A Methanol Poisoning Outbreak in Kentucky

A Clinical Epidemiologic Study

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A change in the brand of shellac thinner used to make an alcoholic beverage resulted in six deaths from methanol poisoning. In screening all potentially poisoned persons available with serum methanol, ethanol, pH, and electrolyte determinations, many asymptomatic methanol poisonings were discovered. A correlation was established between severity of illness and level of ethanol, further suggesting a protective effect.

Among different types of treatment used with the 12 hospitalized cases peritoneal dialysis was shown to be effective, with a clearance rate five to ten times that obtained with forced fluid diuresis.

SEVERAL epidemics of methanol poisoning have been described in the medical literature.[1-9] It is our purpose to describe a group of cases resulting from a common-source epidemic which involved 18 people, of whom eight died. Of the 18 known cases, 11 were treated at the University of Kentucky Medical Center with only one death despite several patients with methanol concentrations of 200 mg or more per 100 ml blood. Fatalities have been reported with serum methanol levels of under 200 mg/100 cc.[1,10] In addition to providing treatment, the hospital instituted an emergency screening program for all suspected methanol imbibers. This procedure facilitated the detection and treatment of asymptomatic persons with markedly elevated blood methanol levels, which added a new dimension to the clinical experience with methanol poisoning. Frequent chemical determinations provided new information on the rate of methanol excretion by various routes and the relative efficacy of different modalities of therapy.

Since Bennett's work[1] in the epidemic of methanol poisoning in Atlanta in 1953, the major therapeutic advance in the treatment of methanol poisoning has been the application of dialysis therapy with special attention focused on extracorporeal treatment. In none of the 11 patients studied here was hemodialysis used. Peritoneal dialysis was evaluated and attempts were made to follow methanol excretion rates with and without dialysis.

Epidemiology

The epidemic began with the distribution of diluted paint thinner from a regular source in a low socioeconomic area of Lexington. "Heads" is an alcohol drink made by diluting shellac thinner, usually a brand with a high ethanol and low methanol content. On this occasion a different compound was employed—a shellac solvent (Thinz-all) with approximately 74 vol% methanol (Table 1). Analysis of fluid contained in bottles found near some of the victims showed that the solvent probably had been diluted to a final concentration of about 37 vol% methanol. Two major population groups appear to have been affected: (1) the Negroes living in the neighborhood of the seller; and (2) the white derelict alcoholics who relied on this seller for their regular supply.

Shellac thinner can be purchased commercially at any paint store for approximately $1.95 a gallon. When this is diluted one or two times (often to the purchaser's taste) it is usually poured into old half-pint bottles which sell as "heads" for approximately 25 cents. The potential profit thus ranges from 100% to 200%.

Reconstructing the events surrounding this particular episode, apparently two or three gallons of thinner were purchased and diluted once with water. About half of the liquor so obtained was served as the major refreshment for a party held at an adjoining house. Part of the remaining lot was sold along the usual channels until the seller himself became ill. At this point

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Table 1.—*Analysis of Various Shellac Thinners*

Compound	Spec Gr at 25 C	Methanol	Ethanol	Acetone	Ethyl Acetate
Quakersol	0.8	2.45*	51.4	0.85	3.33†
Solox	0.8	2.32	52	0.85	1.37†
Jaysol	0.79	2.84	61.5	0.65	1.3†
Thinz-All	0.78	74.4	0.5	<0.5	<0.5‡

* Content measured in volume percent. The total of percent by volume will approach 100% by weight for each of these mixtures when corrections are made for the specific gravity of the components and the product itself, for example, Thinz-All has a methanol concentration of 94% by weight.

† Normally used as basic ingredients for "heads."

‡ Used in mixing "heads" described in this epidemic.

his wife took over the business and continued to distribute the methanol although it had been suspected to be "a bad batch." There was no reliable means of determining how many persons had obtained some of this alcohol.

