subjects and certainly as high as others have reported.[4] That the lower excretions were probably due to a previously low intake of the compound is strongly suggested by the fact that in every case where there was an appreciable difference in the range of the daily excretion, the lower values were observed at admission of the patient, with a progressive rise while on the hospital diet. A typical case was patient No. 5 whose consecutive daily excretions were 2.5, 3.0, 5.6, 6.2, and 7.3 mg. This increase was not due to the parenteral liver extract, for analyses of the preparation indicated that he received less than one mg from this source during the whole course of treatment.

Further evidence that the excretion of pantothenic acid by the patients was not limited by impaired absorption was obtained by the oral administration of calcium pantothenate. Following 100 mg doses of the salt, the patients and controls excreted comparable amounts, as shown in the table.

This study is being extended to include other water-soluble vitamins.

Conclusions. In patients with pernicious anemia, the daily excretion of pantothenic acid both before and after administration of 100 mg of its calcium salt was slightly but probably not significantly less than that of healthy individuals studied in the same manner. This indicates there is no impairment of absorption of this compound in patients with pernicious anemia with which is always associated achlorhydria. The possibility must be considered, however, that the lack of hydrochloric acid may make the pantothenic acid in food less available for absorption. This is undergoing further study.

13566 P

Electrocardiographic Changes Associated with Acute Pancreatitis.

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We have had occasion to observe a series of cases of acute pancreatitis, in which the signs of upper abdominal peritoneal irritation were associated with elevated blood amylase and abnormal electro-

cardiograms. Serial electrocardiograms taken throughout the course of the disease in those who survived showed a return to a normal pattern as the clinical signs and symptoms of the disease abated, and as the elevated blood amylase became normal. The transient character of the electrocardiographic changes suggested to us that they were caused by the acute pancreatitis rather than by any intrinsic cardiac pathology. The following abstract of one of the cases is illustrative of the various changes observed during the course of the disease.

E. B., colored, male, 38, admitted December 5, 1941, because of abdominal pain, nausea and vomiting of 5 days' duration following a drinking spree. Examination showed a dehydrated, acutely ill patient, complaining of upper abdominal pain. The abdomen was distended, and was markedly tender throughout the epigastrium and the left upper quadrant. The entire abdomen was rigid. The clinical diagnosis of acute pancreatitis was made.

The course is outlined below:

Date	Condition of patient	Electrocardiogram	Blood Amylase	Other findings
12/5	Acutely ill Temp. 100, Pulse 80, abdomen tender	Depressed ST segment in leads 2 and 3. Diphasic T waves in leads 1, 2 and 3. Myocardial damage.	410	WBC 11,200.
12/8	Condition unchanged. Temp. 100, Pulse 80.	T 1 inverted T 2 diphasic RT 4 depressed Myocardial damage	374	
12/11	Vomiting persists. Tenderness in left upper quadrant.	T 1 iso-electric T 2 low T 4 inverted Myocardial damage	315	
12/19	General condition improved. Patient ambulatory.	All changes previously noted have disappeared. Electrocardiogram normal.	303	Blood sugar 64 N.P.N. 19 Chol. 164 Chol. esters 68%
1/15/42	Patient well. No complaints.		254	

A study was undertaken on dogs to determine whether these changes could be duplicated in experimental acute pancreatitis.

Method of Study. Under intravenous nembutal anesthesia the accessory pancreatic duct in dogs was exposed, 5 cc of a 12% solution of sodium taurocholate was injected into the pancreas and the duct was then ligated. Pancreatic congestion was noticed immediately, and a fatal pancreatitis was produced, death occurring after 12 to 15 days. Autopsy revealed the presence of acute pancreatitis with abscess formation.

Electrocardiograms were taken preoperatively and at 2- to 3-day

intervals postoperatively until the animal died. Control electrocardiograms were taken in dogs similarly anesthetized but not operated upon.

The electrocardiograms in the operated dogs showed distinct aberrations from the normal preoperative tracings.

Protocol Dog 4 operated 1/13/42. Preoperative electrocardiogram—sinus arrhythmia.

1/14/42 Electrocardiogram—inversion of the T wave in lead 1.
1/16 " inversion of the T wave in lead 1.
1/19 " inversion of the T wave in lead 1.

Changes became progressively more marked, indicative of increasing myocardial damage.

The changes were interpreted in some instances as indicative of myocardial damage and in others as showing evidence of coronary closure with infarction. There was no constancy in the electrocardiographic patterns nor did they always correspond to the definite electrocardiographic changes observed clinically in heart disease.

The exact mechanisms underlying these changes are being further studied. Observations are also being continued on the association of electrocardiographic changes with other acute intraäbdominal lesions.

Summary. Clinical observations have demonstrated the association of acute pancreatitis with transient electrocardiographic changes. We were able to obtain similar changes in dogs with experimentally induced pancreatitis.

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Relationship Between Body Size and Metabolism.

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Recently Kleiber has studied the relation in liver slices of oxygen consumption to body weight in rats, rabbits, and sheep.[1] It was shown that the Q_{O_2} was inversely proportional to a fractional power of the body weight, so that plotted on a log log grid the data gave a straight line represented by the formula

$$Q_{O_2} = 5.26\ W^{-0.24}$$

1 Kleiber, Max, PROC. SOC. EXP. BIOL. AND MED., 1941, **48**, 419.