relative rapidity with which their proteins were digested, absorbed, and metabolized, as shown by the rate of nitrogen excretion during the height of digestion (figures in top row). From the nitrogen balance (figures in the bottom row) it is evident that the order would be practically reversed if the foods were ranked according to their ability to keep the body in nitrogenous equilibrium. It is apparent that rapid digestion does not necessarily further retention of protein. Presumably there is an optimum rate of digestion, which makes possible the fullest use of its products, and this rate may be exceeded. Rapid digestion can then cause imperfect retention. "Predigestion" has recently been shown by Voit and Zisterer to have this effect.[1]

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The cause of cardiac cohypertrophy.

By **HUGH A. STEWART.**

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In a series of twenty experiments in dogs in which cardiac hypertrophy was induced by the production of aortic insufficiency, it was found that all the chambers of the heart were heavier than normal. The largest increase was in the left ventricle, 48 per cent., but there was also found a very marked increase in the auricles which, relatively to their size, was almost as great as in the left ventricle. The cohypertrophy of the auricles has also been observed in man in cases of chronic interstitial nephritis and arteriosclerosis (Hirsch).

It was found experimentally that the increased work of the left ventricle after the production of aortic insufficiency is not associated with a change in venous pressure such as has been assumed to be the direct cause of hypertrophy of the auricles.

Tracings taken of the contractions of the right auricle along with a blood pressure tracing from the right carotid artery showed that the effect of the production of aortic insufficiency is to increase the force of auricular contraction. Similarly the increased work of the left ventricle produced by the compression of the thoracic aorta will also cause increased auricular contractions without any change in venous pressure.

[1] *Z. f. Biol.*, liii, 457.

The experiments would seem to indicate that there exists a coördination between the contractions of the ventricles and the auricles of such a nature that increased systole of the former causes an increased systole of the latter without any apparent change in venous pressure.

To this condition is ascribed the cause of auricular cohypertrophy.

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The origin of convulsions and paralysis following the intravenous injections of the hypertonic solution of sodium chloride.

By **DON R. JOSEPH** and **S. J. MELTZER.**

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In our experiments on the comparative toxicity of chlorides in which, among others, we studied the effects of intravenous injections of a molecular solution of sodium chloride in dogs we observed that after a certain quantity of the solution runs in, twitchings of the muscles of the entire body begin which gradually develop into more or less strong convulsions. Later these gradually grow weaker and finally subside completely, at which time also the respiration stops. The heart continues to beat for several minutes longer. This chain of events is ascribed by pharmacologists to the osmotic action of the hypertonic solution and is generally termed salt action. Loeb however demonstrated that sodium chloride exerts on the living tissue a chemical action also. In the paper embodying the above mentioned experiments we made the following two suggestions: (1) that the twitchings and convulsions are perhaps comparable to the twitching of frog muscles which develop when they are immersed in solutions of sodium chloride; the convulsive movements would be then of peripheral origin; (2) that the subsidence of the convulsions and the paralysis might be due to the curare-like action of the sodium chloride, *i. e.*, to the paralysis of the motor nerve endings.

In the present series of experiments we have tested these two hypotheses. The second hypothesis was tested by investigating