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

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Physiological writers invariably teach that the pneumogastric nerves (vagi) exert a restraining or inhibitory influence over the motor ganglia of the heart, whereby its pulsations are normally rendered slower. The reasons assigned for this opinion are, chiefly, that after section of the vagus, in some animals, the heart beats faster, owing to an assumed paralysis of its inhibitory nerves; while the application to the vagi of a faradic current, which is regarded as a stimulant, slows the heart, and if strong enough, arrests it altogether,—owing to an increase of the inhibitory power placing such an embargo upon the motor ganglia of the heart as to render their power ineffectual to continue its action.

In what is to follow, we are about to call this hypothesis in question, and to appeal to authentic physiological facts in so doing.

The vagus is not a simple nerve-cord connecting the medulla oblongata with the heart, on which experiments can be made implicating this nerve alone. It is in most intimate connection with the roots of the sympathetic in the medulla oblongata, with the fibres of this nerve in the cervical spine, with the cervical (spinal) plexus, with the cervical sympathetic ganglia and the nerve-cords and plexus from these surrounding the great systemic vessels and with the ramifications of the same nerve in the lungs. It materially influences the heart through the respiratory process and the pulmonary circulation; and it is, besides, in reflex relation with the splanchnics,—the chief vaso-motor nerves of the abdominal viscera. Indeed from the medulla oblongata to the splanchnics, embracing the vagus and the intermediate spinal and sympathetic nerve centres, we have a great nervous circle, the several parts of which are *en rapport*, reflexly, and so mutually influence each other, that it seems impossible to disturb the functions of any without causing a perturbation in the whole.

It has been demonstrated that in so far as respiration is concerned, the vagi are sensory or centripetal nerves, and act reflexly through the medulla and cord on the phrenic and other motor nerves concerned in respiratory action.* There is proof that the vagi, instead of being direct motor nerves of the heart (as their assumed inhibitory action necessitates) have centripetal functions as regards the heart also, and modify its action, when they do so at all, indirectly through the cord, the sympathetics and the nervous circle referred to above. This proof consists in the fact that section of the vagi has no effect on the pulse, if the cord below the medulla be previously divided :†—this operation effectually cutting off the channel of its reflex action through the circle mentioned.

We shall have to show, by and by, on direct physiological authority, that neither the so-called "accelerators," nor the "depressor" exercises any *direct* influence on the heart's action; but that in so far as they modify it, they do so solely through "the peripheral circulation." This being the fact in regard to the "accelerators," which are simply a part of the sympathetic vaso-motor system,—and the fact being also shown that the pneumogastrics excite or depress this system through the medulla or spine,—all that is necessary to do is to shew that impressions made on the pneumogastrics are of a stimulating or paralyzing kind, in order to account for the vascular effects which follow.

Section of the vagus, we claim, sends a wave of molecular disturbance through the sensory or centripetal fibres of this nerve, which acts on the vaso-motor centre of the medulla oblongata as an irritation, equivalent to excitation, and is reflected as such upon the vaso-motor nerves of the cord and sympathetic ganglia, as a result of which the peripheral arteries are dilated. As a consequence, the transmission of blood through the systemic channels is facilitated, blood pressure falls, from lessened

* Handbook for the Phys. Lab., p. 336. Prof. Kuss, Lec on Physiol. Duval, Amory., p. 386. † Handbook, etc., p. 284.

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