navy bean trypsin inhibitor does protect rabbits against the acute lethal effect of trypsin. The latter is to be described elsewhere.

Summary. Free amino and phenolic groups do not appear to be essential to the proteolytic or the hypotensive activity of trypsin. Characteristics of the protein groups associated with the hypotensive activity of trypsin are given. These correspond with the properties of free tryptophane although participation of other groups is not excluded. Changes in the proteolytic and hypotensive activities of trypsin are not always parallel.

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On the Relative Efficacy of Emetine in Intestinal and Hepatic Amebiasis.

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Although emetine has been used therapeutically in amebiasis for more than 35 years, the tissue distribution and concentration of this alkaloid have not been reported. Nevertheless, certain limitations of the drug's efficacy are well known. Emetine is considered curative in amebic hepatitis and even in amebic abscess. On the other hand, relapses of amebic dysentery or the recurrence of cysts in the stool are frequently seen after emetine therapy despite the ability of the drug to clear up the acute symptoms.[1]

There are various theories as to why emetine is ineffective in amebic dysentery and curative in amebic liver disease. One widely accepted theory is based on observations that, *in vitro,* emetine is capable of destroying the trophozoites but not the cysts of *Endameba histolytica.* Cysts are as a rule not found in tissues; their origin in the intestinal lumen is in trophozoites leaving the tissues and encysting. According to this theory, emetine is ineffective because both trophozoites and cysts occur in amebic dysentery. On the other hand, since only trophozoites are present in amebic liver disease this type of amebiasis is readily eradicated by emetine. Craig believes that emetine fails to eradicate all the trophozoites from the intestine, leaving some to start the disease process again.[1] No mention is made as to why the drug is capable of destroying the trophozoites in the liver.

TABLE I.
Liver and Intestinal Concentration of Emetine After an Intramuscular Injection of 6 mg per kg. Concentrations are averages of 3 rabbits in each group.

Rabbit group	Time after inj.	Emetine conc. (mg/kg) Liver	Intestine
1	1 hr	24.9	5.5
2	12 "	41.6	1.7
3	3 days	19.2	1.7
4	4 "	15.4	1.2
5	6 "	10.3	0
5	13 "	2.7	0
7	20 "	2.1	0
8	28 "	1.0	0

The role of blood and tissue levels of chemotherapeutic agents as a factor in effectiveness of therapy has assumed great importance in recent years. The following findings are presented to show that the tissue levels of emetine may explain the divergent results obtained with the drug in amebiasis of the liver and of the intestine.

Method. Each of 24 albino rabbits weighing about 2.5 kg were injected intramuscularly with 6 mg per kg of emetine hydrochloride in aqueous solution. They were then sacrificed in groups of 3 at intervals of from 1 hour to

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1 Craig, C. F., *Etiology, Diagnosis, and Treatment of Amebiasis,* Williams & Wilkins Co., Baltimore, 1944.

28 days. A portion of the large intestinal wall, freed of feces, and a portion of liver were analyzed for emetine. The analysis was adapted from the general method of Brodie *et al.*[2]

Table I shows the emetine concentration in the liver and intestine. One hour after injection, the concentration of drug was much higher in the liver than in the intestine. At 12 hours, the liver concentration of drug was 41.6 mg per kg compared to the intestinal concentration of only 1.7 mg per kg. At the end of 4 days the low concentration in the intestine became almost undetectable whereas that of the liver was maintained for more than 4 weeks.

Summary. Injection of emetine into rabbits was followed by a relatively high drug concentration in the liver for more than a month, whereas the level in the intestine was always much lower and became undetectable after 4 days. The findings of a high concentration and prolonged presence of emetine in the liver and a low concentration and transient presence of the drug in the intestine are proposed as the explanation for the efficacy of emetine in amebic liver disease and the drug's failure in amebic intestinal disease in man.

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Phospholipid Utilization in the Diabetic Dog.*

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Depancreatized dogs maintained on insulin developed fatty livers, emaciation, and anemia.[1] Dragstedt maintains that this syndrome is due to the lack of a specific substance present in pancreas, which he has named lipocaic.[2] The syndrome is relieved or prevented by feeding choline, raw pancreas, and various choline-free pancreatic extracts.

It has been shown that the plasma phospholipid level is lowered in lipocaic deficiency.[3] Phospholipid is probably the chemically active form of fat and the significant lipid fraction in the transport of fat. There is evidence that choline deficiency in rats, which resembles lipocaic deficiency, results in an impairment of transportation of fat from the liver to the tissues.[4] In the present study, the plasma phospholipid turnover was measured in normal dogs and in diabetic dogs under a variety of conditions.

Numerous studies on phospholipid metabolism have been performed with the use of isotopic tracers. One conclusion from this work is that the plasma phospholipid is produced largely in the liver.[5]

In the present study, it is assumed that the plasma phospholipid is produced in the liver and withdrawn from the plasma for utilization by the tissues. Since the phospholipid levels remained constant during the period of measurement, the rate of production of new phos-

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