direct method [*14*].

The percentage of cases with completely negative findings has remained similar for the two-year period, indicating the importance of seeking alternate observations at autopsy to elucidate the cause of death.

With reference to Table 2, the first group comprises cases involving deaths by asphyxia due to hanging, use of plastic bags, or obstruction from vomitus or bolus of food. Group 2 involves cases of victims of traumatic injury resulting from such circumstances as a fall onto the tracks of an oncoming train or falls from windows, bridges, staircases, or elevator shafts as well as from injury due to the impact of falling objects and from other work accidents. Group 3 involves cases of victims found floating in water (river, sea, bathtub) with no indication of any other traumatic cause of death other than drowning.

Toxicological findings for the circumstances given in Table 2 merit special consideration. The relative frequency of ethyl alcohol as a single toxicological finding is approximately 20% in Groups 1 and 2 for the two-year period. Actually, the overall incidence of ethyl alcohol is greater than that given in Table 2, because ethanol is frequently found with the other drug classes in most of the cases reported as involving narcotics, hypnotics, analgesics, and tranquilizers. The abnormally high percentage of single positive alcohol findings in drowning deaths may be the result of the anaerobic ethanol-producing decomposition processes occurring in the blood and tissues of submerged victims that are found in advanced putrefied conditions [*28*].

TABLE 2—*Deaths by asphyxia and traumatic injury by moving train, fall, and work accidents, percentage of occurrence.*

Toxicological Results	Asphyxia by Hanging, Plastic Bag, or Physical Obstruction		Struck by Train, Fall, or Work Accident		Asphyxia by Drowning	
	1973	1974	1973	1974	1973	1974
Alcohols	20.31	22.78[a]	19.16	21.01[b]	43.0	36.64[c]
Narcotics	3.12	2.53	2.95	6.81	2.32	6.87
Hypnotics	1.56	16.46	3.18	4.47	3.48	4.58
Analgesics	...	2.53	2.90	7.45	2.32	3.05
Tranquilizers	29.68	16.46	8.50	14.25	12.20	7.63
Other	...	2.53	1.17	0.85	5.22	...

Total alcohol (alone and in combination): [a]29.11%; [b]29.31%; [c]50.38%.

By adding the percentage findings for cases involving alcohol in conjunction with hypnotics and tranquilizers, the following figures are obtained: 31–33% in Group 1 (death by asphyxia); 12–19% in Group 2; below 10% in Group 3. The higher values for Group 1 are assumed to reflect the use or abuse of relative-hypnotic drugs by neurotic people who commit suicide by hanging or by asphyxia using plastic bags.

The incidence of narcotic finding is low for the circumstances described in Tables 1 and 2, but attention is called to the higher values observed for Groups 2 and 3 (1974) in Table 2. This would appear to indicate that narcotics may have had an increasingly significant role in these groups.

Table 3 presents the percentage of positive toxicological findings for the three classes of victims of motor vehicle accidents. It should be emphasized that because meperidine and lidocaine, both of which are included in the analgesic category, are frequently administered during hospital therapy, the observed presence of these compounds should be checked against hospital records in these cases where victims of vehicular accidents die in the hospital. The incidence of ethanol alone for pedestrian victims is found to be 29.3% and 19.5% for the years 1973 and 1974 respectively, which values are lower than the reported incidences of 54.5% in North Carolina [*29*], 44% in Puerto Rico [*30*], and 46% in Cracow, Poland [*31*]. Alcohol with other drugs was found in 4.38% of the cases, which figure is consistent with that of 6.1% observed in pedestrian victims in North Carolina [*29*].

TABLE 3—*Toxicological findings in vehicle accidents, %.*

	Pedestrian		Driver		Passenger	
Results	1973	1974	1973	1974	1973	1974
Negative	64.50	61.73	54.20	39.40	63.49	52.39
Alcohol	29.31	19.53[a]	36.50	34.70[a]	28.54	38.09[a]
Narcotics	1.29	1.60	3.40	2.95	3.16	1.19
Hypnotics	0.60	3.19	0.56	4.71	1.58	3.57
Analgesics	1.60	7.57	3.40[b]	13.53	...[b]	...
Tranquilizers	0.93	5.58	...[b]	4.71	...[b]	3.57
Other	0.90	0.80	1.12	...	1.58	1.19

[a] Total alcohol (alone and in combination): pedestrian, 23.91%; driver, 42.29%; and passenger, 42.85%.

