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Acute Ethanol Poisoning: A Two-Year Study of Deaths in North Carolina

A significantly high blood level of ethanol is one of the most common factors among deaths investigated by medical examiners. There are few, if any, drugs that are as extensively used. Much information is available on forensic problems that involve the use and abuse of ethanol and on deaths contributed to, caused, or precipitated by ethanol [*1–4*].

Unfortunately, the cause and manner of deaths involving acute ethanol poisoning are often overlooked or misinterpreted by the forensic scientist for a variety of reasons [*5*] including incomplete understanding of the significance and variability of blood ethanol levels, socioeconomic pressures, and a reticence of governmental agencies and private concerns to recognize this form of drug abuse properly.

Acute fatal intoxication by excessive ethanol ingestion is a distinct, recognizable entity. This paper reviews a series of 502 medicolegal cases in which blood ethanol levels exceeded 300 mg/100 ml in order to reevaluate the criteria used in determining the cause of death. The data point out some of the salient, recurrent, pathologic, toxicologic, and epidemiologic features of this syndrome. The purpose of this study is to describe the range, coincidental findings, and interpretation of the cause and manner of death with particular emphasis on the relative importance of several factors.

Material and Methods

The case material is composed of all deaths occurring during a 24-month period within the jurisdiction of the medical examiner system of North Carolina in which there were blood ethanol levels of 300 mg/100 ml or more. Medical examiner case reports death certificates, toxicologic reports, and autopsy reports available on these 502 fatalities were correlated. Those deaths certified as caused by methyl and isopropyl alcohol poisoning and those caused by poisoning by glycols were excluded from the study. The data extracted from the material included age, race, sex, occupation, place and time of death, cause and manner of death, blood ethanol levels, presence of any other toxic agents, available medical history, antecedent disease processes, and situations or circumstances at time of death.

The cases were first divided into three categories depending on their blood ethanol levels: those with 300 to 349 mg/100 ml, those with 350 to 399 mg/100 ml, those with 400 mg/100 ml or greater.

During the investigation 24 cases of deaths resulting from well-documented natural

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causes having blood ethanol levels greater than 300 mg/100 ml were found. These were not included in the 502 cases composing the main body of the study although it is recognized that either acute or chronic alcoholism may be a contributing factor. Of these 24 cases, 16 had blood ethanol levels of 300 to 349 mg/100 ml, 8 had levels of 350 to 399 mg/100 ml, and none was found to have a level of 400 mg/100 ml or over. The causes of death in this group are listed in Table 1.

TABLE 1—*Cause of death in the group certified as natural death.*

Cause	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400+ mg/100 ml, *n*
Arteriosclerotic heart disease	3	4	...
Cerebrovascular accident	3	1	...
Pancreatitis	...	1	...
Endocarditis	...	1	...
Tuberculosis	...	1	...
Asthma	1	...	...
Diabetes	2	...	...
Bleeding varices	1	...	...
Total	16	8	...

The cases of unnatural death were further subdivided into three major groups: those in which death occurred from obvious trauma or violence; those which had a combination of a high ethanol factor and additional contributing or related abnormalities which may have been a cause of death; and those cases of acute ethanol poisoning. The specific causes of death in the trauma group and the contributing or related abnormalities found in the mixed group are listed in Tables 2 and 3, respectively. The criteria for those cases of acute ethanol poisoning were based on ethanol levels above 300 mg/100 ml, noncontributory medical history, negative autopsy findings other than those minor pathologic abnormalities attributable to ethanol consumption, and the circumstances at the time of death.

A standard gas chromatographic analysis, including internal controls using *n*-propyl

TABLE 2—*Cause of death in trauma groups.*

Cause	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400+ mg/100 ml, *n*
Blunt trauma	82	27	22
Gunshot wound	28	18	8
Burns and carbon monoxide asphyxiation	17	8	3
Stab and incised wound	5	6	3
Drowning	7	6	1
Beating	1	2	1
Strangulation	1	...	...
Suicide	(11)	(1)	(2)
Gunshot wound	8	...	2
Incised wound	1	...	...
Carbon monoxide asphyxiation	2	...	...
Drowning	...	1	...
Totals	152	68	40

TABLE 3—*Causes of death in mixed groups.*

Cause	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400+ mg/100 ml, *n*
Exposure	5	5	5
Drugs	1	...	2
Positional asphyxiation	...	1	1
Sudden coronary	...	1	...
Hemorrhagic gastritis	1	...	...
Hemorrhage from laceration	...	...	1
Aspiration pneumonia	...	...	1
Sickle cell crisis	1	...	...
Meningioma	1	...	...
Myocardial infarction	...	...	2
Total	9	7	12

alcohol as well as daily ethanol standard determinations, was performed by trained and experienced toxicological chemists in the office of the chief medical examiner on all material. An attempt was made in all cases to discern if the decedent had been a chronic alcoholic. The criteria included history or the pathologic stigmata of chronic ethanol abuse (enlarged fatty liver, cirrhosis, esophageal varices, and so forth).

