rate of the loss of creatine appears to closely parallel that of the total nitrogen.

Still an added factor in this loss of creatine from the body is the excretion of creatinine. In experiments where the creatine of the urine plus that of the tissue does not entirely account for the creatine which should normally be present in the body, it is found that a considerable amount of creatinine has been eliminated in the urine, *e. g.*, in the case of a comparatively long fast. When this creatinine in terms of creatine is added to the creatine of the tissue and urine, this total exceeds the total normal body creatine by about 10 per cent., this excess probably representing the amount of creatine and creatinine formation. It would seem probable from these data that during starvation, the creatine storehouse was depleted not only by a loss of creatine in the urine, but also by the loss of creatinine.

It has been assumed for some time that the creatine appearing in the urine during starvation and in various pathological conditions was derived from the creatine of the muscle, and measured the amount of muscle disintegration, though so far as the authors are aware this point has never been conclusively demonstrated. In our experiments, we have found that when the weight of the creatine excreted in the urine was added to the weight of creatine still remaining in the body after the period of starvation, the amount of creatine was only slightly below that which would have been found in the body had the animal been killed prior to starvation. This would seem to demonstrate that the creatine appearing in the urine in starvation was derived from the creatine of the muscle.

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Reversal of the cardiac mechanism.

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The subject of investigation, H. M., came under observation at the Vanderbilt Clinic July 15, 1912. He has had a persistent

diarrhea for a year with several watery stools a day. Otherwise he has been well until three months before admission to the clinic.

During these three months and subsequently, he has suffered from attacks of dizziness of increasing severity. In one of these attacks he fainted and fell in the street. Muscular effort increases the giddiness. He has had a good deal of headache. He is very drowsy and is annoyed by numbness and tingling of the hands and feet.

Until the onset of symptoms he had been a heavy drinker. He denies lues and gonnorrhea. The Wassermann test was negative. The blood and urine are normal. The heart is not enlarged. The sounds are faint but clear, and there are no murmurs. The third sound has been distinctly audible at most examinations and has been recorded graphically. The pulse is regular, 40 per minute. Systolic bloodpressure varies between 95 mm. and 110 mm. and diastolic pressure is usually about 65 mm. The electrocardiograms show that the heart beat is initiated by the ventricles, the auricular beat succeeding the ventricular after the usual conduction interval. This is evidenced by the presence of an inverted P between R and T in leads II and III. In lead I, P is of very small amplitude, but can be clearly distinguished on close inspection and stands in the same relation to R and T as in the other leads.

This interpretation of the electrocardiograms has been confirmed beyond reasonable doubt by examination with Roentgen rays and fluoroscope. The contractions of the auricle can be distinctly seen.

We are not yet ready to offer an explanation for the peculiarly slow intrinsic rhythm of the auricles which permits the ventricle to initiate the beat. Neither atropin nor digitalis has any measurable effect on the condition. The subject is still under observation.