manipulations necessary to free the preparations from adherent ammonium sulphate. We conclude that the active substance is mainly proteose or is precipitated with it.

Further evidence that the inhibitory substance is one of the higher derivatives of protein digestion appears when our results are compared with chemical data kindly loaned us by Dr. Carl J. Bucher. This comprises an analysis of various commercial peptones for protein nitrogen, non-protein nitrogen, polypeptid nitrogen, amino acids, salt and sugar. The inhibitory effect of the various preparations varies directly with the amount of "protein" nitrogen, inversely with the amount of non-protein nitrogen and polypeptid nitrogen, and is in no way related to the total nitrogen. The analysis shows an insignificant amount of salt and amino acid in the most effective preparations and no sugar (a possible source of error) in Witte's peptone.

We conclude that certain products of protein hydrolysis, probably proteoses, act in the intestine to inhibit gastric peristalsis. The observation has a bearing on the regulation of gastric emptying during protein digestion and completes the list of major foodstuffs, each of which has now been shown to be capable of inhibiting gastric activity when present in the intestine in adequate concentration and in certain stages of digestion.

Further work is contemplated directed toward the isolation of similarly active material from the products of normal digestion in the animal and to learn whether the inhibitory effect is reflex, humoral, or both. Present indications point to a reflex effect inasmuch as the latent period for inhibition is often less than 30 seconds.

8809 P

Creatine Content of Human Hearts.

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Even before the important rôle of phosphocreatine in muscular contraction was recognized, Constabel[1] noted that there was a decreased total creatine content in those human hearts that showed either clinical or post-mortem evidence of damage. Recently

1 Constabel, Fr., *Biochem. Z.*, 1921, **122**, 152.

Cowan[2] has examined a larger group of hearts, and found that myocardial failure is accompanied by a creatine content that is markedly below normal. Seecof, Linegar and Myers[3], whose method for determining myocardial creatine we have employed, have also examined a series of human hearts, emphasizing particularly the higher creatine concentration in the left ventricular muscle as compared to the right ventricle.

In our laboratory we have approached from several experimental angles the question of the chemical changes that initiate or accompany heart failure. Since our primary interest is clinical, we have also analyzed muscle from the left ventricle of human hearts, as this material became available from time to time.[4] In Table I we have

TABLE I.
Creatine Content of Human Hearts.

No.	Cause of death	Mg. %	Solids %	Dried mg. %
11	Traumatic, normal hearts	183 ± 17	20.7 ± 0.62	887 ± 79
23	Infectious normal hearts	172 ± 15.4	20.1 ± 0.98	858 ± 70
34	Normal hearts	175 ± 20.7	20.3 ± 0.87	868 ± 70
	Heart disease without failure			
1	Rheumatic	210	19.9	1058
6	Hypertension	198 ± 15.6	20.4	970 ± 79
11	Coronary sclerosis	157	20.	783
18	Total without failure	173	20.1	860
6	Glomerulonephritis with uremia	159	20.3	781
5	Severe anemia	157	19.3	819
32	Congestive heart failure	122 ± 20.8	20.3 ± 1.2	605 ± 100
10	Infections with microscopic myocardial change	121	20.5	596

summarized creatine determinations on 105 additional human hearts. The total creatine has been determined, the total solids, and from this calculated the percentage of creatine on a dry basis. The normal values have been determined in hearts from patients that died a traumatic or surgical death, and in hearts from those who died from an infectious disease, yet showed no gross or microscopic evidence of myocardial involvement. We list the mean value, with the standard deviation from the mean.

In hypertrophied hearts, which have not failed, the values for total creatine are elevated; in those with myocardial change secondary to coronary sclerosis, the averages are low. The creatine content of hearts that have failed is reduced, averaging 30% below

2 Cowan, D. W., *Am. Heart J.*, 1934, **9**, 378.

3 Seecof, D. P., Linegar, C. R., and Myers, V. C., *Arch. Int. Med.*, 1934, **53**, 574.

4 Herrmann, George, Decherd, George M., and Schwab, E. H., *Southern Med. J.*, 1936, **29**, 386.

normal. Low values were also obtained in hearts that showed microscopic changes, inflammatory or degenerative, from patients that had died of infectious disease.

We have previously noted[5] the drop in creatine content of infarcted heart muscle with experimental animals. Four human cases have shown the same changes. Table II shows the creatine values for the infarcted areas from human hearts, compared with the values from non-infarcted portions of the heart.

TABLE II.
Coronary Occlusion.

	Creatine mg. %		Solids %		Dried mg. %	
Ht. Wt.	Good	Infarcted	Good	Infarcted	Good	Infarcted
610	122	41	23.45	18.0	520	228
350	104	61	21.95	15.05	497	405
800	100	52	19.7	18.2	558	318
900	151	31	19.15	17.25	788	180

8810 C

Embolic Staphylococcus Arthritis and Endocarditis in Rabbits.

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There have been many clinical and experimental studies of arthritis and its associated conditions. The results and hypotheses set forth, however, are confusing and conflicting, especially regarding etiology and mechanism of production. Experimental inflammatory reactions have been produced within joints by intra-articular and intravenous injections of virulent and attenuated micro-organisms, bacterial toxins and various chemical agents. It has been postulated by various authors that the localization of a focus may be influenced by trauma, allergic responses and by the particular structure of joint tissues.

Studies of the blood supply and infarction of the femur in rab-

[5] Decherd, G., Herrmann, G., and Davis, O., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 547; Herrmann, G., and Decherd, G., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 1304.