TABLE I. Porphyrin Content of Peripheral Nerves of Pigs.

	Coproporphyrin III/100 g	Protoporphyrin/100 g	Coproporphyrin / Protoporphyrin
	(μg/100 g tissue)		
	91.5	42.0	2.18
	96.0	47.3	2.03
	93.6	48.2	1.94
	99.0	44.6	2.21
	93.0	42.0	2.21
Avg	94.6	44.8	2.11

separated according to the method of Schwartz and Wikoff(6). Quantitative determinations of copro- and protoporphyrin concentrations were made fluorometrically(5).

Results. The results of 5 determinations are shown in Table I. It is apparent from this that the amounts of porphyrin in peripheral nerves are even more abundant than in the central nervous system. The porphyrins in peripheral nerves were found to be difficult to demonstrate spectroscopically, but the emission bands of porphyrins in peripheral nerves were observed with a Gaertner quartz spectrograph.

The dominant porphyrin in peripheral nerves as in the central nervous system was coproporphyrin III. It was noted, however, that there was a constant ratio of coproporphyrin to protoporphyrin when the nerve tissue was protected from autolysis before the determination.

Summary. Both coproporphyrin III and protoporphyrin were determined in the peripheral nerves of pigs. An average of 5 determinations showed that 100 g of nerve contained 94 μg of coproporphyrin and 45 μg of protoporphyrin. The ratio of coproporphyrin to protoporphyrin was approximately 2:1 and appeared to be constant.

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Absence of Gastro-Intestinal Lesions in Rats Following Ingestion of Silica. (23732)

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The finding of solid silica in lungs and other tissues and its toxicity when inhaled or injected intravenously has led to attempts to produce lesions by ingestion. (Piezoelectric effects of crystalline silica have been blamed but no piezoelectric effect is possible unless the crystal is compressed in one direction.) Silica occurs in food and to the extent of 2% in human blood ash. We wished to determine whether 10% of the diet as silica dust would be toxic. Seventy weanling rats were divided into 7 groups of 10 rats each. One group was fed Purina dog chow and the others 90% Purina dog chow and 10% of one of the forms of silica shown in Table I.

The rats were fed these diets 3 months and then killed and histological preparations made of the stomach, small and large intestine, liver, spleen, pancreas, adrenals, mesenteric lymph nodes and lung. No lesions were found in any of the organs studied.

It may be objected that 3 months is not sufficient time to produce lesions with silica. Clinical symptoms of silicosis have been reported in humans breathing silica dust for 6 months but most cases studied have been exposed to silica dust from 1 to 40 years. If the rat is to be used as an experimental animal it must be assumed that it lives more rapidly and that changes occur in less time than in

TABLE I.

Name	Particle size in μ
Linde silica	.02–.05
Columbia-Southern Hi-Sil 101	.03
Dow Corning silica (unpelletized)	.01–.02
Powdered silica gel	
Sodium metasilicate	
Carborundum Co. Fibrofrax (silica fibers)	

humans.

Summary. Powdered silica or Fibrofrax mixed with the rat diet to the extent of 10%, produces no tissue pathology in the gastrointestinal tract, liver, spleen, pancreas, adrenals, lung or mesenteric lymph nodes when fed continuously for 3 months.

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Cerebrospinal Fluid Enzymes in Central Nervous System Lipidoses* (with particular reference to Amaurotic Family Idiocy) (23733)

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The diagnostic usefulness of certain enzyme levels in cerebrospinal fluid (CSF) and serum derived from cases of central nervous system (CNS) disease has recently been explored (1-5). Various authors have demonstrated transient glutamic oxalacetic transaminase (GOT) elevation in CSF derived from clinically determined or experimentally induced cerebral infarction(1,2). Other investigators, in evaluating the utility of CSF GOT in a broad spectrum of neurologic disorders, were unable to report any conclusive alterations in the levels of this enzyme(4).

Significant rises in serum GOT and aldolase have recently been recorded in cases of Infantile Amaurotic Family Idiocy (IAFI) (5). These findings indicating that a number of determinable serum enzymes is increased in one of the CNS lipidoses led to an assessment of their activity within the CSF of cases in this disease category. Concurrent serum levels were also determined to clarify any possible correlation between the two biological fluids. The activity of lactic dehydrogenase (LD) was also measured in the serum and CSF of selected lipidosis and control cases.

Material and methods. a.) Clinical: Repeated serum and CSF specimens from 9 patients with IAFI (at varying stages of the illness) and 2 cases of Niemann-Pick's Disease (NPD) with CNS involvement were studied in regard to GOT, aldolase and LD levels. Similar enzyme studies were performed on 33 additional children afflicted with other neurological disorders. b.) Experimental: 10 mongrel dogs, each weighing about 13 kg, were injected intramuscularly with 1 g (GOT enzymatic activity: 80,000 u./g) of fresh heterologous canine grey matter. An additional 3 dogs were inoculated with a similar homogenate previously incubated at 100°C for 1½ hours (GOT enzymatic activity, 0.0 u./g). All dogs were then sequentially studied by daily serum GOT determinations for periods up to 8 days. The method employed for determination of serum and CSF GOT was that of Karmen, Wróblewski and LaDue(6) based on spectrophotometric measurement in the near ultraviolet with the Beckman D.U. spectrophotometer. Serum and CSF aldolase activity was estimated with the colorimetric procedure of Sibley and Lehninger(7). The LD activity of the serum and CSF samples were also determined spectrophotometrically at 340 mμ with the Beckman D.U. using the method of Wróblewski and LaDue(8).

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