[b] No complete blood screening.

The percentage of driver victims with only ethyl alcohol was found to be 36.50 and 34.70, both of which are well below the observed incidences found in North Carolina (46.3%) [*29*] and Orange County, Calif. (45.5%) [*32*]. Ethanol alone or in combination with other drugs was present in 42.3% of cases in 1974. This value clearly indicates the major role of alcohol and drugs in vehicle accidents, but is still below what was reported for North Carolina (49.2%) [*21*] and Orange County (55.5%) [*32*].

The low percentage of occurrence of narcotics suggests they are not a major factor in vehicular accidents, but there is a relevant percentage of hypnotic-tranquilizers found in driver victims for the year 1974. This 4.7% incidence for hypnotics and tranquilizers is lower than that observed in Orange County (38%) [*32*], and is greater than the incidence of these same drugs in the population of pedestrian and passenger victims of vehicle accidents in New York City. That these drugs may play a role in vehicular accidents [*32,33*] was anticipated from their known effects of inducing both dizziness and slowing reflex action [*34,35*] which affect driver performance [*36–40*].

TABLE 4—*Traumatic deaths (shooting, stabbing, strangling, beating).*

Toxicological Results	1973, %	1974, %
Negative	41.40	37.01
Alcohol	35.86	34.21[a]
Narcotics	18.03	16.02
Hypnotics	2.21	2.10
Analgesics and lidocaine	2.08[b]	5.87
Tranquilizers	0.92[b]	4.02
Other	1.43	0.77

[a] Total alcohol (alone and in combination): 45.89%.
[b] No complete blood screening for these drugs.

Table 4 contains the percentage distribution of toxicological findings in traumatic deaths involving shooting, stabbing, strangulation, and beating. Most of these involve homicide, but suicide by shooting is also included in this category.

Approximately one in two victims of violent death is found to have ingested ethyl alcohol, narcotics, or both. The frequency of ethanol as a single finding ranges between 34 and 36%, but this figure becomes 45.9% when victims who died under the influence of alcohol combined with narcotics and other drugs are included.

This figure correlates well with the 52% incidence of ethanol in victims of homicide in Wayne County, Detroit [*11*] and agrees with the preliminary results of the current detailed survey of Haberman and Baden [*12*] for the victims of traumatic death.

The high frequency of narcotics (16–18%) found for victims as shown in Table 4 strongly illustrates that in conjunction with alcohol these drugs play an important role in violent crime, thereby confirming previous statistics which show an incidence of 13.1% of addicts among victims of homicide in New York City (1969–1971) [*41*].

The correlation between crime and narcotism was further compared in detail with data recently published [*11*]. Of the total cases of traumatic death by stabbing, shooting, strangling, and beating reported in New York City in 1974, 689 were selected for comparison with the toxicological findings on victims of homicide in Detroit. The combined results are presented in Tables 5–7.

Table 5 shows that the population of victims in both Detroit and New York consists mainly of black males; in New York there is also a large population of white male vic-

TABLE 5—*Comparison of toxicological findings in victims of violent death (shooting, stabbing, strangulation, and beatings).*

	Population Selected up to 35 Years of Age		Deaths Under Influence			
			Ethanol, %		Narcotic Drugs, %	
Groups	Detroit[a]	New York City	Detroit[a]	New York City	Detroit[b]	New York City
Black male	153	285	30.7	34.7	36.0	33.7
Black female	32	67	15.6	31.3	18.8	13.4
White male	17	125	35.3	39.40	17.6	19.2
White female	5	22	60.0	22.9	0	14.3
Total	207	689	29.5	35.85	30.9	24.38

[a] Ref *11*.
[b] Ref *11*. Number of cases with needle tracts (75%) had evidence of a drug indicative of narcotic usage.

tims as a result of the incidence of crime in areas populated by white minority groups. The incidence of ethanol in total victim population is greater in New York City than in Detroit. The incidence of narcotics is also greater for the white population in New York City. This observation can be accurately inferred for blacks, despite the figures shown in the Table 5, on the basis that had these figures been calculated to include those cases with only needle track evidence as the indication of narcotism, the percentages for black males and females in New York would have been significantly increased.