The investigation also included comparisons with county population density, liquor sales for the state for that period, and a survey of all alcohol testing and acute poisoning cases from established medical examiner systems and coroner offices throughout the nation. The latter information included only those cases of acute ethanol poisoning in which there were blood (or the equivalent) levels of 400 mg/100 ml or greater.

Results

The age, race, and sex distribution and the manner of death for the three groups of blood ethanol levels for all cases are listed in Table 4. There is little difference between the age distribution or range of the white and black males whose manner of death was either acute ethanol poisoning or trauma with ethanol blood levels greater than 300 mg/100 ml. The majority occurs between the third and sixth decades of life, with a pronounced peak in the fourth decade. Similar fatalities in the American Indian male have an earlier age range with a concentration of deaths in the third decade. The white females in their fourth decade of these groups have a narrower range with a similar but less pronounced peak as do males. As might be expected, those fatalities in the mixed groups tend to have a more random pattern of age, race, or sex; in the category of natural deaths, all of the individuals are of an older age group and range.

The incidence of chronic alcoholism in all cases of unnatural deaths in each of the ethanol blood level categories is presented in Tables 5, 6, and 7. These data support the conclusions drawn from Table 4 as to the age, race, and sex distribution and the range of fatalities related to acute alcoholism. In this study the overall incidence of proven chronic alcoholism in the 300 to 349 mg/100 ml group is 55.2%; in the 350 to 400 mg/100 ml group, 52.1%; and in the 400 mg/100 ml and over group, 71.8%. These percentages are minimums based on the available data, and the true incidence could certainly be much higher. In only 4 of the 502 cases was there adequate evidence that the decedent was not a chronic alcoholic. It should be noted that, in general, females have higher incidence of chronic alcoholism than males, except for American Indian males. Also, it is worth pointing out that the higher the blood ethanol concentration encountered in these deaths, the higher the proven incidence of chronic alcoholism.

TABLE 4—*Manner of death and age-sex-race distribution in fatalities with blood ethanol levels of 300 mg/100 ml or greater.*

	300 to 349 mg/100 ml, years				350 to 399 mg/100 ml, years				400 + mg/100 ml, years			
Race/Sex	AEP[a]	Trauma[b]	Mixed[c]	Natural	AEP[a]	Trauma[b]	Mixed[c]	Natural	AEP[a]	Trauma[b]	Mixed[c]	Natural
White/male												
Mean	48.7	45.7	47.0[d]	56.3	44.6	45.8	55.0[d]	55.0[d]	45.7	42.2	44.0[d]	...
Median	49	45	...	51	48	44	...	...	47	45	...	...
Mode	50	33	...	51	52	56	...	...	46	45	...	...
Range	28–60	18–70	45–50	44–76	24–67	21–69	50–60	43–64	21–67	27–52	35–86	...
White/female												
Mean	47.1	48.3	26[d]	60[d]	60.7[d]	44.1	54.4[d]	55.5[d]	44.8	45.8[d]	58.0[d]	...
Median	47	48	...	...	...	44	...	...	44	...	...	...
Mode	47	...	...	...	...	...	...	...	44	...	...	...
Range	44–53	37–62	26	60	48–74	36–55	49–60	54–57	30–61	33–58	58	...
Black/male												
Mean	48.3	37.5	44.0[d]	52.0[d]	44.6	41.1	39.0[d]	41.5[d]	43.36	40.9	43.2[d]	...
Median	49	36	...	...	48	43	...	...	46	43	...	...
Mode	42	36	...	...	52	25	...	...	47	42	...	...
Range	27–69	16–63	26–56	40–63	20–58	24–56	33–45	28–55	22–71	19–55	31–51	...
Black/Female												
Mean	47.2[d]	38.2	...	34.0[d]	42.5[d]	43.5[d]	...	45.0[d]	44.7	30[d]	35.0[d]	...
Median	...	40	...	...	...	...	...	...	39	...	...	...
Mode	...	40	...	...	...	...	...	...	38	...	...	...
Range	29–62	18–50	...	34	16–60	35–53	...	45	17–60	25–36	35	...
Other												
Mean	41[d]	28.0[d]	...	...	38.0[d]	38.0[d]	...	...	38.7[d]	...	...	...
Median	...	...	...	...	...	...	...	...	...	...	...	...
Mode	...	...	...	...	...	...	...	...	...	...	...	...
Range	41	27–29	...	...	19–41	38	...	...	21–51	...	...	...