The initial patient (case 11) was admitted to the University of Kentucky Medical Center with a history of drinking denatured alcohol. He became comatose shortly after admission and never regained consciousness. By the following morning reports of several deaths at neighboring hospitals and the finding of a corpse surrounded by empty bottles containing traces of methanol indicated that an epidemic of methanol poisoning might be developing. The police identified the distributor when, after his death, his wife presented herself in our emergency room as a possible victim of the poison within 24 hours of the first death. Although she refused admission after learning that she had no methanol in her blood, she offered us a

Table 2.—*Screening Values in Persons Suspected of Methanol Poisoning*

Case	Age, Sex	Race	Symptoms	HCO_3	pH	Meth	Eth	Disposition
			A. Methanol and Ethanol					
1	36, M	W	CR, BV*	27.5	7.58	48	346	Admitted
2	50, F	N	—	15.5	7.35	218	156	Admitted
3	39, M	W	—	26.5	7.37	372	118	Admitted
4	48, M	W	AP, BV	28.3	7.30	82	273	Admitted
5	47, M	W	BV	24.3	7.32	77	244	Admitted
Averages	44			24.4	7.38	159	227	
			B. Methanol Without Ethanol					
6†	40, M	W	BV	24.5	7.55	110	0	Admitted
7	57, M	W	—	9.0	7.27	30	0	Admitted
8	59, M	N	BV, CR	7.5	7.16	237	0	Admitted
9	60, M	N	—	11.5	7.20	246	0	Admitted
10	39, M	N	—	27	7.42	267	0	Admitted
11‡	55, M	W	AP, NV, SC	4.0	7.04	216	0	Admitted
Averages	54			11.8+	7.22+	199	0	
			C. Others					
12	43, M	W	BV	23	7.47	0	215	Discharged
13	46, M	W	AP, BV, D	25.7	7.41	0	201	Discharged
14	53, M	W	AP		7.46	0	0	VA hospital
15	43, M	N	C	25.5		0	Iso 144 Acet 237	Admitted
16	44, M	W	—	21	7.39	0	143	Discharged
17	44, M	W	BV, AP	25.5	7.36	0	243	Discharged
18	49, M	W	—	20.8	7.30	0	291	Discharged
19	43, M	W	AP, SC	31.5	7.48	0	0	Admitted for other causes
20	66, M	N	AP	19.5	7.40	0	27.3	Admitted for other causes
21	65, M	W	A	26.5	7.34	0	326	Discharged
22	53, M	W	—	24	7.36	0	246	Discharged
23	36, M	W	—	23.5	7.38	15	235	Discharged
24	56, F	W	AP	23	7.44	0	170	Discharged
25	76, M	N	—	27	7.40	0	28.5	Discharged
26	64, M	W	—	26	7.40	0	262	Discharged

* AP indicates abdominal pain; CR, leg cramps; BV, blurred vision; NV, nausea and vomiting; SC, scotomata D, diarrhea; C, comatose; A, asthma; Iso, isopropyl alcohol; Acet, acetone. Plus indicates differences in groups A and B significant at $P<0.05$.

† Given bicarbonate at another hospital.

‡ Index case.

