

\* Spinal cords 86  
\* \* Brown Pigment (stuffed) dehorns in medulla oblongata (85)

1935  
heart - many enlarged (according to me) 80 right ventricular dilation 90  
Lungs - all emphysema, ~~hyper~~ hyper, mucous membrane red 81, 88  
Liver - all fatty 81 color yellowish Brown 89  
Spleen - some enlarged 81

ACUTE METHYL ALCOHOL POISONING

All but three were embalmed before postmortem  
retinas - 88

REPORT OF TWENTY-TWO INSTANCES WITH POSTMORTEM EXAMINATIONS

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nerve 15 88

Dec. 7, 1934, the coroner's office of Multnomah County, Ore., was called on to investigate the death of a man who died from so-called denatured or "dehorn" alcohol poisoning. The death of this man was followed in dramatic and rapid succession by the deaths of twenty-one others in the two succeeding days. All of these victims were known to law enforcement officers as having an addiction to alcohol and were commonly referred to as "dehorns"; their family connections were unknown or distant; they lived in cheap rooming houses, worked occasionally and were incarcerated from time to time for drunkenness and vagrancy but otherwise were harmless. Several of the group were wayward members of prominent families. Their ages ranged from 32 to 65, six being in the third, five in the fourth, nine in the fifth and two in the sixth decade of life. Fourteen died after a short time at the Good Samaritan Hospital (attended by Dr. Fred Ziegler), and one, in the Multnomah County Hospital. Five were found dead in hotel rooms and one in a barn. One died in an unknown place in a neighboring city. Their renegade character, their furtive indulgence in their vice and their isolation from interested relatives and friends obscured many of the details of the final episodes that ended the lives of these unfortunate men.

The available clinical data disclosed only some of the terminal signs and symptoms observed during the brief hospitalization of fourteen of the patients and the details of the illness of another who died at home.

Of the fourteen who were hospitalized, five were conscious on arrival, while a similar number were in deep coma; the rest were semicomatose. Most of them were in profound shock, being cold, clammy, pale and perspiring excessively. It was frequently stated that their clothing or the bed clothing was "soaked with perspiration." Three were sufficiently rational to relate some of their subjective symptoms. In the several instances in which it was possible to record the blood pressure, the systolic readings ranged from 110 to 140 and the diastolic readings from 78 to 90, suggesting an increase of pressure

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\* nerve tissue (91)  
muscle paralysis, 88

Intense craving and decided preference for denatured alcohol (90-91)

due to terminal asphyxia as death approached. Their initial pallor was overshadowed by marked cyanosis. The eyes were occasionally rolled upward but in general were fixed; the pupils were dilated, with either slight or no reaction to light and accommodation. Several complained

TABLE 1.—Clinical Data Concerning Patients

Case	Mental Condition	Blood Pressure		Condition of Skin			Eyes			Respiration	
		Sys-tolic	Diastolic	Temper-ature	Pallor	Sweat-ing	Pupils	Reac-tion to Light and Accom-modation	Condi-tion	Rate	Lungs
1	Unconscious, irrational	140	90	Cold, clammy	4+	4+	7 mm.	Neg.	Fixed, rolled upward	8	Resonant
2	Semiconscious, irrational, complaining of pain in back	?	?	Cold, clammy	4+	4+	4 mm.	Slight	Rolling	18 decreasing	Resonant
3	Semiconscious, irrational, moribund	120	80	Cold, (shock)	4+	4+	Dilated	Neg.	Fixed	15 decreasing	Resonant
4	Comatose, moribund	?	?	Cold, clammy	3+	4+	7 mm.	Neg.	Fixed	12 to 4	Resonant, rales
5	Comatose, irrational	?	?	Cold, clammy	+	4+	Dilated	Neg.	Fixed	Labored, slow	Rales, mucus
6	Comatose, irrational	?	?	Cold, clammy	+	4+	Dilated	Neg.	Fixed	6	Rales, mucus
7	Comatose, irrational	?	?	Cold, clammy	+	4+	Dilated	Neg.	Fixed	8	?
8	Conscious, rational	136	80	Cold, clammy	4+	4+	5 mm.	Neg.	Fixed	20 to 3	Resonant
9	Comatose, irrational	?	?	Cold, clammy (shock)	4+	4+	Dilated	Neg.	Fixed	12 decreasing	Resonant
10	Conscious, rational, complaining of pain	?	?	Cold, clammy	4+	4+	Dilated	Neg.	Fixed	10 to 3	Resonant
11	Semiconscious, irrational	136	86	Cold, clammy	4+	4+	7 mm.	Neg.	Round, fixed	? regular	Resonant
12	Conscious, irrational	134	90	Cold, clammy	4+	4+	5 mm.	Slight	Fixed	12 decreasing	Resonant
13	Semiconscious, irrational	100	60	Warm, moist (97 F.)	Slight	Slight	Dilated	Slight	Nystagmus	40 decreasing	Resonant rales
14	Conscious, going on to comatose, irrational	?	?	Cold, clammy	4+	4+	Dilated	Neg.	Fixed	12 to 2	Resonant
15	Conscious, rational	110	78	Warm, moist	Slight	Slight	5 mm.	Slight	Dim vision	24	Resonant
1st admis.											
2d admis.	Semiconscious, irrational	?	?	Cold, clammy	4+	4+	7 mm.	Neg.	Fixed	26	Resonant

