the organ is accounted for by *direct paralysis of this motor ganglion.*

But the heart is also brought to a standstill, if by pressure, ligature, or other means, the contact of blood with its interior or endocardium is prevented.* Perhaps it would be better to say, the heart is arrested when its interior motor ganglia are no longer supplied with the blood and pabulum on which their generation of the necessary nerve force depends. This deprivation of blood either to the endothelium or to the ganglia, or to both, is brought about by the spasmodic constriction of the sinus at the point of its entrance into the auricle, which is the spot faradized in the experiment, as stated by Dr. Burdon-Sanderson.† When the spasm has time to relax,—in other words when the paralyzed nerves of the part have time to recover, as they slowly regain their wonted power, they are able to restrain the contractile power of the muscular fibres of the orifice, to dilate it, and as blood again enters the heart, and reaches its ganglia, its pulsations are gradually resumed, as recorded by Weber. Doubtless both the direct paralysis of the motor ganglion and the constriction of the orifice of the sinus act together in arresting the heart, and surely either view is as natural and as truly physiological as to assume that in a conflict of antagonizing nerves in the heart, one overpowers the other. The force of this last remark is heightened when it is remembered that the faradic "excitation" is really applied in closer proximity, if not directly to, the motor ganglion in the sinus, than it is to the inhibitory one in the septum, and yet of the two, it is only the latter which is "stimulated." Had the motor ganglion of the sinus been also "excited," and it is really difficult to see how it could escape, the two rivals might have fought it out indefinitely!

As to the arrest of the heart by faradization of the trunk of the vagus, it might suffice (in view of the facts elsewhere presented) to point to a paralyzing wave transmitted to the same chief motor ganglion; but the compound functions of the vagus and its extensive connections render it highly probable that the arrest of the heart is here due to a combination of causes.

The reader of Prof. Kuss's excellent Lectures on Physiology (Duval, Amory) will find a reason for the arrest of the heart by faradization of the vagus, altogether apart from any inhibitory influence of this nerve, in the spasm produced by that agent in the diaphragm and expiratory muscles, resulting in compression of the heart and serious disturbance in the relative pressure at the arterial and venous extremities of the circulating current. Thus the highest point of blood pressure is at the origin of the aorta, where it may be represented as 1-4th or 25-100ths of the weight of the atmosphere: the lowest is at the entrance of the vena cava into the right auricle, where it may be set down at 0. Spasm of the expiratory muscles, or a very violent effort at expiration, raises blood pressure at the latter point from 0 or 1-100ths of the atmosphere to 16-100ths, "an enormous pressure for this part of the circulatory system, an essential feature of its working condition being the absence of all pressure." So soon as this pressure in the vena cava becomes equal to that in the aorta, the circulation ceases,* and Weber has shown that the effects of a very forcible expiration (which is equivalent to a much less spasm of the expiratory muscles than that produced by faradization) slows and soon stops the heart.†

While faradization of the central end of the cut vagus, and especially of its superior laryngeal branch, affects respiration in the way just indicated through the medulla oblongata, and the circuit of the phrenic and other motor nerves of respiration (Kuss, 336),—faradization of its peripheral end, which ramifies in the lungs, and there, as elsewhere, is intimately associated with the sympathetic,‡ cannot fail to produce contraction of the muscular fibres of the pulmonary tissue, especially that of the envelope of the alveoli, known as "the muscles of Reissesen,"‖ thus preventing access of air to the cells, which of itself suffices to arrest the circulation, and with this, the heart's action. But paralysis of the vagus, through its connection with the sympathetic at various points in the thorax as well as in the lungs, may be held to paralyze also the dilating power of the pulmonary vaso-motor nerves; contraction of the pulmonary vessels necessarily follows, arresting the circulation, which in turn is followed by speedy cessation of the heart's action.§ This explanation suffices for the arrest of the heart by faradization of the vagus without invoking any "inhibitory" motor power on the part of that nerve, and is the more feasible in that it is

* Kuss, Physiol., p. 169. † Handbook, etc., p. 276.

* Handbook, etc., p. 218. † See Prof. Kuss, Lectures, etc., pp. 143, 313, 314. ‡ Dr. Carpenter's Phys., p. 155. ‖ Prof. Kuss, p. 296. §Dr. Carpenter's Human Phys., p. 536, etc.