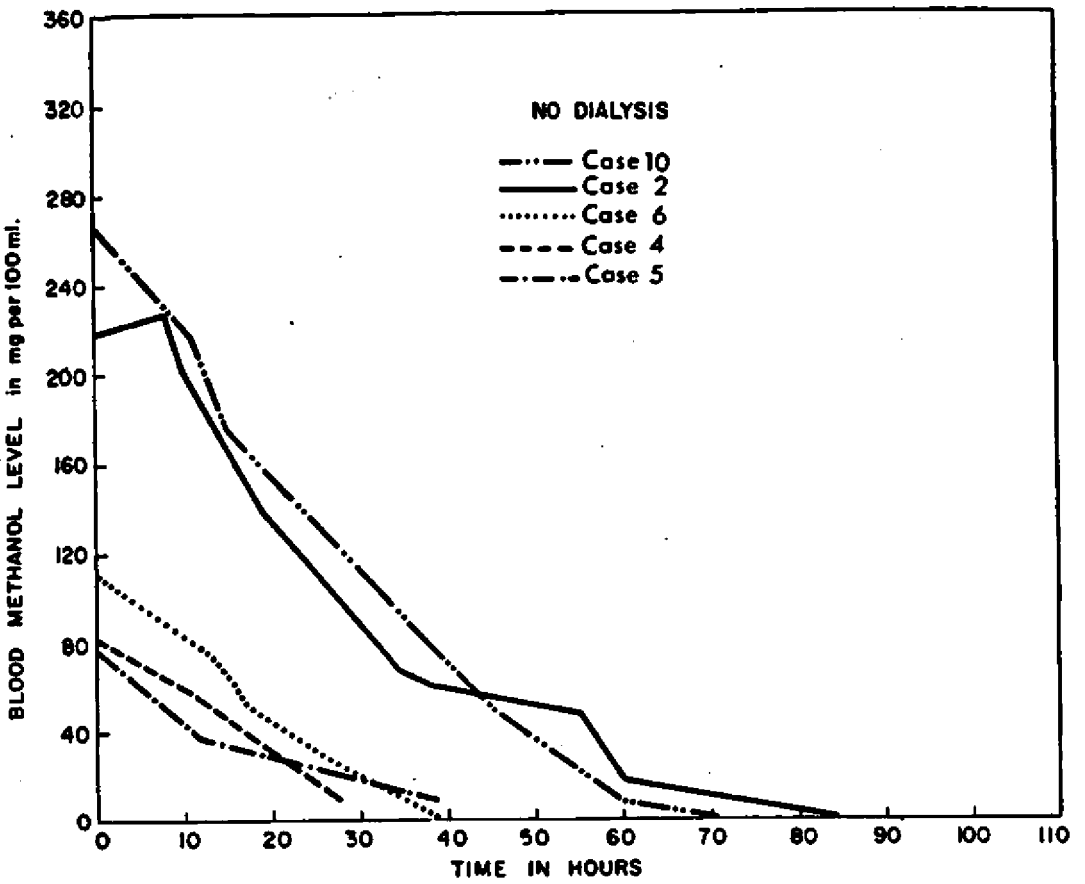

Fig 1.—Rate of disappearance of methanol from the blood of patients not treated with peritoneal dialysis (time 0 = admission).

list of names of persons to whom she claimed to have sold the "heads." The newspapers and mass media publicized the story and many people referred themselves to the emergency room for screening. The police searched the known haunts of alcoholics and brought in more suspected imbibers for investigation. The area where the alcohol had been made was visited by one of the authors (R. K.) after the admission of the second case, and several additional victims identified themselves for treatment at that time.

In all, some 26 persons were screened in the emergency room, of whom 13 were admitted, 11 for methanol intoxication. The screening procedure in the emergency room was based on immediate determinations of electrolytes and venous pH, with blood methanol levels reported within 12 hours. On the basis of clinical examination and acid-base status all suspects were either admitted, held in the emergency room, or held by the police until the report of blood methanol levels was available. The values of these parameters as screening indices are shown in Table 2.

It was not possible to ascertain reliably the association of any of those screened to the source of methanol. Several of those seen in the emergency room had referred themselves for drinking "bad heads" but this proved to be isopropyl alcohol (case 15) or ethanol.

Once the patients had been convinced that our laboratory studies indicated serious potential illness and possible death, they agreed to hospitalization but were not universally cooperative. Over half the patients left against hospital advice during the recuperative phase of their illness and several refused to permit various procedures. We had hoped to follow these patients after discharge, but they neither returned for further care nor could they be located at the addresses given. Many of our patients were chronic alcoholics with no fixed address.

Table 3.—Summary of Laboratory Data

Case	Age, Race, Sex	Meth	Eth	pH	HCO_3	Na	K	Cl	BUN	Plasma Cortisol
11	55, W, M	216	0	7.04	4.0	138	4.6	98	16	75.3 after 24 hr, 22.3 after 36 hr
7	57, W, M	30	0	7.27	9.0	134	3.5	94	8	23.4 on admission, 6.2 after 24 hr
1	36, W, M	48	346	7.58	22.5	143	3.7	102	8	—
8	50, N, M	237	0	7.50	7.5	136	5.0	98	12	11.7 on admission, 26.7 after 24 hr
9	60, N, M	246	0	7.20	11.5	135	4.5	97	21	—
10	39, N, M	267	0	7.42	27	134	3.4	97	12	—
2	50, N, F	218	156	7.35	15.5	140	3.8	106	9	—
3	39, N, M	372	118	7.37	26.5	141	3.7	104	4	—
4	40, W, M	82	273	7.30	28.3	144	4.4	109	13	—
5	47, W, M	77	244	7.32	24.3	145	4.1	104	10	—
6	40, W, M	110	0	7.55†	24.5†	140	3.3	96	13	7.3 on admission; 7.5 after 24 hr.

* PD indicates peritoneal dialysis; Eth, ethanol; $NaHCO_3$, sodium bicarbonate; GL, gastric lavage; A, vitamin A; Man, mannitol; Ur, urea.
† After receiving intravenous $NaHCO_3$ at another hospital.

