blood cells which can mature beyond the megaloblast stage and assume the adult characteristics in untreated patients.

The volume of gastric juice* secreted per hour and the red blood cell counts were determined in 17 patients with pernicious anemia in relapse. A total of 117 observations were made. Whereas the average gastric secretion for normal individuals is approximately 150 cc. per hour, the gastric secretion in patients with pernicious anemia was found to vary between 5 and 29 cc. per hour. If the average red blood cell count (ordinate) Fig. 1, is compared to the volume of gastric secretion per hour (abscissa), a direct relationship becomes apparent. That is, the greater the gastric secretion, the higher the red blood cell level. Apparently erythropoiesis depends in part, at least, upon the action of the intrinsic factor and the rate of red blood cell formation is related to the amount of the intrinsic factor produced.

Furthermore, there was suggestive evidence that as the gastric secretion was depleted by constant drainage, in each patient there was a decrease in the red blood cell count of the peripheral blood. These experiments are being repeated and amplified with various modifications to verify the conclusions.

7730 P

Creatine Mobilization in Myocardial Damage.

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In our clinical studies[1] the discovery of a hypercreatinemia and creatinuria following coronary thrombosis with cardiac infarction and in acute myocardial insufficiency with congestive failure prompted us to attempt to establish a curve of creatine excretion.

Old male dogs that showed no creatinuria, on a diet of 150 gm. of dried bread and 200 cc. of evaporated milk with chopped cabbage

* Gastric juice was obtained by continuous suction for 3-hour periods. Histamine hydrochloride injections did not influence the volume of secretion, and, therefore, was not used routinely. All specimens were obtained at least 6 hours after the ingestion of food.

[1] Herrmann, G., Decherd, G., Erhard, P., and Klippel, P., *J. Lab. and Clin. Med.*, in press.

and charcoal, were selected and varying grades of obstruction to the coronary circulation were produced with a minimal amount of skeletal muscle trauma. Under amytal anesthesia the right carotid artery was carefully exposed by a separation of the muscles, a lead impregnated ureteral catheter was guided, under the fluoroscope, through an opening in the carotid down into one of the aortic sinuses. With the tip opposite a coronary orifice a large or a small drop of metallic mercury was run in from a syringe. Thirty grams of glucose in 200 cc. evaporated milk was given by tube immediately after operation and repeated in 12 hours, after which the dog usually took all of his diet. A constant intake of food was maintained. A flash roentgenogram established the position of the mercury in the coronary system. Electrocardiograms taken before and at intervals following the injection presented graphic evidence of myocardial infarction.

In a series of 12 dogs a creatinuria developed within a day except in the very slight infarctions in which it sometimes appeared on the second or third day. The creatinuria then increased as is shown in Table I of Dog No. 12, to reach its maximum about the sixth

TABLE I.
Dog No. 12. Daily Excretion.

Days post-op.	Creatinine, mg.	Total mg.	Creatine, mg.	Blood Creatine, mg. %
Control	343	343	0	
Control	300	300	0	.58
1	342	342	0	.74
2	364	364	0	.94
3	384	397	13	.37
4	377	407	30	.49
5	329	373	44	
6	324	378	54	.55
7	331	363	32	
8	410	427	17	
9	365	372	7	.44
10	232	235	3	
11	315	315	0	.49
12	245	245	0	
13	257	257	0	

day. It then dropped off to disappear according to the extent of the damage between the tenth and fourteenth day. The nitroprusside test for acetone was constantly negative. In the instances of massive infarction hypercreatinemia appeared within 12 hours, and lasted 24-48 hours, and the creatinuria was more marked. The curves vary in contour and in height in proportion to the degree of muscle damage and are of some diagnostic and prognostic significance.