

This syndrome has been produced by both the sulfate and the lactate of aluminum. The lactate has been used in most of the experiments because solutions of it may be buffered at pH 6.0 to 7.0 (phenol red indicator) without precipitation of aluminum hydroxide. Intracerebral injection of doses of sodium lactate solution (pH 6.3) chemically equivalent to the amount of aluminum used had no effect upon the experimental animals. Moreover, these same animals succumbed typically to subsequent intracerebral injection of aluminum lactate.

Cultures from the brains of typical cases on blood agar and in Difco brain-heart infusion broth remained sterile. The possibility was considered that the syndrome was caused by a latent virus infection which was activated in the presence of the aluminum. However, negative results were obtained in attempts to transmit the disease in series by intracerebral inoculation of suspensions of brain tissue, from rabbit to rabbit and from rabbit to monkey.

Aluminum determinations have been made on the central nervous system of rabbits sacrificed in the terminal stage. A modification of the aurin method of Eveleth and Myers<sup>4</sup> was used. The results show that 25 to 37% of the injected aluminum is retained in the central nervous system. Of this amount 50 to 70% was in the cerebral hemispheres, 17 to 29% in the pons, medulla and cerebellum, and 13 to 23% in the cord. Normal controls contained an insignificant amount of aluminum in the central nervous system.

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### Blood Fats During the Dietary Production of Fatty Livers in Dogs.

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Studies of blood fats were made to determine if any change might be associated with the production of fatty livers in dogs which received a high fat diet. The lipids studied were the neutral fats of the plasma; which were determined by the volumetric method of Allen;<sup>1</sup> and cholesterol, by the Lieberman-Burchard method as used by Bloor.<sup>2</sup> Four dogs were kept on the high fat diet for 35 to 40

<sup>4</sup> Eveleth, D. F., and Myers, V. C., *J. Biol. Chem.*, 1936, **113**, 449.

<sup>1</sup> Allen, N. N., *Proc. Soc. Exp. Biol. and Med.*, 1934, **31**, 991.

<sup>2</sup> Bloor, W. R., *J. Biol. Chem.*, 1914, **19**, 1.

days. They were given the equivalent of 102 calories for each kilo of body weight daily, as 4 gm. of a standard mixed diet and 10 gm. of lard for each kilo of body weight. Three developed fatty liver while the fourth, which had received lipocaic<sup>3</sup> in addition to the fat diet, had a liver of normal fat content. Fasting blood samples taken at intervals fluctuated in the concentration of neutral fat, but always within normal range, and bore no relationship to the amount of fat in the liver (Table I).

TABLE I.  
Neutral Fats in Plasma\* (mg. per 100 cc.) During Production of Fatty Livers by a High Fat Diet.

Days on diet	Dog			
	1	2	3	4†
1	258	211	230	76
7	118	106	218	114
13	146	185	277	217
20	161	130	304	210
26	135	114	209	217
28	264	244	342	394
32	321	260		
Liver fat, %	28.0	25.7	18.7	4.4

\*Blood specimens taken 24 hours after feeding.

†Received lipocaic.

One day each week specimens of blood were taken at intervals, usually of 3, 6, 9, 12, 15, and 24 hours, following the regular fat meal. In spite of the high percentage of fat in the diet, which is known greatly to increase the emptying time of the stomach, definite lipemia occurred in 3 hours, particularly during the first few weeks that this diet was used. Later, perhaps because of intolerance to the diet, there may be no increase in neutral fat during this interval, or there may be even a decrease. Although in some instances the amount of postprandial fat in the blood was similar to that described by Bloor, Pelkan and Allen,<sup>4</sup> with a maximum in 6 hours, followed by a prompt return to the fasting level, often there were sporadic increases and decreases throughout the 24 hours. These are no doubt attributable to the irregular and prolonged absorption of fat which may occur with long-continued use of such a diet. A few of the typical curves are represented in Table II. Cholesterol in the plasma fluctuated within the normal range during these periods of 24 hours but apparently independently of either the blood fat or liver fat.

<sup>3</sup> Dragstedt, *Am. J. Physiol.*, 1936, **117**, 171.

<sup>4</sup> Bloor, W. R., Pelkan, K. F., and Allen, D. M., *J. Biol. Chem.*, 1922, **52**, 191.

TABLE II.  
Neutral Fats in Plasma (mg. per 100 cc.) During Production of Fatty Livers.

Days on diet	Hours following fat meal						
	0	3	6	9	12	15	24
	Dog 1—28% liver fat.						
1	258	397		446	360	276	237
13	146	261	263	124		315	104
26	135	380	313	347	254		202
	Dog 2—25.7% liver fat.						
1	211	330		347	300	240	219
13	185	248	187	124		252	144
26	114	260	170	228	262		135
	Dog 4—4.4% liver fat.*						
1	76	100	99	144	126	113	114
13	217	224	276	212		263	172
26	217	362	328	428	338		276

\*Animal also received lipocaic.

Four dogs were given alcohol in addition to the fat diet since fatty livers can thus be produced more rapidly.<sup>5</sup> Fat curves of these dogs again showed values well within the normal range (Table III).

TABLE III.  
Neutral Fats in Plasma (mg. per 100 cc.) of Dogs on a High Fat Diet Plus Alcohol.

Dog	Days on diet	Time after feeding, hours				Liver fat, %
		0	3	8	24	
5	14	344	263	291	272	20.7
6	8	300	203	308	202	11.1
7*	8	268	260	249	260	9.2
8*	14	327	392	417	257	6.5

\*Received lipocaic.

*Conclusions.* The neutral fats of plasma are not elevated beyond the normal range during the production of fatty livers by a high fat diet or a fat diet plus alcohol. Fluctuations which do occur during the 24 hours following a meal are probably associated with varying rates of absorption.

<sup>5</sup> Bollman, J. L., and Mann, F. C., *Arch. Path.*, 1935, **20**, 156.