Methods

Methanol determinations were performed on a gas chromatograph (Perkin-Elmer F-11) using a "K" column (Hallcomid M'18 and Carbowax 600 on a Teflon-6 support) and an internal acetone standard. Serial analyses gave very little variation in the peak height of the internal standard using a 1μ sample for injection. In those cases in which isopropyl alcohol was detected or in which the internal standard peak did not closely match its expected peak height, the analysis was repeated without the internal standard. No case was observed in which complete conversion of isopropyl alcohol to acetone had occurred which would compromise the reliability of the results obtained using an internal acetone standard. The method used separated methyl, ethyl, and isopropyl alcohol and was "specific" for these substances considering the samples used. Quantitation was obtained by comparison of peak heights of closely related standards. This necessitated the use of two acetone and alcohol concentrations, each 79 and 158 mg/100 cc. These concentrations were satisfactory for most specimens and less serum was added to the internal standard for exceptionally high values.

Results

Of the 11 persons with significant blood methanol levels (greater than 30 mg/100 cc), all but the original patient were alert and oriented on admission. Table 3 and Fig 1 and 2 describe the therapeutic regimens and rate of methanol removal. In general all patients were given sodium bicarbonate ($NaHCO_3$), as needed, either orally or intravenously, to maintain normal serum pH. Force fluid regimens were used throughout with inputs of 6 to 8 liters a day for each patient. Ethanol therapy was attempted but proved unsuccessful. Initially three patients were treated with peritoneal dialysis; a fourth

spitalized Patients

Electroencephalogram	Treatment	Complications
Severely depressed brain function, bifrontal δ-waves (16 hr)	PD*, GL, Eth, $NaHCO_3$	Coma, blindness, convulsions, cardiac arrhythmias, death after 120 hr
Abnormally slow basic rhythm, slightly more slow activity in δ range on the left	$NaHCO_3$, Man, A	—
Excess slow activity in θ and δ ranges	$NaHCO_3$, A, pentobarbital	—
Abnormally slow basic rhythm, slight increase in δ activity	$NaHCO_3$, Eth, PD, A	—
Eleptogenic activity in right sylvian region	$NaHCO_3$, Eth, GL, PD	Coma, convulsions shock, urinary tract infection
Abnormally slow basic rhythm	$NaHCO_3$, Eth, GL, UR	Pneumonia
Abnormally slow basic rhythm, excess irregular slow activity	$NaHCO_3$, GL, Man	Transient isopropyl alcohol in blood
No abnormalities	$NaHCO_3$, Eth, PD, Man	Transient isopropyl alcohol in blood, transient fever
—	$NaHCO_3$	—
—	$NaHCO_3$	—
Excess slow activity	$NaHCO_3$, GL	—

patient (case 9) was begun on dialysis after more conservative therapy was unsuccessful. The dialysate of one patient was analyzed for methanol content as shown in Table 4. One pair of patients (8 and 9) with approximately equal methanol and ethanol levels, both asymptomatic, can be used, in retrospect, to appreciate the clinical and laboratory improvement with and without dialysis. However, as complications ensued in the patient on the diuresis regimen, he too was begun on dialysis with substantial improvement. In several instances vitamin A was given as recommended by Tonning et al.[9]

Urine samples and gastric drainage were collected from those patients on forced fluid and dialysis regimens in an effort to assess clearance rates by these routes. (Tables 4, 5 and 6). Liver function tests, amylase values, and glucose levels were obtained on most of the patients and are reported in Table 7. A lumbar puncture was performed on nine patients within one to six hours after admission as soon as it was feasible (two patients refused this procedure). In addition to the standard measurements of cell count, total serum protein, and glucose, this fluid was also analyzed for methanol and ethanol and is compared to the appropriate blood values in Table 8.

Seven persons outside the medical center died in connection with the epidemic. Of these, two were autopsied and in all but two cases (ironically, one was the dispenser of this poison) blood was obtained for methanol levels. These unconfirmed cases were included because of their strong historical connection with the source of the methanol. Unfortunately one had been embalmed which prevented toxicological analysis. The other had been found amidst a number of empty bottles which later analysis confirmed were "heads." Table 9 describes the information available on these seven deaths.

It was originally hoped that the patients might provide some insight into the amount of methanol each had consumed. However, on repeated questioning, they gave markedly varied responses which showed little trace of reliability. Furthermore, a few denied drinking any "heads" at all until methanol had been proven to be present in their blood, emphasizing the danger of screening the asymptomatic patient in an epidemic on the basis of history alone or in conjunction with pH and bicarbonate determinations. All did at some time admit to obtaining their "heads" from the same general area but most were reluctant to implicate any particular source.