of dimness of vision, while most of them were too moribund to comment on such disturbances.

Marked irregularities of respiration were noted in all. The breathing was irregular, labored and often spasmodic. The rates varied greatly, being at times increased, slowed or normal, but usually diminishing to only 2 or 3 respirations per minute, and terminating in respiratory failure.

The heart rates varied considerably. Many of the patients were pulseless on their arrival at the hospitals. In a number of others the heart was beating rapidly. In most of them, however, there was a slowing comparable to that in their respiration although not always

*Dying from Acute Methyl Alcohol Poisoning*

Heart		Cyanosis	Reflexes in General	Abdominal Pain	Sensitiveness	Convulsions	Duration in Hospital	Duration After Drinking	Amount of Liquor Consumed, Ce.	Respiratory Failure
Rate	Action									
?	Feeble, distant	4+	Negative	?	3+	0	47 min.	10 hr.	400	+
80 decreasing	Clear	4-	Active (Babinski positive, bilateral)	Rigidity	3+	Clonic, later tonic	70 min.	10 hr. ?	?	+
72	Regular	4-	Normal	0	0	0	50 min.	12 hr. ?	500	+
?	Regular	4-	Absent	?	?	0	40 min.	24 hr. ?	500	+
?	Feeble	4+	Absent	?	?	0	5 min.	24 hr. ?	?	+
Pulseless	?	4-	Absent	?	?	0	5 min.	24 hr. ?	130	+
Pulseless	?	4+	Absent	0	0	0	2 min.	24 hr. ?	400 ?	+
30 to 52	Regular	4+	Slight, slowed	4+	4+	2+	2 hr.	24 hr.	1,000 with friends	+
?	Regular	4+	?	?	0	?	2 hr.	24 hr. ?	200 ?	+
Rapid	Regular, weak	4+	?	4-	4+	0	1 hr., 30 min.	24 hr. ?	?	+
?	Regular	4+	Absent	?	?	+	3 hr., 15 min.	24 to 30 hr. ?	?	Heart +
45	Regular	4+	?	4+	4+	4+	1 hr., 40 min.	24 to 48 hr. ?	?	+
Regular	Strong	4+	Hyperactive	Babinski +	0	Present	9 hr.	27 hr. ?	?	+
108 to 120	Rapid	4+	Absent	4+	4+	0	1 hr., 40 min.	24 to 48 hr. ?	?	+
116	Regular	None	Normal	4+	4+	0	7 hr.	7 hr.	?	None
100	Rapid	4+	Absent	?	?	0	6 hr.	?	?	+

coincident with it. The tones were clear and regular, finally becoming distant and feeble. It is evident from the brief clinical notes that cardiac arrest followed rather closely in the wake of respiratory failure.

