strictly in accord with physiological principles, and moreover accounts for the arrest of the heart in diastole; the right ventricle being found choked with blood in consequence of its being no longer able to empty itself into the vigorously contracted pulmonary vessels.

Confirmatory proof of the position here assumed is found in the fact that not only does faradization of the vagus arrest the heart in diastole by its centripetal action, but faradization of the mesenteric nerves produces the same effect. The mesenteric nerves communicate with the vagus in the solar plexus, and are also in communication with the aortic plexus;* but that the effect in question is produced not directly on the heart, but through the centripetal channel of the vagus, the medulla, the spinal cord and the nervous circle referred to, is proved by the fact that previous section of the vagi, or of the cord below the medulla, prevents faradization of the mesenteric nerves from thus arresting the heart.†

Now this reiterated proof of the centripetal action of the fibres of the vagus through which these results are produced, shows that this nerve is not the channel for a peripheral inhibitory motor influence between the medulla and the heart, as its assumed inhibitory power necessitates it to be. Seeing also that besides the vagi, the sympathetic furnishes the only other medium of communication between the cerebro-spinal centres and the heart,‡ it is evident that it is through the sympathetic the heart is influenced in these experiments; and as already remarked, we shall see in treating of the "accelerators," which are a part of the sympathetic, that their influence over the heart is no more direct than that of the vagus, but is solely exerted, in the words of Physiologists, "through the peripheral circulation." We therefore claim that the physiological facts demonstrate, not only that the vagus is not an inhibitory nerve of the heart, but also that the vagi and so-called "accelerators" are not rivals or antagonists in influencing the heart, as is commonly asserted.

It is a little remarkable that while faradization of the vagus, or sinus venosus, arrests the heart's action, this result does not occur, if in a frog, $\frac{1}{1000}$th of a grain of atropia or less, is first introduced into the circulation. The problem for us here, is, why a paralyzer like atropia should prevent the effects of faradization, which we hold to be also a paralyzer of nerve tissue.

The explanation of the Physiologists is, that faradization is a stimulus, and stops the heart by exciting Ludwig's inhibitory ganglion, which in consequence, puts such an embargo on the two motor ganglia as to arrest their activity and so stops the heart: that atropia, by paralyzing this inhibitory ganglion, renders the subsequent excitation of faradization powerless, and the motor ganglia being unopposed, the heart continues to beat.

The chief objections to this explanation are:

That it necessitates two systems of motor nerves of antagonistic function.

It requires that the circulation of poisoned blood should paralyze one portion of nervous tissue and not another, in close proximity in the same organ, from the same dose and at the same time.

It requires that the faradic current should excite one nerve ganglion, and produce no effects on another in closer vicinity to the point of contact.

If the inhibitory ganglion be paralyzed by the atropia, and if electricity be a "stimulus," having an affinity for that ganglion, it ought to counteract the paralyzing effects of the atropia, and, (on the theory) still arrest the heart.

If the faradic current fails to excite the inhibitory ganglion because it is already paralyzed, why does it fail to excite the motor ganglion of the sinus, (which is assumed to be unaffected by the atropia,) and so drive the heart faster?

The explanation is not in accord with the centripetal relations of the heart with the medulla through the vagus.

There is besides the anomaly of predicating the *arrest* of the vital activity of an organ by *exciting* a portion of its mechanism. What would be thought of a theory for bringing a horse to a standstill by applying a particular kind of spur, or applying it in a particular place?

The explanation of the problem which we have to suggest is necessarily a matter of inference rather than of demonstration. It is that atropia may produce such a change in the molecular condition of the nervous (motor) ganglion of the heart, and of the vagus or spinal centres, as to prevent electricity from inducing in them its ordinary molecular disturbance, and that as a consequence. it fails to produce its characteristic effects on the

* Wilson's Anatomy, pp. 407, 441. † Dr. Burdon-Sanderson, Handbook, etc., p, 283. ‡ Handbook, etc., pp. 286, 287.