Comment

Methyl alcohol poisoning has been recognized since before the turn of the century but only relatively recently has there been the potential for efficacious therapy. The classic descriptions of blindness, acidosis, coma, and death[11] can often be altered now by earlier diagnosis and more effective intervention. Although peritoneal dialysis had been experimentally described as early as 1947,[12] the mainstay of therapy in that era was an alkali regimen[2,13-16] with some use of ethanol,[4,7,8,17-19] although the efficacy of the latter was not always clear.[20]

Experimental work has now shown that

Table 4.—*Course of Methanol Removal by Peritoneal Dialysis in Patient 3*

Dialysis Exchange	Time (hr after admis)	Volume (ml)*	Methanol Concentration (mg/100 cc)	Methanol Content (mg)	Cumulative Methanol Removed by Dialysis (mg)	Methanol Blood Level (mg/100 cc)	Clearance Rate (ml/hr)
1	6¾	—	—	—	—	—	—
2	7¾	6,500	251	14,558	—	377	1,052
3	9¼	—	—	—	—	—	—
4	10½	2,200	238	5,236	19,894	—	—
5	12¼	1,850	211	3,903	23,797	—	—
6	13½	1,900	189	3,591	27,388	—	—
7	14¾	3,050	170	5,185	32,573	—	—
8	16¼	1,500	161	2,415	34,988	—	—
9	17¾	1,000	141	1,410	36,390	—	—
10	18¾	850	119	1,011	37,401	—	—
11	20	2,450	108	2,646	40,047	—	—
12	21¼	4,000	96	3,840	43,887	170	3,387
13	22	2,000	72	1,440	45,327	—	—
14	22¾	2,300	75	1,725	47,052	—	—
15	23½	2,450	74.5	1,825	48,877	—	—
16	24½	2,400	63	1,512	50,389	125*	1,476
17	25	2,400	64	1,536	51,925	—	—
18	25¾	2,200	56	1,232	54,157	—	—
19	26½	2,100	50	1,050	55,207	—	—
20	27¼	2,200	42	924	56,131	—	—
21	27¾	1,800	43	774	56,905	—	—
22	28½	2,100	45	945	57,850	—	—
23	29½	2,050	35	718	58,568	—	—
24	30¼	2,000*	36	720	59,288	—	—
25	31¼	1,900	37	703	59,991	—	—
26	32	2,200	31	682	60,673	—	—
27	32¾	2,100	32	672	61,345	—	—
28	33¼	2,000*	31	620	61,965	—	—
29	34½	1,550	31	480	62,445	—	—
30	35½	2,100	27	576	63,021	—	—
31	36¼	1,800	24	432	63,453	41	1,260
32	37	2,750	24	660	64,113	—	—
33	37¾	1,750	19	332	64,445	—	—
34	38¾	1,800	21	378	64,823	—	—
35	39½	2,250	17	382	65,205	—	—
36	40½	2,000	21	420	65,625	—	—
37	41½	2,100	16	336	65,961†	—	—

* Extrapolated value.
† This is equivalent to about 80 cc of absolute methanol.

ethanol will effectively block the oxidation of methanol into its toxic products—formic acid and formaldehyde.[21-44] However, the biochemical literature is yet unclear as to whether alcohol dehydrogenase or a catalase-peroxidase pathway is the primary route of metabolism,[25-33,36] although recent evidence appears to favor the former route in primates.[45] It has been suggested that the acidosis may be the result of an uncoupling effect of formaldehyde or formic acid or both on the oxidative phosphorylation system[1,34] rather than a mere accumulation of acid metabolites.[10,13,43,44,46,47] Because of the experimental and clinical efficacy of ethanol in delaying methanol metabolism, together with its tendency to promote a water diuresis even in the face of dehydration,[48] we had hoped to use ethyl alcohol in our series. However as seen in Table 4, adequate blood levels of ethanol (100 mg/100 cc as described by Roe[7]) were obtained in only one patient after admission, due to inadequate loading doses.