The extremely urgent conditions of the patients made examination of the body reflexes inaccurate. In only one was a positive bilateral Babinski sign recorded; in two, the reflexes were normal; in many, absent. In six of the patients there was evidence of marked abdominal

TABLE 2.—*Postmortem Findings in Twenty-Two*

Patient	Number	Age, Yr.	Weight of Patient, Lb.	Weight of Brain, Gm.	Weight of Heart, Gm.	Coronary Disease*	Arterio-sclerosis	Weight of Lungs, Gm.
L. M.	1	52	Obese	Normal	<u>580</u>	None	None	885-700
A. R.	2	48	165	1,370	375	Slight sclerosis	Slight generalized	Light
E. N.	3	39	175	1,580	<u>480</u>	Narrowing 3+	2+	510-460
E. W.	4	52	150	1,220	390	Sclerosis 2+, endocarditis	None	?
C. M.	5	57	160	1,420	880	3+	2+	350-340
B. V.	6	46	150 (fair nutrition)	1,495	not increased	Sclerosis 1+	None	?
R. H.	7	54	135	1,690	370	No sclerosis	None in aorta	580-560
O. O.	8	52	180	1,450	340	None	None	?
C. S.	9	33	180	1,520	<u>500</u>	None	1+	620-510
J. S. (A)	10	45	200 †	1,590	370	None	1+	?
M. S.	11	41	Well nourished	1,690	<u>460</u>	None	None	?
C. F.	12	65	135	1,790	370	None	1+	630-465
J. H.	13	50	150	1,600	<u>410</u>	None	1+	?
B. P.	14	54	175	1,495	<u>430</u>	Focal narrowing 2+	2+	740
J. J.	15	58	Emaciated	Not examined	330	None	1+	550
J. B.	16	34	Well nourished	Not examined	<u>440</u>	1+	1+	390-390
M. K.	17	60	Emaciated, 145	1,620	<u>410</u>	1+	1+	700-550
A. J.	18	32	165	1,300	<u>540</u>	None	None	760-490
C. T.	19	42	230	Heavy	380	None	None	970-830
L. B.	20	38	180	Not examined	350	None	None	Normal
W. L.	21	58	Moderately well nourished	Heavy	<u>410</u>	None	None	830-770
J. A.	G.S.H. 8-1537	32	160 (good nutrition)	Heavy	320	Soft, pliable and patent throughout; heart muscle unchanged	None	200-250

\* In this and subsequent columns 1+ means slight; 2+ and 3+, moderate, and 4+, marked.