[a] Acute ethanol poisoning.
[b] Gunshot, fire, drowning, beating, and so forth.
[c] Large ethanol factor plus other significant related process.
[d] Those categories containing five or less cases.

TABLE 5—*Frequency of chronic alcoholism (number of chronic alcoholics/total fatalities) for individuals with blood ethanol levels between 300 and 349 mg/100 ml.*[a]

Age, years	White Males	Black Males	White Females	Black Females	American Indian Males	Total
16–19	1/1	0/3	...	1/1	...	2/5
20–29	0/7	5/12	0/1	2/2	1/2	8/24
30–39	6/14	19/22	0/3	2/4	...	27/43
40–49	18/40	11/22	6/10	3/7	1/1	39/80
50–59	21/30	8/12	2/3	1/1	...	32/46
60–69	7/13	1/5	2/2	2/2	...	12/22
70–79	2/2	...	...	...	...	2/2
80+	...	...	...	...	...	...
Unknown	1/1	...	...	...	...	1/1
Total	56/108	44/76	10/19	11/17	2/3	123/223

[a]Chronic alcoholics: 123 (55.2%); insufficient data: 99 (44.4%); adequate evidence of not being chronic alcoholic: 1 (0.4%).

TABLE 6—*Frequency of chronic alcoholism (number of chronic alcoholics/total fatalities) for individuals with blood ethanol levels between 350 and 399 mg/100 ml.*[a]

Age, years	White Males	Black Males	White Females	Black Females	American Indian Males	Totals
16–19	...	...	...	0/1	1/1	1/2
20–29	2/3	5/7	0/1	...	...	7/11
30–39	4/11	5/9	1/1	2/2	0/1	12/24
40–49	4/15	9/18	3/6	...	0/1	17/40
50–59	8/15	7/14	2/2	1/2	...	18/33
60–69	3/6	...	3/3	1/1	...	7/10
70–79	...	...	1/1	...	...	1/1
80+	...	...	...	...	...	...
Totals	21/50	26/48	10/14	4/4	2/3	63/121

[a]Chronic alcoholics: 63 (52.1%); insufficient data: 58 (47.9%).

TABLE 7—*Frequency of chronic alcoholism (number of chronic alcoholics/total fatalities) for individuals with blood ethanol levels of 400 mg/100 ml or greater.*[a]

Age, years	White Males	Black Males	White Females	Black Females	American Indian Males	Total
16–19	...	0/1	...	1/1	...	1/2
20–29	3/6	4/8	...	0/1	1/1	8/16
30–39	10/15	11/14	4/6	9/15	...	34/50
40–49	24/26	16/24	10/14	5/5	0/1	55/70
50–59	11/16	7/11	2/2	4/4	1/1	25/34
60–69	3/4	1/3	2/2	1/1	...	7/10
70–79	...	1/1	...	...	...	1/1
80+	1/1	...	...	...	...	1/1
Total	52/68	40/62	18/24	20/27	2/3	132/184

[a]Chronic alcoholics: 132 (71.8%); insufficient data: 49 (21.2%); adequate evidence of not being chronic alcoholics: 3 (1.6%).

The month of the year in which the unnatural, alcohol-associated fatalities occurred and the numbers of deaths in each category (that is, the acute ethanol poisoning group, the trauma group, and the mixed group) were compared to the whiskey sales in the state during the period of study. The results are presented in Fig. 1. There is a distinct concentration of unnatural fatalities during the fall and winter months, which coincides with an increase of liquor sales during this period. In this survey there also appears to be a smaller grouping of deaths in the trauma and mixed groups during the late spring and early summer. It is noteworthy that the lowest frequency of fatalities in all of these groups occurs during July.

A similar comparison of the frequency of death in these cases and the day of the week on which the fatalities occurred may be seen in Table 8. Those cases of acute ethanol poisoning are distinctly clustered around the weekends and there is a smaller group of fatalities during the middle of the week. The mixed group shows a similar but less distinct pattern, while the trauma group has the characteristic peak during the weekend but no mid-week grouping. Unfortunately, the records of these cases were not so complete as to allow determination of the time of day of these fatalities with reliable accuracy.