Table 5.—*Course of Methanol and Ethanol in Blood and Various Routes of Excretion*

Case	Source	Methanol and Ethanol Levels (mg/100 cc)									
		Admis	12*	24	36	48	60	72	84	92	108
11	Blood	216 (0)†	135 (0)	97 (10)	72.5 (30.5)	—	6 (0)	—	—	—	—
	Urine		471	—	317	—	51	0	—	—	—
	Gastric		—	—	—	—	—	—	—	—	—
7	Blood	30	0	—	—	—	—	—	—	—	—
1	Blood	48 (346)	43 (35)	0 (0)	—	—	—	—	—	—	—
8	Blood	237 (0)	145 (54)	90 (35)	37.5 (5)	8.6 (0)	—	—	—	—	—
	Urine		204	—	124	—	—	—	—	—	—
9	Blood	246 (0)	199 (111)	183 143	145 (267)		111 (0)	40.5 (0)	14.6 (0)	0 (0)	—
	Urine		236	—	176	—	111	—	34	—	8
	Gastric		37	—	—	—	44	—	—	—	—
10	Blood	267 (0)	217 (93)	176 (0)	83 (0)	51 (29)	8 (0)	0 (0)	—	—	—
	Urine		270	214	87	34	0	—	—	—	—
	Gastric		—	27	—	31	—	—	—	—	—
2	Blood	218 (156)	201	138	68 (7)	—	18 (8)	—	0	—	—
	Urine		241	138	84	33	9	0	—	—	—
	Gastric		27	—	25	—	6	—	—	—	—
3	Blood	372 (118)	300	170 (10)	41 (11)	16 (9)	7 (3)	0 (0)	—	—	—
	Urine		261	146	36	15	7	3	—	—	—
	Gastric		57	—	—	—	—	—	—	—	—
5	Blood	77 (244)	34 (0)	9 (0)	—	—	—	—	—	—	—
4	Blood	82 (273)	57 (0)	8 (0)	—	—	—	—	—	—	—
6	Blood	110 (0)	74 (0)	24 (0)	—	4 (0)	—	—	—	—	—
	Urine		90	20		5‡	—	—	—	—	—

* Hours after admission.
† Ethyl alcohol levels are in parenthesis
‡ Isopropyl alcohol, 8 mg/100 cc.

The patients themselves offer an experiment in nature. Analysis of Table 2 shows that group A (the ethanol-protected group) had significantly less acidosis than group B who had no ethanol in their blood. In support of this protective effect of ethanol, the blood determinations from the deaths outside the University of Kentucky Medical Center showed no ethanol.

With the first clinical use of peritoneal dialysis by Stinebaugh in 1959[10] and the subsequent application of Schreiner's criteria for hemodialyzable poisons[10,49,50] to the treatment of methyl alcohol poisoning, a new era of treatment by hemodialysis was begun.[5,51-55] Of these the largest series was from patients with serum methanol levels of 194, 275, 277, and 860 mg/100 cc. Of these, only one survived.[52]

Although the role of hemodialysis had been firmly established, at the time of the methanol epidemic the hemodialysis facilities of the university hospital were committed to chronic renal patients awaiting kidney transplantation. Any dialysis therapy had to be peritoneal.

With the exception of our original patient (case 11) all of our patients were virtually asymptomatic (Table 2). The decision to institute dialysis was therefore based on the serum methanol levels, with two exceptions. One patient (case 10) refused dialysis and in another (case 9) dialysis was not begun until complications in acid-base management developed.

Figures 1 and 2 compare the rates of methanol disappearance from the serum of the dialyzed and nondialyzed patients. In Fig 3 the average rates of disappearance for the dialyzed and nondialyzed groups are juxtaposed against the rate of disappearance in patient 9 who demonstrated both rates during this clinical course.

Table 5 represents a record of each dialysis exchange on patient 3.

Clearance rates have been calculated with available serum values or extrapolations. When compared to the urinary clearances shown in Table 6, it can be seen that peritoneal dialysis was five to ten times as effective in removing methanol.

Since visual disturbances are so much a part of the clinical picture of methanol poisoning, it was very surprising to note the low incidence of visual complications in our series. Of the 11 patients hospitalized, only four had any visual symptoms, of which only one (case 11) resulted in fixed dilated pupils and funduscopic changes. In light of

the theory by Potts et al,[42] there was no correlation between presence of visual symptoms and the degree of acidosis on admission to suggest a failure to reach at least the second stage of the disease. Serum methanol values were well in excess of those frequently associated with blindness and death. Ethanol levels were, if anything, higher in several of those patients with visual symptoms.

