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ON THE ACTION OF THE SO-CALLED "INHIBITOR," "ACCELERATOR" AND "DEPRESSOR" NERVES OF THE HEART.

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(Continued)

THE SO-CALLED "ACCELERATORS" OF THE HEART.

The sympathetic nerve derives its motor fibres from the spinal cord, through nerve filaments passing through the anterior spinal nerves to the corresponding parts of the ganglionic chain lying in front of the vertebral column. The connecting fibres between the two systems which supply the branches of the sympathetic nerve as accelerators, pass from the cord to the inferior cervical ganglion.* The distribution of the nerves of this ganglion to the cardiac plexus varies much in different animals. Thus in the frog no sympathetic nerves reach the heart, and the vagus is the only link of communication between it and the cerebro-spinal centres. In the dog and cat the vagus and sympathetic are united in a single trunk; ‡ while in the rabbit and higher mammalia they are distinct, though intimately and variously connected through the interlacing of their branches. These differences serve to account for minor differences in the effects of experiments on these animals.

Dr. Burdon-Sanderson lays down the rule that "the pulse is retarded by *increase*, accelerated by diminution of arterial pressure;" † increase of arterial pressure attending arterial contraction, and lessened arterial pressure resulting from dilatation of the vessels. The rule quoted however is by no means an invariable one, and Dr. B. Sanderson's adjacent pages shew sundry exceptions. The effect produced on the heart through the nerves we are considering, is much more uniform as regards blood pressure than as regards the frequency of the pulse, which under varying conditions of the animal may be frequent and strong, while in many cases increased frequency denotes cardiac weakness, so that as Prof. Kuss remarks, "the frequency of the pulse yields no indication as to the state of the circulation properly so called" (Lec. p. 168).

As the sympathetic has its origin in part in the medulla oblongata, section of the cord at the atlas necessarily affects this nerve and its ramifications, and the effect is to produce a fall in blood pressure (arterial relaxation). On the other hand faradization of the cord below the point of section, causes an increase of blood pressure (arterial contraction). Von Bezold found the heart's action increased in frequency after faradization of the cord, and regarding the faradic current as an excitant, thus apparently stimulating the heart, he applied the term "accelerators" to the nerves, which connect the cord with the cardiac plexus, through which this effect seemed to be produced. But Ludwig and Thiry showed that faradization of the distal end of the spinal cord, divided at the atlas, caused a rise in blood pressure in the systemic vessels after the *communicating filaments which through the sympathetic, connect the cord with the heart were severed*, showing that it could not be through the direct agency of the "accelerators" on the heart, that blood pressure was increased or the pulse accelerated. Ludwig and Thiry inferred from this that the cord has no real influence on the heart itself, but upon the peripheral circulating system."*

Ludwig and Cyon "proved by new experiments that this influence on the peripheral circulating system "is chiefly effected through the splanchnics, the great vaso-motor nerves of the abdominal viscera. Cyon has further demonstrated that this vaso-motor influence may be also induced reflexly through a centripetal nerve—to be hereafter referred to as the "depressor:" † and Dr. Burdon Sanderson in a special section devoted to the "functions of the accelerator nerves admits that V. Bezold *was wrong in believing that the spinal nerves have any power of augmenting the energy of the heart's contractions, or of causing it to do more work in a given time.*" ‡

*Handbook p. 315. † Ib. p. 23

* (Prof. Kuss Lectures on Phys. (Duval Amory) p. 167). †Ib., p 168. ‡ Handbook, etc., p. 231.