The place of death of the unnatural fatalities with blood ethanol levels of 300 mg/100 ml or more is catalogued in Table 9. The majority of those caused by acute ethanol poisoning are found in homes, whether the decedent's or a friend's. In this group it is also significant that 26 cases were discovered in parked motor vehicles. This is not infrequent in this cause of death and it seems to be of no consequence whether the motor vehicle is functional or not. Of those deaths in the mixed category, the place where the decedent is found has a random distribution. In those individuals with high ethanol levels dying of trauma there are two major groups: those who die in a home and those who die in the streets or highways. This is considered to be a reflection of the violent nature of the deaths associated with high blood ethanol levels, usually resulting from motor vehicle accidents, gunshot wounds, fires, beatings, and so forth (see Table 2).

The occupational groups of those decedents with excessively high blood ethanol levels (greater than 400 mg/100 ml) are listed in Table 10. The categories of work used here are the same as those used by the Bureau of Vital Statistics of the State of North Carolina. In all fatalities, regardless of the cause of death, between 70 and 80% are in those groups considered to be of a lower socioeconomic class. Those deaths attributed to acute ethanol poisoning do not commonly occur within the professional strata. The range of deaths from a high ethanol factor plus a contributing related circumstance is sporadic. The deaths resulting from trauma occur mostly in those groups involved with manual labor, disabled persons, or the unemployed.

Those fatalities with blood ethanol levels of 400 mg/100 ml or more were compared with the population density of the counties in which the deaths occurred. The results are presented in Table 11. Those counties having less than three acute ethanol fatalities during the period of study were excluded from this chart. No pattern related to county population or either urban or rural areas could be discerned. Of the five counties having the most frequent occurrence/population ratio, two were densely populated areas and three were less densely populated. Of the 16 counties listed, the most densely populated (Mecklenburg) ranked sixth in frequency of acute ethanol poisonings. The least densely populated (Union) ranked eighth.

Despite North Carolina's reputation as being among the nation's leaders in the production of illicit whiskey, lead poisoning from the consumption of this material is extremely rare. During the two years of this study, there was a concerted search for lead in "white whiskey" specimens obtained from decedents who were known to consume it habitually and in liver specimens from autopsy subjects. There were no elevated levels of lead in the cases presented in this paper. The great majority of reports of lead-contaminated, un-

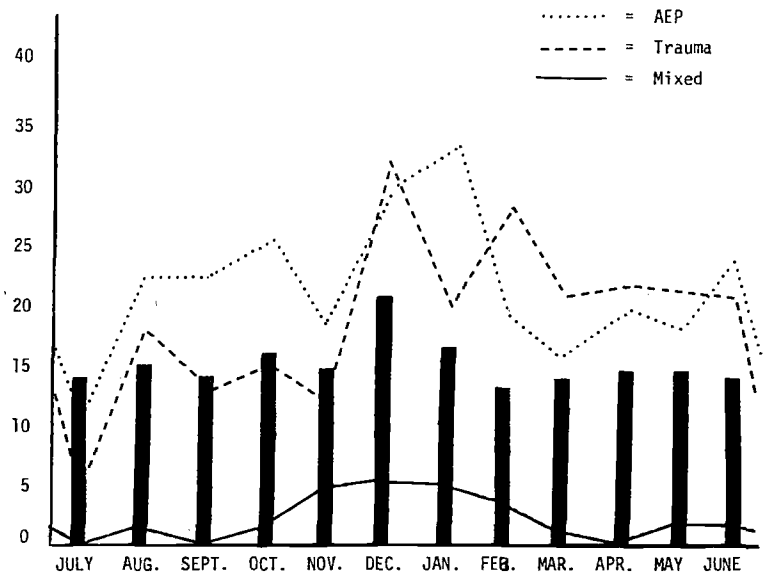

FIG. 1—*Comparison of whiskey sales and alcohol-associated deaths by month. Whiskey sales for each month were averaged and rounded off to the nearest $100 000. This does not include beer and wine sales. All fatalities had a blood ethanol level of 300 mg/100 or greater.*

bonded whiskey have come from Alabama and Georgia, which rival North Carolina in the production of non-taxed whiskey.