With the exception of the many individual cases cited by Wood and Buller,[11] the epidemics of methanol poisoning previously described did not occur among habitual "heads" drinkers. It is possible that our group represents some sort of acquired tolerance to the ophthalmologic toxicity of methanol (note the case described by Shinaberger[55] of a chronic Sterno drinker who suffered no visual symptoms despite severe acidosis, semicoma, and hypotension).

It was of particular interest that the patients' symptomatology, as outlined in Table 2, bore little resemblance to the classical picture of methanol poisoning. Many of those screened who were not symptomatic had no methanol in their blood and most of those with methanol in their blood were asymptomatic. This accentuates the need for methanol determinations to identify victims independent of historical and clinical information if treatment is to be offered at the earliest possible moment.

Bennett[1] noted that the methanol concentration of spinal fluid generally exceeded that of simultaneously collected blood. Tonning[9] found elevated spinal fluid methanol levels in 11 of the 14 patients measured on their first hospital day but did not correlate these findings with blood levels. In the nine patients whose spinal fluids were studied, the methanol and ethanol levels were comparable to those levels of simultaneously obtained blood (Table 8).

Table 6.—*Urinary Excretion of Methanol*

Case	Time (hr)	Volume (ml)	Methanol Conc (mg/100 ml)	Methanol Content (mg)	Total Methanol Excretion	Clearance Rate (ml/hr)
3	11-35	2,250	261	5,003		
		2,425	146	3,541		255
		2,200	86	1,892	10,436	
	36-59	3,275	36	1,179		
		6,600	15	990	2,169	570
	60-83	2,000	7	140		
		2,000	4	80		—
		2,000	3	60		
		2,700	0	—	280	
					12,885	
2	4-11	500	241	1,210	1,210	76
	12-35	2,000	178	3,560		
		2,000	138	2,760		
		2,000	107	2,140		309
		500	84	420	8,880	
	36-59	1,500	58	870		
		3,900	33	1,287	2,157	—
	60-83	1,900	18	342		
		2,000	9	180		
		3,800	3	114		—
		475	0	—	636	
					12,283	
9	0-11	2,400	226	5,424		
	11-35	2,300	176	4,048		
	36-59	3,850	111	4,244		—
	60-83	2,075	34	706		
	89-107	2,325	8	186	14,608	
10	0-6	1,625	270	4,388	4,388	293
	7-30	2,000	258	5,160		
		1,900	214	4,066		
		2,000	168	3,360		356
		1,350	128	1,228	13,814	
	31-54 (urea)	1,200	87	1,044		
		1,500	60	900		221
		4,400	34	1,496	3,440	
	55-78	2,000	5	60		
		1,900	0		60	—
					21,702	
6	0-6	300	150	450		76
	7-18	3,100	90	2,790		310
	19-42	6,950	20	1,390		232
	43-52	600	5	180		
					4,810	
8	0-11	1,300	204	2,652		
	12-35	625	124	775		—
					3,427	

Conclusions

Although peritoneal dialysis is not as rapid as hemodialysis in removing methanol and its toxic products, it is much more available to the practicing physician in virtually

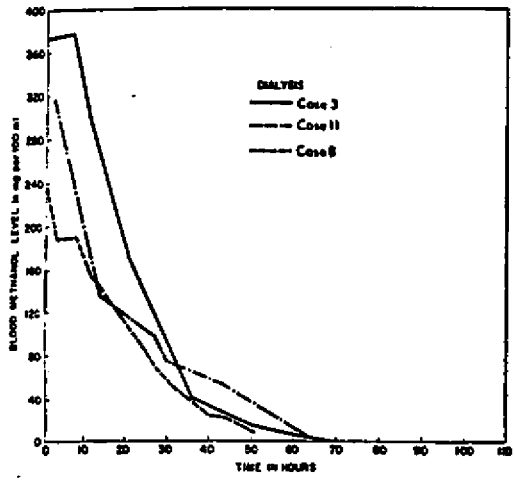

Fig 2.—Rate of disappearance of methanol from the blood of those patients treated by peritoneal dialysis.

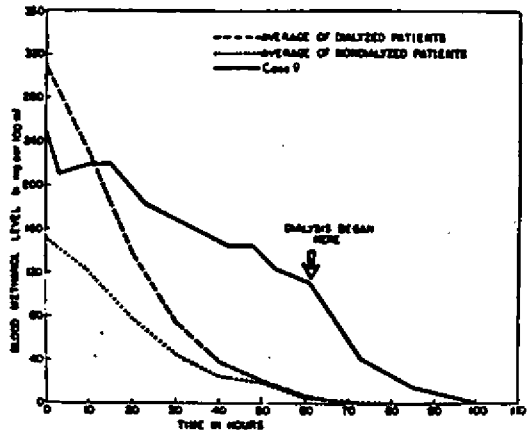

Fig 3.—Comparison of the average rates of methanol disappearance in the dialyzed and nondialyzed patients contrasted with the course of patient 9.

