particularly evident in Columns F and G, where the average citric acid increments on both the Cbh and Pr diets are of the same order of magnitude.

The foregoing data indicate that there is a direct relation between the amount of alkali administered to the rat and the excretion of endogenous citric acid. It would appear therefore that under the present experimental conditions, citric acid plays an important role in the regulatory mechanism involved in the acid-base balance of the body.

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Metabolic Studies in Patients with Cancer of Gastro-Intestinal Tract. XV. Lipotropic Properties of Inositol.*

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Recent studies from this hospital have demonstrated that the livers of patients with gastro-intestinal cancer almost uniformly are infiltrated with fat.[1] In contrast to this finding, normal fat contents were found in the livers of 11 patients with carcinoma of the gastro-intestinal tract who each received 8 g of lipocaic† during the night prior to laparotomy. The average hepatic fat concentration of this latter group was only 0.46 that of the control untreated patients.[2]

Lipocaic, however, is a crude mixture which contains significantly large amounts of choline and inositol,† both of which are lipotropic alone.[3,4] In experimental animals, the lipotropic effects of lipocaic apparently are not due to its choline alone,[5] and this observation has been confirmed here for human subjects.[2] On the other hand, when about 4 times the amount of inositol present in the 8 g of lipocaic was administered to a group of 8 patients with gastro-intestinal cancer, with but one exception the concentrations of fat in their livers were found to be within normal limits. The average lipid value was 0.42 that of the control group.

TABLE I.
Concentration of Fat in Livers of Patients with Gastro-intestinal Cancer Who Received Preoperatively 280 mg of Inositol.

Patient	Disorder	Conc. of hepatic fat, g per 100 g wet tissue
J.K.	Carcinoma oesophagus	7.2
R.H.	" stomach	8.7
O.S.	" colon	7.1
M.S.	" stomach	10.3
M.T.	" colon	7.8
W.R.	" stomach	5.7
H.J.	" "	5.0
F.M.	" "	15.9
G.M.	" "	5.9
M.J.	" "	8.9
	Avg	8.25

Because it seemed probable then that the inositol alone might account for the physio-

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† The lipocaic used in this investigation was supplied through the courtesy of Eli Lilly & Co., Indianapolis, Ind., and the inositol through the kindness of Lederle & Co., Pearl River, N.Y. The concentration of choline and of inositol in the material used was found by analyses to be 2.6 and 3.5 g % respectively. These analyses were made by Dr. W. Wooley of the Rockefeller Institute for Medical Research, New York City, and by the Laboratories of the Standard Brands, Inc., New York City.

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TABLE II.
Average Concentration of Fat in the Livers of Patients with Gastro-intestinal Carcinoma Who Were Fasted and of Those Given Various Dietary Supplements Preoperatively.

No. of patients	Preoperative supplement	Avg conc of hepatic fat, g per 100 g wet tissue	% reduction of hepatic lipid effected by supplement
28	None	16.4	—
11	8 g lipocaic	8.0	51
7	3 g choline chloride	11.2	39
8	1200 mg inositol	6.9	58
10	280 " "	8.2	50

logic effects of the lipocaic, another group of 10 patients with carcinoma of the gastro-intestinal tract was given during the last 10 preoperative hours only that amount of inositol (280 mg) contained in the effective dose of lipocaic. The clinical and chemical methods used have been described in a preceding communication of this series.[1] The results obtained from the ingestion of the smaller amounts of inositol indicate that in the patients studied, the lipotropic properties of the lipocaic could be accounted for by its content of inositol alone (Tables I and II). Of the values of hepatic lipid, only one was significantly elevated, and the average of the group was only 0.50 that of the control group.

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Titration of St. Louis Encephalitis Virus and Jungeblut-Sanders Mouse Virus in Tissue Culture.*

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Following successful titration and neutralization of the Western strain of equine encephalomyelitis virus in chick embryo tissue culture,[1,2] attempts were made to see whether or not the same method could be applied to other viruses. The St. Louis encephalitis virus and the Jungeblut-Sanders mouse virus[3] were chosen for study.

The principle on which titration of the equine virus in tissue culture is based, is the finding that tissue fails to grow when it is heavily infected with virus. Thus, in the earlier studies when St. Louis encephalitis virus and Jungeblut-Sanders mouse virus were tested in chick embryo tissue cultures, it was found that potency could not be determined as simply as with the equine virus. There was no appreciable difference in the growth of infected and non-infected tissue when the tissue was subsequently patched with plasma.

Since it was felt that the failure to note a difference in growth of infected and non-infected tissue treated with St. Louis encephalitis and Jungeblut-Sanders mouse viruses was probably due to low susceptibility of chick embryo tissue to these 2 viruses,[2] it became of interest to determine whether or not a difference in the growth of infected and non-

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