when both consume the same amount of dietary protein. Other adrenal cortical factors seem necessary for an over-all normal protein metabolism. This is further indicated by Winternitz[7] who found that the weight gain of adrenalectomized rats on sodium chloride was largely water and protein whereas pair fed normal rats exhibited an increase in fat but no change in protein. Our data would also suggest that digestibility is essentially normal in salt-maintained adrenalectomized rats although the low gastric secretion, with a low acid and enzyme content, of the adrenalectomized rat is not improved by salt.[8] Hartman *et al.*[9] have reported that the plasma proteins of the adrenalectomized dog are improved by sodium salts but not to the same degree as by cortical extract. Recent clinical studies on patients with Addison's disease reveal that these individuals have a low plasma albumin, in agreement with the results of animal experiments.[10]

The fresh weight of a number of organs was taken at autopsy to determine whether the adrenalectomy and/or the self-imposed starvation influenced organ weight. No significant organ weight differences for the pituitary, thyroid, testes, seminal vesicles, ventral prostate, spleen, kidney, or liver were obtained regardless of the groups compared.

*Summary.* The adrenalectomized rat maintained on sodium chloride for 20 days has a subnormal plasma albumin concentration. Pair fed rats exhibit the same low plasma protein concentrations and the same impaired growth rate. In the replacement in part of adrenal cortical function sodium salts appear to permit the adrenalectomized rat to manufacture plasma proteins in a normal manner although appetite is subnormal. Organ weights are not affected.

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## 15156

## The Sensory and Motor Axons of the Chorda Tympani.*

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The gross relations of the chorda tympani are well known and it is accepted that, functionally, it is a mixed motor-sensory nerve. The motor component is composed of preganglionic visceral efferent axons which pass to the submaxillary and associated terminal ganglia[1] and the sensory unit is made up of afferent axons which are distributed to the taste buds on the anterior two-thirds of the tongue[2,3] and possibly to the lingual mucosa.[2]

Until recently but little information has been available on the morphological types and sizes of axons in the chorda tympani. We have shown[4] that the nerve of the cat contains myelinated and unmyelinated axons. Both varieties of nerve fibers are of small caliber with the myelinated nerve fibers ranging from 1.5 to 6 micra in diameter while the unmyelinated axons are less than 1.5 micra

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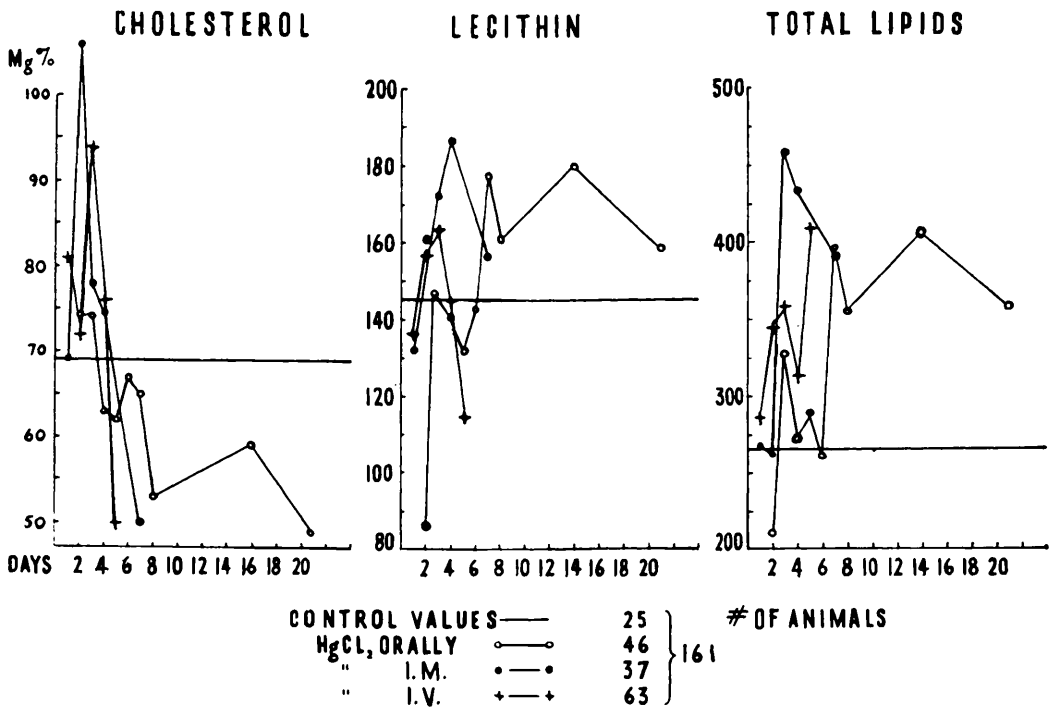


**BLOOD LIPIDS IN RATS AFTER ADMINISTRATION OF $HgCl_2$**
**(AVERAGE VALUES)**

FIG. 1.