Table 12 presents data on acute ethanol deaths collected from other medical examiners and coroner systems throughout the nation for a comparable period of time and are correlated with the data from North Carolina during the first year of this investigation. During the second year of investigation the medical examiner system encompassed approximately 80% of the population and land area of the state. The number of deaths certified by medical examiners during the 24-month period was 9433, 47% of which were sampled for ethanol analysis. The percentage of deaths from acute ethanol poisoning resulting from blood ethanol levels of 300 mg/100 ml or more for this two-year period was 2.5%. Recognition of acute ethanol poisoning is, to some extent, dependent on the number of deaths competently certified by knowledgeable medical examiners and coroners and is based on the percentage of the total number of cases tested for blood ethanol levels. These data and other sources indicate a much higher incidence of acute poisoning in the southeastern states than in other parts of the country. When this is contrasted with the data and conclusions of the report from the National Institute on Alcohol Abuse and Alcoholism [*6*] on alcohol consumption throughout the nation covering the same period, one finds that alcohol consumption was heaviest in the Pacific states and Alaska, followed by New England and the Middle Atlantic states, Wisconsin, and Florida. The southeastern states were listed as having moderate to low consumption, with Alabama being the state with the lowest consumption in the nation. These data were calculated from the amount of taxed alcoholic beverages sold and the population of each state [*6*].

Discussion

The significance of the various biological effects of ethanol has been the subject of numerous reports [*7–14*], including investigations and reviews on the physiology, pharma-

TABLE 8—*Day of week of death for fatalities with blood ethanol levels of 300 mg/100 ml or greater.*

	Acute Ethanol Poisoning			Mixed[a]			Trauma[b]		
Day	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400 + mg/ 100 ml, *n*	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400 + mg/ 100 ml, *n*	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400 + mg/ 100 ml, *n*
Sunday	8	12	37	1	3	3	41	15	8
Monday	8	4	16	1	1	0	18	11	5
Tuesday	2	0	10	0	0	2	12	6	1
Wednesday	5	7	14	2	0	2	11	2	4
Thursday	3	3	11	1	0	0	13	3	6
Friday	7	3	12	1	1	2	14	8	5
Saturday	10	9	32	4	2	3	43	23	11
Total	43	38	132	9	7	12	152	68	40

[a] Large ethanol factor plus other significant related process.
[b] Gunshot, fire, drowning, beating, and so forth.

TABLE 9—*Place of death for fatalities with blood ethanol levels of 300 mg/100 ml or greater.*

Place	Acute Ethanol Poisoning			Mixed[a]			Trauma[b]		
	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400 + mg/ 100 ml, *n*	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400 + mg/ 100 ml, *n*	300 to 349 mg/100 ml, *n*	350 to 399 mg/100 ml, *n*	400 + mg/ 100 ml, *n*
Decedent's home	29	21	88	3	4	6	41	19	12
Friend's home	3	2	13	1	...	...	16	6	4
Parked car	(6)	(7)	(13)	(1)	(1)	...	(4)	...	...
Disabled	4	1	7	...	1	...	1	...	...
Functional	2	6	6	1	...	...	3	...	...
Jail	1	5	...	...	...	...	...	1	...
Open area	2	3	5	4	1	4	7	13	2
Place of work	1	3	4	...	...	...	3	...	...
Highway-street	...	...	...	...	...	...	(75)	(25)	(20)
Driver	...	...	...	...	...	...	30	11	6
Passenger	...	...	...	...	...	...	17	2	6
Pedestrian	...	...	...	...	...	...	28	12	8
Other	...	2	4	...	1	2	4	3	2
Not stated	1	...	...	...	...	...	3	1	...
Total	43	38	132	9	7	12	153	68	40

[a] Large ethanol factor plus other significant related process.
[b] Gunshot, fire, drowning, beating, and so forth.

TABLE 10—*Occupational groups for fatalities with blood ethanol level of 400 mg/100 ml or greater.*

	Cause of Death							
	Acute Ethanol Poisoning		Mixed[a]		Trauma[b]		Total	
Occupational Groups	*n*	%	*n*	%	*n*	%	*n*	%
Unskilled or semiskilled labor	24	18	4	33	16	40	44	24
Skilled or semiskilled labor	28	21	1	8	7	18	36	20
Sales; service; domestic	30	23	0		4	10	34	18
Unemployed; disabled; retired	23	17	4	33	4	10	31	16
Housewife	13	10	1	8	2	5	16	9
Farmer	3	2	1	8	3	8	7	4
Manager	2	2	0				3	2
Student; military	2	2	0				2	1
Professional	1	1	1	8	1	2	3	2
Not known	6	4	0		2	5	8	4
Totals	132		12		40		184	

[a] Large ethanol factor plus other significant related process.
[b] Gunshot, fire, drowning, beating, and so forth.

