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The effect of stimulation of the vagus nerves upon the development of rigor mortis of the mammalian heart.

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Stimulation of motor nerves hastens the onset of rigor mortis in the corresponding skeletal muscles. Cutting the nerves retards it, the retardation being due, it is believed, to the elimination of subminimal nerve impulses. Would the retardation of the rigor be still greater if inhibitory nerves could be stimulated? This question is not applicable to skeletal muscles, but it is a definite problem with reference to the onset of rigor in the heart muscle. Would a prolonged effective stimulation of the vagus nerves retard the onset of cardiac rigor? There were reasons to expect that the effect of such a stimulation would be indeed a retardation. The action of the cardiac vagus is inhibitory and the reverse of the action of a motor nerve; we might then expect that the effect of its stimulation upon cardiac rigor would also be the reverse of the effect of stimulation of a motor nerve upon the skeletal muscles, that is, retardation instead of hastening. Furthermore, increased muscular activity hastens the onset of rigor; it seemed reasonable to anticipate that the diminished activity, such as frequent standstill or slowing of the heart, would retard the onset of its rigor.

We have studied this question in 42 dogs, 16 cats and 10 rabbits. Both vagi were stimulated for half an hour before death and frequently also after death. Death was caused uniformly by bleeding and opening of the thorax. The outcome was a surprise; the obtained results were just the reverse of what was expected. But the results were uniform and unmistakable. We shall state them very briefly. They are as follows:

In all animals in which the vagi were stimulated, left and right ventricles stopped beating after death sooner than in the controls. The interval between the time of death and the beginning of the rigor in the left as well as in the right ventricle is in the experimented animals shorter than in the controls. The time elapsing

between the beginning of rigor and the attainment of its maximum is in the stimulated animals again shorter than in the controls.

We shall not burden our present statements with figures or other details. The essential point in our results is that with regard to the cardiac rigor, stimulation of the inhibitory nerves had the same effect as that obtained by stimulation of motor nerves upon skeletal muscles, although the two kinds of nerves have opposite functional characters. How is this puzzling result to be explained? We are inclined for the present to give our results the following interpretation. It is known that anemia and venous stasis hasten rigor. The onset of rigor in the lower extremities of a living animal following compression of the abdominal aorta is a well-known experiment. We believe that the frequent standstills and slowing of the heart with its attendant anemia, venous stasis and asphyxia of the tissues is the cause of the hastening of cardiac rigor in the animals whose vagi were stimulated. In support of this interpretation we may cite the fact that the rigor of the skeletal muscles also sets in earlier in the animals whose vagi were stimulated than in the controls, a fact for which the disturbance of the circulation seems to be the only possible explanation.

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The antagonistic action of calcium upon the inhibitory effect of magnesium.

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Calcium and magnesium are chemically closely related elements. They are also close companions in the tissues of the animal body. It is the prevailing view that the physiological effect of both elements is similar in character. Many physiologists are at present of the opinion that calcium as well as magnesium exerts an inhibitory influence in the functions of the animal body. Loeb published in 1899 his observations of the inhibitory action of calcium upon the twitchings of frog muscles brought on by solutions of sodium chloride. It was then assumed by Loeb that all