The total number of victims of the indicated violent death in whom narcotics were found is 74 and 168 for Detroit and New York City, respectively, as shown in Table 6. Also shown is the frequency of the combination of narcotics and ethyl alcohol, which is greater in the New York City area (8.42%) than in Detroit (6.28%).

TABLE 6—*Correlation of toxicological findings in victims of violent death by shooting, stabbing, beating, and strangulation.*

	207 Cases of Homicide up to 35 Years of Age, Detroit (1973)[a]		689 Cases of Violent Death up to 35 Years of Age, New York City (1974)	
Toxicological Results	Narcotics	Narcotics Plus Ethanol	Narcotics	Narcotics Plus Ethanol
Heroin	56	7	35	12
Heroin and methadone	6	1	25	3
Methadone and others	2	1	77	32
Quinine	10	4	31	11
Total	74	13	168	58

[a] Ref *11*.

Another noteworthy point of Table 6 relevant to New York City is that methadone is the most common narcotic found (14.8%); second is heroin or quinine, or both (13.2% of the total victim population). In Detroit, by contrast, morphine and quinine alone or in conjunction with other drugs are the most common finding (34.8%), and methadone occurs with much lower frequency (3.9%). The data in Table 7 confirms that in both cities black males comprise the largest single group of victims, with white males (mainly of Hispanic origin) the second largest group in New York City.

TABLE 7—*Comparison of race and sex of victims of shooting, stabbing, strangling, or beating who died under the influence of drugs (percentage in total population).*

	Distribution of Deceased Under the Influence of Narcotics	
Population up to 35 Years Old	Detroit 1973[a]	New York City 1974
Black male	73.9	57.2
Black female	15.5	5.4
White male	8.2	34.4
White female	2.4	3.0

[a] Ref *11*.

Tissue Concentration of Drugs

The importance of quantitation in toxicological analysis in cases related to circumstances of traumatic death is well illustrated by the procedure employed for the determination of CO saturation in the blood of fire victims. These analyses are given high priority to complement and complete the autopsy. The specific degree of saturation of CO in blood may transform what had been thought to be a traumatic death into a death attributable to smoke inhalation alone or in conjunction with traumatic injury. The probability of death prior to the fire may also be indicated in certain instances. The judgment and criteria of the pathologist are of paramount importance in the final assessment of all such cases.

The presence of drugs in viscera in cases where only a traumatic circumstance had been suspected has resulted in unexpected revelations for both pathologist and toxicologists.

The examination of liver tissue extracts for drugs with UV spectrophotometry [*22,23*] allows a semiquantitative control of all samples for the purpose of differentiating therapeutic from toxic concentrations as a preliminary screening for possible further analysis. In the last three months of 1974 eleven cases of traumatic death initially thought to involve only the routine toxicological screening analysis for such cases now gave evidence of the involvement of abnormal concentrations of tranquilizers. They were consequently submitted to more complete analysis, the results of which are presented in Table 8. The

TABLE 8—*Toxicological findings in traumatic deaths, abnormal levels in liver.*

Drug	Samples, no.	Observed Levels, Concentration in mg/100 ml		Reference Levels, mg/100 ml[a]
		Median	SD	
Amitriptyline	4	9.9	8.8	5
Phenothiazine derivative	4	8.0	8.6	5
Propoxyphene	3	5.2	3.8	3

[a] Ref *1*.

median concentrations of amitriptyline, chlorpromazine, and propoxyphene are above those generally accepted as toxic, with a large range of values being observed. This clearly indicates the possibility of association of poisoning with traumatic injury as a cause of death. The barbiturate content in liver in cases of suicide by suffocation using a plastic bag to cover the head is at variance with that found for typical acute barbiturate poisoning (Table 9). In the past two years six cases have been observed involving this type of asphyxia in combination with high liver levels of barbiturates (that may also be associated with ethanol consumption).