TABLE 11—*Rate of fatal acute ethanol poisoning with blood ethanol levels of 400 mg/100 ml or greater relative to total deaths.*

County[a]	Acute Ethanol Poisoning	Population (in 1000s)	Total Deaths (All Causes)	Acute Ethanol Poisoning per 1000 Deaths,[b] %	Rank
Buncombe	6	145	2 930	2.0	12
Cabarrus	3	75	1 390	2.2	11
Catawba	5	91	1 370	3.6	4
Durham[c]	6 (× 2)	133	2 390	5.0	1
Forsyth	6	214	3 700	1.6	15
Gaston[d]	5	145	2 500	2.0	13
Guilford	18	288	4 650	3.9	3
Harnett[d]	3	49	1 010	3.0	8
Mecklenburg	17	354	5 350	3.2	6
Nash	3	59	1 200	2.5	9
Orange	3	58	670	4.5	2
Randolph[d]	4	76	1 230	3.2	7
Roberson	3	85	1 660	1.8	14
Rutherford[d]	3	47	990	2.5	10
Union	3	55	860	3.5	5
Wake	5	228	3 280	1.5	16
Totals	99	2102	35 180	...	...
Average	6.2	131.4	2 200	2.8	...

[a] Counties with three or more cases.
[b] Percentage correlated with frequency of submission of blood samples.
[c] Durham data available for only one year. Value was doubled and treated as two years for consistency.
[d] No medical examiners during period of study; active coroner.

TABLE 12—*Acute ethanol poisoning deaths; data solicited from county and state medical examiner systems.*

Area[a]	Deaths Certified by Medical Examiner or Coroner	Tested For Ethanol, %	Acute Ethanol Deaths	Acute Ethanol Poisoning Deaths, 400 + mg/100 ml	
				n	%
Sedgwick Co., Kans. (Witchita)	450	...	1	0	0.00
Clark Co., Nev. (Las Vegas)	796	65	1	0	0.00
Nassau Co., N.Y.	2 600	98	24	1	0.04
Hennepin Co., Minn. (Minneapolis)	3 006[b]	31	10[b]	5[b]	0.04
Hamilton Co., Ohio	3 058	15	6	2	0.06
Dallas Co., Tex.	2 720	97	11	2	0.08
New York City	25 000	7	300	20	0.08
Delaware	1 120	50	9	1	0.10
Cook Co., Ill. (Chicago)	11 108	22	16	12	0.10
Utah	848	63	4	1	0.12
Monroe Co., N.Y. (Rochester)	1 655	33	7	2	0.12
Dade Co., Fla. (Miami)	4 547	34	10	6	0.13
Maryland	7 052	33	33	9	0.13
Cuyahoga Co., Ohio (Cleveland)	11 104[b]	57	17[b]	15[b]	0.13
Oklahoma	6 134[b]	40	14[b]	10[b]	0.16
Philadelphia Co., Pa.	4 059	73	7	7	0.17
Wayne Co., Mich. (Detroit)	11 000	27	27	20	0.22
Alameda Co., Calif. (Oakland)	2 798	21	10	7	0.25
Los Angeles, Calif.	13 781	41	50	40	0.29
Shelby Co., Tenn. (Memphis)	1 750	25	9	4	0.30
Virginia	15 742[b]	32	200[b]	48[b]	0.30
San Francisco, Calif.	2 501	97	15	10	0.40
Harris Co., Tex. (Houston)	4 500	...	25	20	0.40
Allegheny Co., Pa. (Pittsburg)	3 500	100	21	20	0.57
North Carolina	4 570	42	87	63	1.38
Georgia	2 040	98	72	70	3.43

[a] Data from Sedgwick Co., Kans.; Clark Co., Nev.; Cook Co., Ill.; Cuyahoga Co., Ohio; Alameda Co., Calif.; San Francisco, Calif.; and Allegheny Co., Pa. are from the respective coroner's systems.
[b] Data for two years.

cology, chemistry, related disease processes, epidemiology, psychosociology, and medicolegal aspects pertaining to this universally distributed compound and its relatives. These investigations have greatly contributed to our understanding of ethanol and related problems, but broad gaps remain in our knowledge of many aspects of alcoholism.

This is particularly true in the forensic sciences. The medical examiner or his equivalent seeks to detect cause of death as well as contributing, aggravating, precipitating, and other relevant factors. A physician need not have extensive experience to recognize the high frequency of blood ethanol levels in the decedents whom he investigates. He further realizes that ethanol is either a highly significant factor in a vast proportion of these deaths or that at any given time a great percentage of the population has been consuming this substance.