*Men Dying of Acute Methyl Alcohol Poisoning*

Condition of Lungs	Weight and Condition of Liver	Condition of Stomach	Condition of Intestines	Weight of Kidneys, Gm.	Weight of Spleen, Gm.
Ant. emphysema, post. hyperemia	Large, fatty	Hyperemia, mucus	Hyperemia in upper portion	434	95
Ant. emphysema, post. hyperemia	Fatty	Blood-streaked mucus, small amount food	Empty	400	60
Emphysema	Fatty 2+ 2,360 Gm.	350 cc. mucoid material and food; edema, hyperemia	Edema 2+, hyperemia, dilatation +	365	110
Emphysema 4+, edema 2+	Cirrhosis 2+	500 cc. food; hyperemia, petechial hemorrhage	Edema 3+, hyperemia 3+, gas 3+	340	185
Emphysema, hypostatic hyperemia	Hyperemia 1,700 Gm.	Hyperemia; thick mucoid material	Hyperemia, edema	280	110
Emphysema 4+, hypostatic edema	Fatty 2+, hyperemia 3+	Mucous material	Hyperemia, edema	Normal	Normal
Ant. emphysema 4+, hypostatic hyperemia 2+	Fatty 3+, hyperemia 3+, 1,500 Gm.	Hyperemia 3+, edema, mucoid material 3+	Hyperemia, edema	390	250
Ant. emphysema 4+, hypostatic hyperemia 2+	Fatty 2+, hyperemia 2+	Hyperemia 4+, mucus 4+	Hyperemia, edema	Normal	Normal
Emphysema 2+, hypostatic hyperemia 3+	Fatty 2+, hyperemia 3+ 1,440 Gm.	200 cc. bloody mucoid material	Hyperemia, edema	Normal	Normal
Emphysema 3+, hypostatic edema 2+	Fatty 2+, hyperemia 2+	Hyperemia 3+, edema, bloody mucus 3+	Hyperemia, edema	485	185
Emphysema 4+	Fatty 1+, hyperemia 2+	Hemorrhage 3+, large amount of food material	Hyperemia, edema	460	220
Emphysema 2+, hypostatic hyperemia 2+	Fatty 1+, hyperemia 3+	Hyperemia, edema	Hyperemia, edema	440	135
Emphysema 4+	Fatty 2+, hyperemia 3+, edema	Hyperemia 1+, distention 3+	Hyperemia, edema, distention	Normal	Normal
Emphysema 3+, hypostatic hyperemia 2+	Fatty 2+, hyperemia 2+	Hyperemia 2+, edema 2+, mucous material 2+	Hyperemia, edema	450	95
Emphysema 2+, hypostatic hyperemia 2+	Hyperemia 3+, fatty 1+	Hyperemia, 500 cc. bloody mucus	Hyperemia, edema	440	80
Emphysema 2+, hypostatic hyperemia 2+	Fatty 3+, hyperemia 1+	Hyperemia, edema	Hyperemia, edema	440	Normal
Emphysema 1+, hypostatic edema 3+	Fatty 2+, hyperemia 2+	Hyperemia	Hyperemia, edema	?	180
Emphysema 3+, hypostatic edema 2+	Fatty 3+, hyperemia 2+	100 cc. bloody mucus, hyperemia, edema	Hyperemia, edema	440	140
Emphysema	1,840 Gm.	Marked hyperemia, edema	Hyperemia, edema	460	130
Emphysema, hyperemia	Fatty, increased in size	Mucoid matter; walls congested, red	Hyperemia, edema	Normal	Normal
Emphysema, hyperemia	1,840 Gm.	Hyperemia, edema	Hyperemia, edema	380	140
Lungs fluff, collapsed; bronchial mucous membrane red	Fatty, nonciclotic 2,800 Gm.	100-150 cc. greenish watery fluid, hyperemia	Marked hyperemia, edema	555	150

pain and tenderness. Terminal convulsions were present in three. These variable sensations may have been altered because of paralyses due to terminal intoxication.

There is no way of knowing exactly how much of the alcohol each one consumed. Scattered notations indicate that much of the drinking was done in groups. In a few instances a single empty pint bottle was found in the bed room containing the dead body. It may be assumed, therefore, that in some cases at least as much as 500 cc. of alcoholic liquor was drunk by one man. It is also difficult to arrive at any conclusion as to the exact time of the consumption of the poisonous liquor with reference to the onset of the symptoms. In one or two cases, however, there were definite statements indicating that symptoms developed in from sixteen to twenty-four hours after the intake. It is doubtless true that in many of the cases the effect was felt much earlier.

The sketchy nature of the clinical observations is mainly due to the brevity of the hospitalization. Six patients died within an hour; three within five minutes, and the remainder within from one to seven hours. The following more detailed citations of events in a few instances will serve to portray better the probable specific clinical manifestations:

#### CASE HISTORIES

M. S. was admitted to the hospital complaining of diffuse abdominal pain, exhibiting general discomfort and sensing impending death. He stated that with some companions, on the afternoon of Dec. 7, 1934, he drank one or two pint bottles of what was believed to be denatured alcohol. He had no symptoms until the following morning, when he noted a feeling of uneasiness followed by occasional brief but recurrent violently cramping abdominal pains. He was covered with profuse clammy perspiration, which soaked his clothing. He improved slightly after treatment, but later his respiration became slow (3 per minute) and his pulse imperceptible. Terminally, there was convulsive twitching of the face with progressive marked cyanosis and respiratory failure, resulting in death two hours after his admission.

A. J., on admission to the hospital, stated that he had consumed "dehorn" at intervals for two years. Although he was somewhat mentally unstable, he complained of general cramping pains and disturbance in vision. He stated that he partook of portions of two bottles of "dehorn." His pupils were round (5 mm.), equal and reactive only slightly to light and accommodation. The skin was cold and covered with clammy perspiration. Just prior to death a generalized spasm developed. Complete cessation of respiration was noted, then sudden relaxation and death three hours and forty minutes after admission.

