plexus of the sympathetic. Thus it has not been proved to enter the heart at all, though it probably does so. It is a centripetal nerve, and as a consequence its functional activity is not directed towards the heart, but towards the medulla oblongata.

To show how unworthy this nerve is of its present name, and how false is the idea naturally associated with it, as a "depressor" of the heart, we need only refer to Dr. Burdon-Sanderson again.* He states that neither section of this nerve nor faradization of its peripheral end, has any effects whatever on either the arterial pressure (in the carotid) or in the pulsations of the heart. If the central, or cranial end of the cut nerve be faradized, still there is "no change whatever either in the character or frequency of the pulsations; the only effects produced being diminution in pressure. In other instances there is perceptible slowing, but the variations of the two effects are never parallel." Further, "The diminution of the arterial pressure cannot be referred to any direct influence exercised by excitation (faradization) of the depressor on the heart, but to diminution of the resistance in the arterial system; *i. e.*, to relaxation of the minute arteries."† This conclusion is confirmed by other experiments to which we need not here refer.

Thus this nerve, like the vagus, has been shown to have no direct influence upon the heart, but to act centripetally on the medulla, and through this upon the cord and vaso-motor nerves. Cyon has shown that this reflex influence reaches the splanchnics, and through them modifies the vascularity of the abdominal viscera.

It becomes necessary to enquire how does the "depressor" produce even this moderate effect on the peripheral circulation? Dr. Burdon Sanderson says of it:—"The depressor contains centripetal fibres, the function of which is to diminish the activity of the vaso-motor centre and thereby diminish the arterial pressure." On this view, the depressor is to be regarded as a centripetal inhibitory nerve, restraining the power of the vaso-motor centre in the medulla, and of course its "excitation" by the faradic current is held to increase its inhibitory power, and by thus counteracting the centractile power of the vaso-motor nerves, it favors relaxation of the arteries, which result, on the accepted theory, is depending on vaso-motor paralysis, to which the exercise of this inhibitory power is equivalent.

On the theory we present, the faradic current, by paralyzing a sensory nerve (acting of course centripetally) induces reflex paralysis of the vaso-motor nerves, putting an end to the dilating power of these over the arteries, the calibre of which is reduced by the inherent contractile power of their muscular walls. It is quite true that faradization of a sensory nerve causes arterial contraction, and our theory is in full accord with the fact. But here is a nerve which resembles a sensory nerve, in that it acts centripetally, faradization of which *dilates* the arteries. Is our theory then at fault? If the depressor be a purely sensory nerve our theory is at fault. That the depressor is not a purely sensory nerve (although it acts centripetally) is shown from the stand-point of the physiologists themselves, by the fact that the inhibitory system of nerves is regarded by them as separate and distinct from the nerves of common sensation; and also for the reason that if an inhibitory nerve annuls the power of motor centres in nerves, it must itself possess motor power. No such function can be predicated of purely sensory nerves. An inhibitory nerve, even if it act centripetally, therefore differs materially in function from the proper nerves of sensation.

Again the effect of faradization on this nerve shows that it is not an ordinary sensory nerve, for the faradic current acting on sensory nerves causes arterial contraction, but acting on this nerve it causes arterial dilatation. Furthermore, faradization of a nerve trunk produces anæsthesia or paralysis, and not pain, in the nerve trunk (see quotation from Dr. Radcliffe, ante), and if pain be caused by it, the pain is produced through the agency of sensitive nerves in the tissue in which the faradized nerve terminates, the muscular fibres of which are thrown into vibratile spasms. When therefore Prof. Kuss tates that faradization of the central end of the depressor nerve "is painful," the legitimate inference is that the painful sensation originates at the termination of the nerve fibres in the medulla. All that is necessary to produce this result here, is that the current should act on the contractile connective tissue of the medulla as it invariably does on such tissue elsewhere,

* (Handbook, &c., pp. 288, 291). † (Ibid., p. 292). (Prof. Kuss see p. 163). (Handbook, etc., p. 291).