TABLE III.
Organ Weights of Rats Fed Various Nitrogen Sources.
Wet weights per 100 g of body weight. The results are averages obtained on 10 rats.

Group	Liver, g	Kidney, g	Adrenals, mg	Thyroid, mg	Testes, g	Seminal vesicles, g
I	2.66 ± .11	.617 ± .012	10.1	3.95 ± .26	.955	.310
II	3.15 ± .07	.856 ± .023	12.6	4.66 ± .19	1.07	.264
III	2.96 ± .05	.632 ± .014	8.8	4.01 ± .18	.912	.266
IV	2.91 ± .15	.707 ± .011	9.5	4.04 ± .07	.926	.312
V	2.79 ± .13	.788 ± .036	9.9	3.99 ± .10	.977	.228
VI	2.32 ± .11	.625 ± .016	9.1	3.88 ± .22	.960	.313

TABLE IV.
Water and Nitrogen Contents of Livers and Kidneys of Rats Fed Various Nitrogen Sources.
Averages on 10 rats.

	Water		Dry wt		Nitrogen		Total nitrogen	
Group	Liver %	Kidney %	Liver g/100 g B.W.	Kidney g/100 g B.W.	Liver %	Kidney %	Liver g/100 g B.W.	Kidney g/100 g B.W.
I	70.8	76.1	.815	.146	10.28	11.63	.084	.0169
II	70.9	76.4	.915	.201	10.23	11.61	.094	.0234
III	71.3	76.0	.846	.151	10.12	11.55	.086	.0174
IV	71.9	75.8	.794	.171	10.42	11.34	.083	.0194
V	70.3	76.9	.829	.181	10.53	11.16	.087	.0202
VI	70.7	76.0	.675	.149	10.86	11.29	.073	.0168

4.8% glycine plus 1.7% L-arginine to a casein diet containing excess methionine counteracted the weight loss and in part the kidney hypertrophy caused by the excess methionine. The slight increases in thyroid weights, associated with excess methionine, were also antagonized by glycine and arginine. Under the conditions of these experiments urinary creatinine and creatine excretion was not increased in rats by feeding excess glycine. The significance of these results is discussed.

16917

Therapeutic Effect of Choline Chloride in Dogs with Fat Emboli Produced by Bone Marrow Curettage.*

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Choline chloride as therapy for fat embolism was apparently used for the first time at Minneapolis General Hospital in 1948.

Moosnick, Schleicher,† and Peterson[1] observed that the fat content of human bone marrow is decreased 90% when a 1% choline chloride solution is given intravenously.

Methods. Twelve dogs were used to determine the value of choline chloride as a

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† The authors wish to express their thanks to Dr. Emil Schleicher for offering the suggestion that choline might be of value in treatment of fat embolism and for his invaluable advice. They also express thanks to Dr. Steven Barron of Department of Pathology for his advice and assistance in preparation of microscopic material.

[1] Moosnick, F. B., Schleicher, E. M., and Peterson, W. E., *J. Clin. Invest.*, 1945, **24**, 228.

therapeutic agent for fat embolism. Examinations of blood and urine were done to determine presence and size of fat globules.

Fat embolism was produced in 12 dogs according to Friesen *et al.*,[2] under pentobarbital anesthesia by doubly drilling through both cortices of the lower ⅓ of the humerus, and lacerating the bone marrow with a sharp curet. These dogs were observed for a period of 5 days, and then autopsied.

After surgery half the dogs were given .25 cc 1% choline chloride per kilogram body weight intravenously 3 times a day at 5 hour intervals into the leg vein opposite the traumatized humerus. The other half served as controls.

Blood specimens were collected daily from each dog.

Daily specimens of urine were collected by catheter. Five cc of urine was centrifuged for 5 minutes. One drop of urine from the meniscus was placed on a glass slide and one drop Sudan III was added. The preparation was then examined under high power after standing for five minutes. The size and number of fat globules were recorded.

Results. In the choline treated dogs, fat globules were found in the blood plasma between the second and fourth day and these varied from 4 to 12 micra. Three globules were the maximum per field. None was observed in blood plasma on the fifth day.

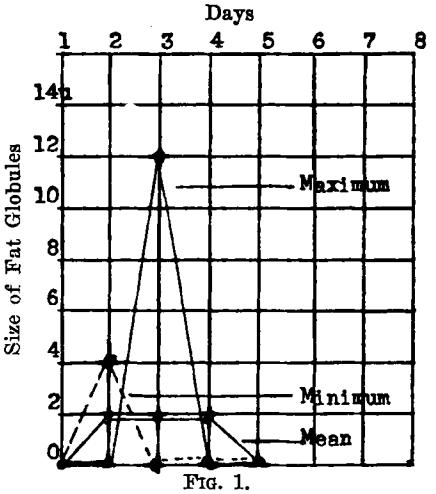

FIG. 1.
Fat in Blood of Choline Dogs.