B. P., aged 54, a longshoreman divorced, had been known as a periodic drinker and user of denatured alcohol for at least five years. His mother stated that on Saturday, Dec. 8, 1934, he awakened at 8 a. m. She observed that he had been drinking. He asked for his breakfast, which she prepared. She then left for about an hour and on returning found her son sitting quietly at the table with his breakfast untouched. He said he had no pain but "couldn't see very well." About thirty minutes later he went back to bed. He awakened at 2 p. m. and asked

the time, dressed unassisted, spoke normally and with only a slightly wobbly gait left for downtown, complaining that he would be late. In about an hour he was brought back in a delivery truck. He had collapsed on a street corner but was still able to give the necessary directions for being brought home. He was assisted to bed. He removed his jacket carefully, then suddenly pawed and tore his shirt and underclothing from his chest and cried out for air. At the same time he complained of severe, steady, burning pain in the upper part of the abdomen, which caused him to press his abdomen firmly with both hands. The pain and the hunger for air were so intense that he worked himself off the bed and turned over several times on the floor. There was also an unquenchable thirst. He took two glasses of water soon after he was brought home and in the next several hours consumed from seven to ten glasses of water containing sodium bicarbonate.

The difficulty in respiration seemed to disappear at times (for five minutes or longer) only to recur again. The blurring of vision persisted until death. At 5 o'clock his speech became incoherent and rambling; he stopped complaining of pain; his breathing became increasingly difficult; cyanosis was marked, and approximately ten hours after the onset of symptoms he died. Many times in previous years the patient had been seen violently ill as a result of his consumption of denatured alcohol; hence no physician was called.

J. A. was admitted to the hospital Dec. 8, 1934, stating that he had been in good health until he took denatured (?) alcohol ("cut with water") on the day before admission. He said that about six hours after drinking the liquor he began to have severe abdominal pain. His pupils reacted sluggishly to light and accommodation, and except for the presence of abdominal pain he appeared in fair condition. His respiratory rate was 24 and his pulse rate 116. Although when he came in he was at first somewhat irrational, he improved and left the hospital after seven hours. About sixteen hours later he was readmitted, having been in the meantime in the emergency hospital of the city jail. It could not be learned whether or not he had had more liquor in the interim. On the second admission his pupils were found dilated, with only sluggish reaction to light. He was almost totally blind. Later in the afternoon (about five hours after admission) his breathing became labored, his pulse weak, and he anxiously gasped for breath. His eyes finally became fixed; his carotid pulse rate was 100; the respiration was 26; cyanosis was marked; he stopped breathing approximately thirty-two hours after drinking the alcohol.

Practically none of the details of these histories were available at the time of the necropsies.

#### POSTMORTEM EXAMINATIONS

Twenty-one of the bodies were examined by the staff of the department of pathology (coroner's physicians) and one by Dr. C. H. Manlove, of the Good Samaritan Hospital. The excitement of the emergency and the scattering of the bodies in various undertaking parlors so occupied the coroner's staff (Dr. R. Edwin, coroner) that the request for postmortem examinations was not made until the second day of the incident and not until most of the bodies (all but three) were embalmed.

Many of the bodies were found in a good state of nourishment; only two were emaciated (table 1). The weights ranged from 135 to 200 pounds (61 to 91 Kg.). emaciated. The weights ranged from 135 to 200 pounds (61 to 91 Kg.).

Except for the terminal cyanosis, there was commonly seen in these addicts a general pasty pallor of the skin. The hands and feet were dirty, the palms of the hands were slightly workworn, and the hair on the head was unkempt—all attesting to the shiftless indifference of character of these men. In many, the facial expression was agonized. There were some with fresh bruises and lacerations of the face and extremities. Lividity was posterior or lateral, and rigor mortis was in general intensified. One found dead in a barn died lying on his right side. His head was extended; the conjunctivae were suffused; the trunk was distorted and twisted; the hands were clenched with marked extension at the wrists; the lower extremities were flexed at the knees and the hips; the testes were drawn up into the inguinal rings; all this indicated intensive terminal abdominal pain. Such evidences may have been present in all of the bodies and then altered by hospitalization and embalming.