The systematic examination in 1974 of all submitted blood samples has provided us with 471 analytical results relating to observed levels of various drugs (Table 10). These levels probably are not directly relevant as regards the assignation of a traumatic cause of death by the pathologist, but they do provide an excellent base for the interpretation of toxicological findings when a drug overdose is suspected.

The relatively high levels of short- and intermediate-acting barbiturates and also of lidocaine (Table 10) coupled with the large standard deviations suggest some degree of overtreatment by hospitals of the victims of potentially lethal traumatic injury. Levels of diphenylhydantoin, diazepam, and meperidine and slow-acting barbiturates are more consistent with therapeutical levels.

TABLE 9—*Liver concentration of barbiturates, in mg/100 ml, under different circumstances.*

Barbiturate	Selected Cases of Suicide with Plastic Bag and Barbiturate Ingestion		Barbiturate Content in Acute Poisoning Under Normal Environmental Conditions		
	Concentration, mg/100 ml	Other Findings	Cases, no.	Median Concentration, mg/100 ml	
Pentobarbital	11.7				
	2.0	alcohol 0.18 g/100 ml	18	16.9	11.8
Amobarbital and Secobarbital	2.8	alcohol 0.14 g/100 ml	17	14.2	13.6
	3.1				
	16.7				
Secobarbital	29.5	chlordiazepoxide	22	8.8	6.1

TABLE 10—*Toxicological findings in traumatic deaths.*

Drugs	Observed Values				Reference Levels, mg/100 ml[a]		
	No.	Sample	Concentration, mg/100 ml		Blood, Therapeutic	Blood, Toxicological	Liver, Toxicological
			Median	SD			
Barbiturates, rapid and intermediate	20	blood	1.57	1.26	0.05	1.0	...
	3	brain	2.42	1.04			
	11	liver	2.77	3.90			
Barbiturates, slow acting	25	blood	1.64	1.04	2.3	5.0	...
	5	brain	1.90	1.34			
	11	liver	2.27	1.10			
Diazepam	20	blood	0.33	0.26	0.20	...	...
Diphenylhydantion	8	blood	0.36	0.32	1.00	7.0	10.0
Lidocaine	118	blood	0.70	1.59	...	...	...
Meperidine	113	blood	0.12	0.14	0.10	1.0	...
Methadone	83	blood	0.13	0.14	0.086–0.045 [*42*]	0.3	1.0
	18	brain	0.13	0.10	0.070–0.040 [*43*]		
	43	liver	0.53	0.42	0.101–0.055 [*44*]		

[a] Ref *1*.

Sufficient data for methadone are presented in Table 10 to define a median "social level" of this drug as 0.13 ± 0.14 mg/100 ml in blood and brain. The variation of methadone concentrations may be taken to be the range from 0.0 to 0.27 mg/100 ml, which is well above the therapeutic peak level expected in addicts after ingestion of methadone (Table 10).

The blood methadone concentration as reported by different workers for cases classified as overdoses shows a wide variation: a range of 0.02 to 0.30 mg/100 ml was found in 11 cases studied by Robinson and Williams [*4*], Segal and Catherman [*5*] found ranges of

0.0 to 0.25 mg/100 ml in 11 cases after methadone ingestion, 0.0 to 0.1 mg/100 ml was reported in 21 cases after methadone ingestion, and 0.0 to 0.12 mg/100 ml in 10 cases where other causes of death were involved [5].

Irey and Froede [6] have stated the range of toxic levels of methadone in blood to be 0.02 to 0.45 mg/100 ml. Garriott [9] describes six cases of methadone death within the range of 0.0 to 0.13 mg/100 ml.

Most of these overdose levels in blood are in the same range as the social level of methadone, that is, 0.0 to 0.27 mg/100 ml, suggesting that the so-called overdose of methadone is better explained as a physiological over-utilization of normal doses ingested by addicts, which quantities provide the same blood level of methadone in both traumatic and narcotic deaths.

We hope that these findings concerning the distribution of methadone in different tissues in cases of traumatic death will provide a firm reference point for an understanding of the lethal syndrome of narcotism.