Certainly, moderate to high levels of blood ethanol can be found coincidentally with or can cause aggravation of an existing disease process. It may obscure a disease or injury and may increase susceptibility to all manners of death (natural, accidental, suicide, or homicide). The stigmata of chronic alcoholism and its related diseases are well documented [*8*]. Sudden, unexpected deaths of known heavy drinkers with low or no blood

ethanol or other alcohol levels have occurred [*9*]. One of the mechanisms for the body to dispose of excess fats mobilized by excessive ethanol ingestion is to convert some of it into water-soluble breakdown products (ketone bodies) and secrete them into the bloodstream. In some susceptible people this response may be exaggerated, resulting in an elevation of ketone bodies in the blood that mimics the condition of ketoacidosis, which may be fatal, in diabetic patients [*15*]. Another hepatic abnormality resulting from excessive ethanol ingestion is an imbalance of hydrogen ions within the liver cells which blocks gluconeogenesis by the diversion of pyruvate to lactate and results in low blood glucose levels. This direct complication of acute ethanol toxicity is often overlooked. When an intoxicated person is brought for medical attention, it is important that the possibility of hypoglycemia be determined since crucial organs, including the brain, can be critically affected by a lack of glucose [*16*]. Some deaths of "unknown origin" in alcoholics may be attributable to this condition. For a variety of reasons, acute ethanol poisoning has not been widely recognized or correctly categorized by lay investigators, physicians, or even some experienced medical examiners.

One major reason for this lack of recognition is the graphs and charts of alcohol toxicity that are so widely circulated in popular texts and journals [*17,18*]. These indicate that coma and death do not occur until blood alcohol levels reach 400 mg/100 ml. A dose of 3 g of ethanol per kilogram of body weight distributed throughout total body water has been reported to be lethal to 50% of consumers. The usual mechanism is respiratory depression [*19*]. However, this LD_{50} is dependent on a wide individual susceptibility to the drug and is affected by many factors such as the individual's ratio of weight to body fat content, the speed and pattern of drinking, the presence or absence of food in the stomach, the effects of physiologic or pharmacologic alteration of gastric motility, and numerous other variables. The toxic effect of large quantities of alcohol is a stress on an organism, and any individual who is already under stress from a disease process, either overt or latent, can withstand less additional stress than a healthy one [*20*]. Because of his ability to continue to metabolize ethanol, an intoxicated person may survive long enough in coma so that on death the blood ethanol concentration found may be below that having a high probability of causing death from acute poisoning.

The legends of racial group variation in biological tolerance of ethanol [*21*] have been found to be scientifically unsound; the basis of the reports of differences in the incidences of alcoholism in these groups may be found in cultural and acquired factors [*22,23*]. Acquired tolerance of the nervous system modifies the degree or effect produced at any given blood ethanol concentration [*24*].

The significance of simultaneous presence of another drug or drugs and moderate blood ethanol levels may be ignored or misinterpreted. Most depressants and sedatives will have an additive effect. Some drugs, particularly the barbiturates, will have a potentiating or synergistic effect [*25*]. Because of alterations in the liver cells' endoplasmic reticulum and changes in mitochondrial function in heavy ethanol consumers, the effects of many foreign compounds, which may be harmless to some people, may be toxic or accentuated, not only because the effect of the two drugs on the brain may be additive but also because the presence of ethanol can interfere with the liver's capacity to inactivate the drug so that at a given dosage more of the drug remains active for a longer time and becomes synergistic. Paradoxically, there is a metabolic adaptation as well in the increased capacity of the alcoholic liver to inactivate and excrete sedatives detoxified by the endoplasmic reticulum; therefore, larger doses of many drugs to achieve a given effect are required in alcoholics. This is true, however, only when the individual is sober; when the alcoholic has been drinking, the effect is quite the opposite [*9,20,26,27*].

The phenomenon of acute poisoning is relatively infrequent in may parts of the nation. These acute toxic deaths are believed to be almost invariably accidental. Suicide by this

method may occur but must be very rare. When there is no conclusive autopsy finding but an appropriate history of the circumstances and high blood ethanol levels exist, acute ethanol poisoning is the correct term for the cause of death, and the manner of the fatality is accidental. If there is another contributing circumstance, then the appropriate cause and manner of death may be listed with acute alcoholism as a contributing factor; the mention of ethanol should not be avoided because of concern for real or imagined social stigma.

Even when this phenomenon is recognized, there is a tendency to certify the death as "natural." From a survey made of many parts of the nation, some of the reasons for determining this mode of death as natural were collected and are as follows: "I never thought of calling it anything else," "I refuse to give those drunks that break," "The International Classification of Disease Code requires it," "They are accidents but the Boss says call them 'natural'," and "The insurance companies prefer 'natural'." It would seem important for a variety of reasons, insurance settlement not the least, to consistently determine with accuracy the manner of death in these cases.