The following pathologic changes were observed in the different organs:

Brains.—All but four of the brains were removed, strenuous objection being offered by relatives in two instances. When the calvariums were taken off, varying degrees of tenseness of the duras were observed. In the several bodies that were not previously embalmed there was marked engorgement of the circulatory system in both the dura and the pia-arachnoid. In many instances the latter was lifted high above the convolutions by clear spinal fluid. In others the sub-arachnoid spaces were almost obliterated by swelling of the parenchyma. The weights of the brains (after fixation in solution of formaldehyde U. S. P.) were in general increased, ranging from 1,290 to 1,790 Gm.

Most of the brains were pale and wet. Variations in the amount of fluid in the subarachnoid spaces seemed to depend on the degrees of swelling of the brain substances. In practically every one there was some herniation of the cerebellum into the foramen magna. Chronic meningitis was not observed; the membranes were thin. In only one brain was there evidence of a preexisting old cerebral hemorrhage.

In the microscopic examinations the different brains disclosed relative uniformity of appearance. Variations were due to different degrees of edema and hyperemia. There were no old or recent hemorrhages in the dura or in the pia-arachnoid, but in the latter there were small fibrous patches and spreading edema. The optic chiasms disclosed occasional small punctate hemorrhages with, infrequently, other more minute interstitial extravasations of red blood cells. No decisive, disrupting hemorrhages were found here. There was no edematous spreading of optic nerve fibers. If anything, the latter appeared more compact, as though from intrinsic swelling. In the regions of the precentral and postcentral gyri there was moderate to marked subpial edema, with engorgement of small cortical blood vessels and perivascular edema, especially in the subcortical zones, where larger edematous vacuoles were often seen. The edema appeared to extend along the blood vessels, with slight to moderate degrees of perineuronal extension. Nissl granules were often found to be powdery, ragged and frayed, with occasional slight indentation of the cells. In addition, there was some slight satellitosis. Other changes, such as cytoplasmic accumulation of lipochrome (younger brains) and presence of calcospherites in white matter (older brains), were observed. Similar but less marked changes were found in the calcarine convolutions. Sections through the uncus in the different brains revealed in one instance, in addition

to the extension of edema already described in other locations, an old area of hemorrhage (recognized clinically) with amyloid cytoid bodies, powdery granules of hemosiderin and many pyknotic nuclei (glial). In the older brains senile lipochromes were more abundant. In the neurons there were edematous perinuclear clear spaces; there was some incompleteness of nuclear membranes, with a tendency to eccentric position of the nuclei. In some of the brains there was marked satellitosis with perivascular rows of mononuclear cells in the adventitia (one brain). No striking changes were seen in the basal ganglions; small patches of gliosis with slight to moderate edema and a sprinkling of lipochromes in some cells were noted.

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Brown  
pigment  
(stuffed)  
neurons  
of  
the  
medulla

Similarly, the medullae oblongatae disclosed varying edema, often marked accumulation of brown pigment in most of the neurons (so that they appeared stuffed) and occasional bloating and desquamation of the ependyma of the medial portion of the ventricular floor. There was no demonstrable alteration in the Nissl granules. In sections of the cerebellum nothing but edema was seen.

The minute alterations observed in the central nervous system (brain and medulla oblongata) consisted, therefore, of marked subpial and moderate cortical and subcortical interstitial edema with spotty perivascular and perineuronal extension. Only occasional minute focal hemorrhages were noted. On the whole, the cellular changes were not marked. The "extensive degeneration of ganglion cells and vascular endothelium with subsequent hemorrhage in the midbrain, pons and medulla oblongata" referred to by Weil<sup>1</sup> was not observed, probably because of the acuteness of the intoxication. In a report concerning six deaths among thirty persons with acute wood alcohol poisoning, Gettler and St. George<sup>2</sup> noted only pronounced cerebral congestion with an increase of spinal fluid and engorgement of blood vessels. Detailed studies of tissues from the human central nervous system in methyl alcohol poisoning are wanting, since many of the patients recovered, and in others microscopic investigations were not made. However, in the brains of rabbits forced to inhale 0.2 per cent wood alcohol for periods of two, four, six, eight and ten months, Eisenberg<sup>3</sup> found degeneration of varying degrees, with an indefinite line of demarcation between the gray and the white matter, diminution of neurocytes with spindling and disappearance of Nissl granules, and a scattering of brownish pigment. In the later stages of the severe intoxications in these rabbits there was marked decrease in the size and number of parenchymal cells. The nuclear changes varied from peripheral wandering to complete karyolysis. Scott and his associates<sup>4</sup> exposed monkeys, rabbits and rats to methyl alcohol by cutaneous absorption,