TABLE III.
Effect of Oral Administration of Carbon Tetrachloride on Serum Lipids of Dogs.

| Dog No. | Avg values before administration of $CCl_4$ in mg% | | | Lowest value after administration of $CCl_4$ in mg% | | | No. days after first dose | cc | Dose |
|---|---|---|---|---|---|---|---|---|---|
| | Cholesterol | Lecithin | Total lipids | Cholesterol | Lecithin | Total lipids | | | |
| 31 | 115 | 260 | 490 | 100 | 220 | 450 | 14 | 15 | once |
| 33 | 188 | 300 | 680 | 100 | 220 | 580 | 13 | 15 | " |
| 60 | 118 | 200 | 400 | 72 | 130 | 300 | 4 | 5 | every other day |
| 56 | 131 | 330 | 550 | 50 | 130 | 320 | 21 | 5 | " 2-4 " |
| 47 | 148 | 340 | 520 | 50 | 150 | 120 | 25 | 5 | " 2-3 " |
| 44 | 190 | 374 | 630 | 49 | 150 | 350 | 24 | 5 | " 2 " |

lecithin, and total lipids in serum. This strongly suggests that the hyperlipemia previously observed was not due to carbon tetrachloride but to the tissue necrosis resulting from its intramuscular injection. The hypolipemia observed after its oral administration may well be connected with the hepatic damage so produced.

*Summary and Conclusions.* (1) Unilateral nephrectomy was performed under local anesthesia in 3 dogs and was followed by a transient hyperlipemia involving cholesterol, lecithin, and total lipids. It thus is shown that the general anesthesia used in previous studies had no part in the development of the hyperlipemia following renal ablation.

(2) Mercury bichloride given by intravenous injection to 5 dogs produced in all animals a hyperlipemia identical in pattern and intensity with the reaction observed when intramuscular or subcutaneous injections had

been used. The local tissue necrosis which occasionally results from intramuscular and subcutaneous administration of mercury bichloride thus cannot be the cause of the hyperlipemia previously described.

(3) The same hyperlipemic reaction is observed in rats after intramuscular and intravenous injection of mercury bichloride. However, no immediate rise in serum lipids was observed after the oral administration of mercury bichloride. It remains to be investigated whether an effect of this agent upon the liver might explain the different action of enteral and parenteral administration of mercury bichloride.

(4) It was found in 6 dogs that the oral administration of carbon tetrachloride leads to hypolipemia involving cholesterol, phospholipids, and total lipids. This makes it clear that the hyperlipemia previously observed after it had been injected intramuscularly was not due to carbon tetrachloride but to the necrosis of the tissues in which it had been injected.

## 15161

## Influence of Pyridoxine, Inositol, and Biotin on Susceptibility of Swiss Mice to Experimental Poliomyelitis.*

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Previous reports from this laboratory[1,2,3,4] have shown that the nutritional state of the Swiss mouse plays a role of varying importance in the susceptibility of this host to experimental poliomyelitis. Similar results with thiamine have been reported by the Pennsylvania group.[5] The most striking data were obtained with thiamine,[1] namely, mice fed a synthetic ration deficient only in this vitamin, exhibited a marked protection against infection with the poliomyelitic viruses, and indeed, in some series this resistance was 100%.[4] An attempt to ascertain the fundamental reason for these results[4] showed that the addition of pyruvate to thiamine optimum diets prolonged the incubation period of the infection in some instances, but did not manifest the marked resistance noted in frankly thiamine-deficient mice.

The specificity of the effect of nutrition in this disease is suggested by the fact that in pantothenic acid deficiency there is a definite increased resistance to Theiler's encephalomyelitis, but little or none to the Lansing strain of poliomyelitis virus,[2] while mice deficient in riboflavin manifest no altered susceptibility to Theiler's infection and a very slight increased resistance to the Lansing virus.[3] These results as well as those obtained with thiamine deficiency[1] suggest that factors such as inanition play a less significant role than the specific vitamins.

In the present report studies are presented for pyridoxine, biotin, and inositol deficiencies.

*Methods and Materials.* The synthetic optimal diets employed were composed of the following parts per 100: sucrose, 73; vitamin-free casein, 18; salt mixture, 4;[6] and corn oil, 5. The vitamin supplements per 100 g of diet

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