Some vital statistic offices of state health departments throughout the nation frequently do not even code this form of death as accidental even though the manner of death may have so been indicated on the death certificate. If on the certificate the cause of death is certified as acute alcoholism, acute ethanol intoxication, acute ethylism, or other similar terms, the cause is coded with chronic fatal complications of alcoholism such as cirrhosis, fatty liver, Wernicke's encephalopathy, or other abnormalities associated with chronic abuse. The cause of death having been classified among these phenomena, the manner of death is then pigeon-holed as natural rather than accidental in the certifying offices. Unfortunately, this renders state and national statistics, at least those from death certificates, valueless in terms of valid data about the pathology, occurrence, and epidemiology of acute and chronic alcoholism.

The data presented in this paper define the syndrome of acute ethanol poisoning as a distinct entity and compare many of the features of this cause of death with those fatalities with equally high ethanol levels in which contributing or related factors were found and those which had violent or traumatic deaths. These modes of death have some factors in common, but in the differential diagnosis of acute ethanol poisoning there are certain characteristics and patterns that are helpful in certifying these deaths accurately.

Those persons who die unnatural deaths with blood ethanol levels of 300 mg/100 ml or greater without evidence of related processes, violence, or trauma are most often white males between the third and sixth decade of life, with the most frequent incidence being in the fourth decade. Black males are the next most frequent having this mode of death, and the age pattern is similar to that found in the white males. Females of both races are less frequent victims of this fatality and have a similar age grouping, except for black females who tend to have a slightly younger age range. A little more than 50% of the fatalities involving blood ethanol levels of from 300 to 399 mg/100 ml have either a well-documented history or pathologic findings of chronic alcoholism. In those deaths with greater than 400 mg/100 ml blood ethanol levels, there is a frequency of chronic alcoholism of greater than 70%. In very few of all these deaths is there sufficient evidence of the decedent not being a chronic alcoholic.

The most frequent acute ethanol poisoning is during the fall and winter months, which correlates with the increased liquor sales during this period. There is a pronounced decrease of acute ethanol poisoning in July. Deaths caused directly by the toxic effects of ethanol occur most frequently during the weekends and holidays, and there is an increase in deaths from ethanol toxicity plus other contributing factors during these times. Most of these deaths occur in the home; however, one recurrent feature is a significant number who die in parked motor vehicles. Most fatalities are laborers, the unemployed, or the

disabled. A smaller but significant percentage were housewives. No correlation could be drawn between the frequency of acute ethanol poisoning and population density. Although the recognition of acute alcoholism as a cause of death is dependent on the numbers of deaths certified by competent officials and the percentage of these cases tested for ethanol, the data revealed a marked increase in the percentage of these types of death in the southeastern states. This feature does not correlate with the quantity of liquor consumed when compared to the population of the other regions of the nation.

The abuse of ethanol is a series of difficult problems in this and other countries that can lend itself to many well-established problem-solving techniques. One has to proceed with as little prejudice as possible, with increasing awareness, concern, and effort to collect as much relevant data as possible to generate information needed by many organizations and agencies, private, governmental, and academic. From such a cooperative effort, criteria delineating the abuse of this drug can be reassessed and appropriate changes made. Some specific contributions from the forensic sciences would include (1) the identification of acute and chronic alcoholism among the population investigated that would not otherwise be identified; (2) making available for the students of ethanol-related problems information derived from those cases found to have ethanol in the blood at the time of death and the levels present; (3) information on ethanol blood levels being made available to other appropriate agencies such as insurance companies, departments of motor vehicles, and attorneys in criminal and civil suits; (4) epidemiologic data on ethanol abusers; (5) identification of specific problem areas such as the numbers of acute ethanol poison deaths; (6) interpretation of ethanol levels for law enforcement agencies; and (7) encouragement of insurance companies to apply economic leverage on those insured through ethanol-consumption limitations in insurance coverage.

Summary

A 24-month study of fatalities in North Carolina with high blood ethanol levels (300 mg/100 ml or over) revealed 502 cases with either acute alcoholism or the effects of this range of blood ethanol concentration having caused or contributed to death. This investigation reassessed the criteria for ethanol poisoning, including its cause and manner of death, and revealed recurrent patterns common to this syndrome. This inquiry also contrasted the frequency of ethanol poisoning in different areas of the country.

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