Summary

The toxicological findings from 6037 analyses of viscera obtained from victims of traumatic death are used to correlate the relative incidence of carbon monoxide, ethyl alcohol, narcotics, hypnotics, analgesics, and tranquilizers-antidepressants in deaths occurring under the following circumstances: fire related; asphyxia by hanging, by use of plastic bags, from physical obstruction of trachea, and by drowning; traumatic injury from impact of moving train, fall from height, and occupational accident; traumatic injury to pedestrian, driver, and passenger from vehicular accidents; and from violent death by shooting, stabbing, strangulation, and beating. The influence of alcohol, narcotic drugs, and tranquilizers on carbon monoxide can be seen in some of these traumatic deaths.

Ethanol alone and in combination with other drugs was present in 42.3% and 19.5% of driver and pedestrian victims, respectively, of vehicular accidents in the year 1974. Comparative analysis is presented for the toxicological data obtained on victims of homicide (shooting, stabbing, strangulation, and beating) in New York City and similar data reported for victims of homicide in Detroit. In New York City 45.9% of such victims died while under the influence of alcohol or narcotic drugs, or both, with methadone predominating in the latter category.

Tissue concentrations of drugs found in victims of traumatic death are presented. Diphenylhydantoin, diazepam, meperidine, and slow-acting barbiturates were found in normal therapeutic levels. Higher concentrations of amitriptyline, chlorpromazine, propoxyphene, short-acting barbiturates, and methadone were observed.

The concentration of methadone in blood and brain (0.13 $\pm$ 0.14 mg/100 ml) and in liver (0.53 $\pm$ 0.42 mg/100 ml) in cases of traumatic death are not different from those observed in deaths classified as due to methadone overdose.

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References

[1] McBay, A. J., *Clinical Chemistry,* Vol. 19, No. 4, 1973, pp. 361-365.
[2] Ruedy, J., *Canadian Medical Association Journal,* Vol. 109, No. 7, 1973, pp. 603-606.
[3] Winek, C. L. and Wecht, C., *Legal Medicine Annual,* Appleton, Century & Crofts, Inc., New York, 1973.