Table 7.—*Liver Function Tests, Amylase, and Glucose Determinations of Patients Admitted for Methanolism**

Case	SGOT*	SGPT	Alk Phos	Bilirubin (Tot/Dir, mg/100 cc)	Amylase	Fasting Glucose on Admis (mg 100 ml)
11	280	140	5.8	1.0/0.5	352	144
7	22	14	3.2	1.2/0.2	—	125
1	172	44	5.0	0.6/0.1	32	104
8	84	21	2.6	0.5/0.1	128	129
9	36	16	4.2	0.6/0.1	48	98 (diabetic)
10	48	8	5.8	0.4/0.1	120	134
2	74	21	4.2		72	86
3	480	160	4.2	0.7/0.2	16	130
4	55	16	5.4	0.5	120	90
5	95	74	5.8	0.5/0.1	—	66
6	48	20	3.8	0.7/0.1	72	—

* Serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) measured in Reitman-Frankel units. Alkaline phosphatase measured in Bessey-Lowry-Brock units. Amylase measured in Somogyi units.

Table 8.—*Spinal Fluid Values With Comparable Blood Values*

	Spinal Fluid					Blood		
Case	Cells	CSF Prot	Glucose	Meth	Eth	Glucose	Meth	Eth
11*	2 WBC	47	76	—	—	144	316	—
	—	—	—	154	0	—	72.5	30.5
7	3 RBC 1 WBC	18	74	18	0	125	10	0
3	9 RBC	20	77	351	129	130	372	118
1	1 RBC	22	74	48.5	310	104	48	346
6	10 RBC 0 WBC	20	86	107	0	—	95	0
10	270 RBC†	15	70	212	64	—	217	93
5	0	32	66	36	20	—	37.5	7
9	0	16	68	262	0	98	210	23
8	0	22	64	232	28	129	188	24

* Upper figures indicate values upon admission; lower figures indicate values 21 hours after admission.
† Traumatic lumbar puncture.

Table 9.—*Deaths Outside UKMC*

Age, Sex, Race	Blood Methanol	Ethanol	Autopsy	Misc
48, F, W	632	0	No	—
44, M, N	—	—	No	Dispenser of "heads"
57, M, W	—	—	No	Found surrounded by empty bottles
34, M, W	415	0	No	Comatose seizures, fixed dilated pupils
61, M, N	310	0	Yes	Stuporous, comatose, fixed dilated pupils
44, M, W	310	0	Yes	Minimal pericholangitis, focal areas of lipid depletion in adrenals
46, M, W	240	0	No	Bottle found beside him 31 vol % methanol

any medical institution. With the technical advances in clinical toxicology it is now possible to obtain blood methanol determinations rapidly, thus enabling the clinician to assess the patient's present condition and prognosis in light of acid-base status and methanol levels. Many instances like those described may be found which will add a new dimension to our understanding of methanol poisoning, namely the clinically asymptomatic case which will readily respond to peritoneal dialysis.

We feel that several of the patients described in this report represent a new syndrome in the lore of methanol poisoning—specifically, asymptomatic persons with high serum levels of methanol who have been discovered and treated before the terrible effects of the poison could become manifest. As more of these epidemics are described at a time when technical developments make possible rapid blood methanol determinations, more of the answers to the mysteries of the human form of this very species of specific disease may become clear.

We would recommend that future investigators consider the use of compounds such as Tris-buffer or urea, which may serve a dual role either as a buffer-binder or diuretic-binder, respectively. Utilizing modern laboratory facilities, detailed analyses of patients' urine may yield valuable information about methanol metabolism.

Summary

An epidemic of 18 persons who drank methanol is described, including several patients who represent a new dimension of asymptomatic methanol poisoning treated by peritoneal dialysis. Various parameters of methanol excretion and treatment are discussed. The literature on biochemical experimental and therapeutic work in methanol poisoning is reviewed.

Generic and Trade Names of Drugs

Pentobarbital—*Nembutal.*
Mannitol—*Osmitrol.*

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