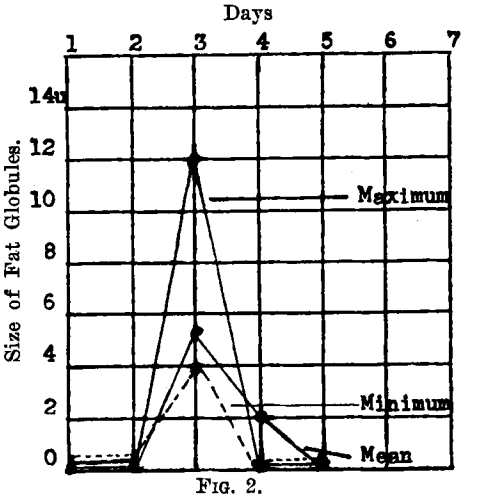

FIG. 2.
Fat in Urine of Choline Dogs.

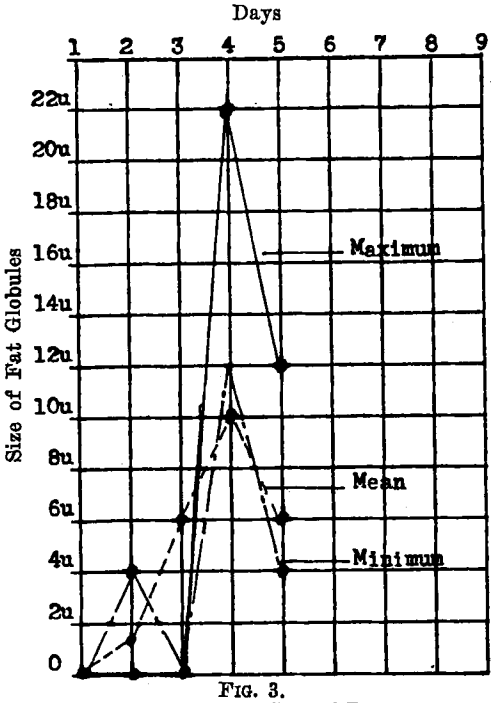

FIG. 3.
Fat in Blood of Control Dogs.

Fat globules appeared in the urine of the dogs treated with choline on the third and fourth day after surgery, varying from 4 to 12 micra in diameter. but not more than 2 fat globules per field were noted. None was seen in the urine on the fifth day.

[2] Friesen, S. R., Merendino, A. K., Baronofsky, Ivan, Mears, Frederick B., and Wangensteen, O. H., *Surg.*, 1948, **24**, 148.

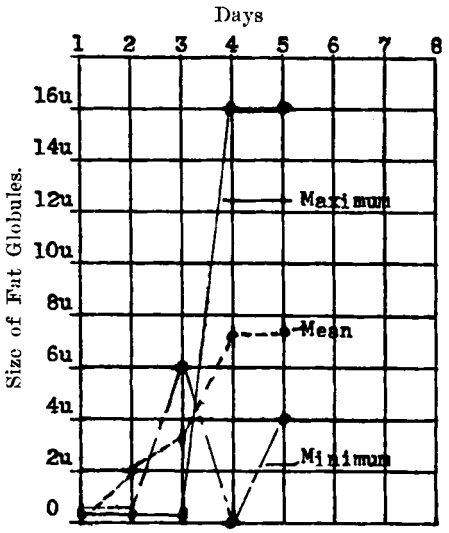

FIG. 4.
Fat in Urine of Control Dogs.

In the control dogs, fat globules were present in the urine and blood plasma from 2nd day after surgery to the time of autopsy. Maximum fat was present from the third day on. The size of fat globules varied from 4 to 20 micra; many were seen per high power field.

After autopsy, formalin fixed, hematoxylin and eosin stained sections of lungs showed fat emboli in all dogs with no appreciable difference between treated animals and controls.

Conclusion. Choline appears on the basis of plasma and urine examinations to have been of value in the management of experimentally produced fat embolism in dogs. Lung sections at five days after trauma proved inconclusive.

Note. Choline has been used in clinical cases of fat embolism with apparently very striking results. This is to be reported.

16918 P

Acute Occlusion by Ligature of the Portal Vein in the *Macacus rhesus* Monkey.*

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It is commonly accepted today that sudden occlusion of the portal vein both in the human and in animals is followed within a matter of minutes to hours by death. The first demonstration of this phenomenon is credited to Oré,[1] who noted that rabbits in which the portal vein was ligated survived but a short time. Since this original observation a large number of confirmatory reports can readily be found. The most definitive study is that of Elman and Cole,[3] who proved not only that ligature of the portal vein in dogs and cats is followed promptly by the death of the animal but also that failure to survive the procedure was due to loss of effective circulating blood volume secondary to pooling within the obstructed splanchnic venous bed. In addition to animal experiments such as these, there are a number of reports in the literature of occlusion of the portal vein in the human by thrombosis, either septic or bland, and by ligature made necessary either in the course of a surgical operation or secondary to intra-abdominal trauma.

The conclusion drawn from the animal experiments is that sudden obstruction of the portal vein is incompatible with life; from a review of the phenomenon as it is reported in the human the implication is also that in general a patient cannot survive sudden portal occlusion. A few reports are available for study, however, which indicate that occasionally the so-called "hepatopetal" veins may be sufficiently well developed to take over im-

* Supported by U. S. Public Health Service grant.

1 Quoted by Neuhof.[2]

2 Neuhof, Harold, *S. G. and O.*, 1913, **16**, 481.

3 Elman, R., and Cole, W. H., *Arch. Surg.*, 1934, **28**, 1166.