[4] Robinson, A. E. and Williams, F. M., *Journal of Pharmacy and Pharmacology,* Vol. 25, No. 5, 1971, p. 353.
[5] Segal, R. S. and Catherman, R. L., *Journal of Forensic Sciences,* Vol. 19, No. 4, 1974, p. 64.
[6] Irey, N. S. and Froede, R. C., *American Journal of Clinical Pathology,* Vol. 61, No. 6, June 1974, pp. 778–784.
[7] Arieff, A. I. and Friedman, E. A., *The American Journal of the Medical Sciences,* Vol. 266, No. 6, 1973, pp. 405–426.
[8] Coumbis, R. J. and Kaul, B., *Journal of Forensic Sciences,* Vol. 19, No. 2, 1974, p. 307.
[9] Garriott, J. C., Sturner, W. Q., and Mason, M. F., *Clinical Toxicology,* Vol. 6, No. 2, 1973, pp. 163–173.
[10] Greene, M. H., Luke, J. L., and DuPont, R. L., *Journal of Forensic Sciences,* Vol. 19, No. 3, July 1974, p. 575.
[11] Monforte, J. R. and Spitz, W. V., *Journal of Forensic Sciences,* Vol. 20, No. 1, 1975, p. 186.
[12] Haberman, P. W. and Baden, M. M., *The International Journal of the Addictions,* Vol. 9, No. 6, 1974, p. 771.
[13] Machata, G., *Fortschrifte der Chemischen, Forschung,* Vol. 6, No. 4, Springer-Verlag, Berlin-Heidelberg, New York, 1966.
[14] Freireich, A. W. and Landan, D., *Journal of Forensic Sciences,* Vol. 16, No. 1, 1971, p. 112.
[15] Williams, L. and Sunshine, I., *Manual of Analytical Toxicology,* Chemical Rubber Press, Cleveland, 1971, p. 63.
[16] Sunshine, I. and Bath, R., *Sunshine Manual of Analytical Toxicology,* Chemical Rubber Press, Cleveland, 1971, p. 36.
[17] Mulé, S. J., Bastos, M. L., Jukofsky, D., and Saffer, E., *Journal of Chromatography,* Vol. 63, 1971, pp. 289–301.
[18] Bastos, M. L., Jukofsy, D., Saffer, E., Chendekel, M., and Mulé, S. J., *Journal of Chromatography,* Vol. 71, 1972, p. 549.
[19] Baden, M., Valanju, N., Verma, S., and Valanju, S., *American Journal of Clinical Pathology,* Vol. 57, No. 1, 1972, pp. 43–51.
[20] Mulé, S. J., *Journal of Chromatography,* Vol. 55, 1971, p. 255.
[21] Bastos, M. L., Mulé, S. J., and Stolman, A., *Progress in Chemical Toxicology,* Vol. 5, Academic Press, New York, 1974.
[22] Goldbaum, L. R. and Domanski, T. J., *Journal of Forensic Sciences,* Vol. 11, No. 2, 1966, p. 233.
[23] McBay, A. J., Turk, R. F., Corbett, B. W., and Hudson, P., *Journal of Forensic Sciences,* Vol. 19, No. 1, 1973, p. 81.
[24] Inturrisi, C. E. and Kaiko, R. F., *Journal of Chromatography,* Vol. 65, 1972, pp. 361–369.
[25] Davidow, B., Petri, N. L., and Quame, B., *The American Journal of Clinical Pathology,* Vol. 50, No. 6, 1968, pp. 714–719.
[26] MacFarland, R. A., *Archives of Environmental Health,* Vol. 27, No. 12, pp. 355–359.
[27] Wright, G., Randell, P., and Shephard, R. J., *Archives of Environmental Health,* Vol. 27, No. 12, 1973, pp. 349–354.
[28] Gonzales, T. A., Vance, M., Helpern, M., and Umberger, C. J., *Legal Medicine, Pathology and Toxicology,* 2nd ed., Appleton, Century & Crofts, Inc., New York, 1954.
[29] Turk, R. F., McBay, A. J., and Hudson, P., *Journal of Forensic Sciences,* Vol. 19, No. 1, 1974, p. 90.
[30] Kaye, S., *Boletin de la Asociacion Medica de Puerto Rico,* Vol. 61, 1969, p. 7244.
[31] Grochowska, Z., *Polskie Archiwum Medycyny Sadonej i Kryminologii,* Vol. 22, No. 1, 1971, p. 19.
[32] White, J. M. and Graves, M. H., *Journal of Chromatographic Sciences,* Vol. 12, 1974, p. 219.
[33] Finkle, B. S., Biasotti, A. A., Crim, M., and Bradford, L. W., *Journal of Forensic Sciences,* Vol. 13, No. 2, 1968, p. 236.
[34] Tansella, M., Zimmermann, A., Tansella, C., and Lader, M., *Psychopharmacologia* (Berlin), Vol. 38, No. 1, 1974, pp. 81–90.
[35] Furney, R. B., Hughes, F. W., *Combined Effects of Alcohol and Other Drugs,* Charles C Thomas, Springfield, Ill., 1968.
[36] Milner, G., *Drugs and Driving,* Adis, New York, 1972.
[37] Betts, T. A., Clayton, A. B., and Mackay, G. M., *British Medical Journal,* Vol. 4, No. 46, 1972, p. 580.
[38] Milner, G. and Landauer, A. A., *Blutalkohol,* Vol. 10, 1973, p. 247.
[39] Linnoila, M. and Mattila, M. J., *European Journal of Clinical Pharmacology,* Vol. 5, No. 3, 1973, p. 186.
[40] Morland, J., Setekeiv, J., Hoffner, J. F. W., Stromsaether, C. E., Danielsen, A., and Wethe, G. H., *Acta Pharmacologica et Toxicologica,* Vol. 34, No. 1, 1974, p. 5.
[41] Baden, M. M., *Human Pathology,* Vol. 3, No. 1, 1972, pp. 91–95.

[42] Inturrisi, C. and Verebely, K., *Clinical Pharmacology and Therapeutics,* Vol. 13, No. 5, Part 1, 1972, p. 633.
[43] Kreek, M. J., *New York State Journal of Medicine,* Vol. 73, No. 23, 1973, pp. 2773–2777.
[44] Sullivan, H. R. and Blake, D. A., *Chemical Pathology and Pharmacology,* Vol. 3, No. 3, 1972, p. 467.

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