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1. Weil, A.: Textbook of Neuropathology, Philadelphia, Lea & Febiger, 1933, p. 191.

2. Gettler, A. O., and St. George, A. V.: J. A. M. A. 70:144, 1918.

3. Eisenberg, A.: Am. J. Pub. Health 7:765, 1917.

4. Scott, E.; Helz, M. K., and McCord, C. P.: Am. J. Clin. Path. 3:311, 1933.

inhalation and ingestion. They found in their animals capillary congestion, edema and patchy degeneration of the neurons. These changes were more often found in the spinal cords than in the brains. These authors quote Rühle as having found in dogs scattered hemorrhages along the blood vessels of the pons, medulla and cord, as well as large amounts of lipoid in the vascular endothelium and perivascular tissue. The deposition of lipoid often preceded the hemorrhage. Scott and his co-workers concluded that only parenchymal and neuronal tissues were affected. Such experimental evidence is probably of more value in depicting the injurious effect of methyl alcohol on the central nervous

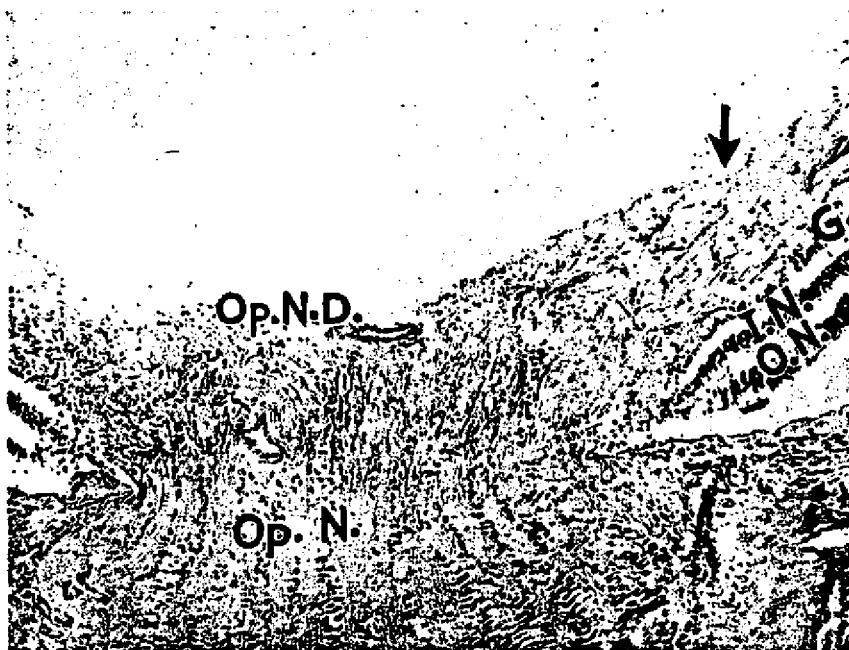


Fig. 1.—Photomicrograph of one of the eyes, showing obliteration of the "optic cup" at *Op. N.D.* *Op.N.* indicates the optic nerve; *G.*, the ganglion cell layer of the retina; *I.N.*, the inner nuclear layer, and *O.N.*, the outer nuclear layer. The arrow indicates the region of the magnification shown in figure 2.

system than are the changes observed in such acute conditions as I have described in human beings, material which is not so accurately controlled. However, the susceptibility of the tissues of animals to wood alcohol must be considered in the evaluation, since there is such wide variation in the effects in the different animals studied. The derivatives of formic acid and the alcohol itself may becloud the real changes in the parenchyma of the central nervous system because of their fixative action.

