SPASM OF THE GLOTTIS DUE TO NERVE PARALYSIS.

We now come to a still more striking illustration of the truth of the proposition laid down above. The aperature of the glottis is closed by one set of muscles and opened or dilated by another. The constricting muscles are the arytenoidei and crico-arytenoidei laterales, while the dilators of the glottis are the crico-arytenoidei postici.

Dr. Burdon Sanderson states that "the widening of the glottis is a condition of general muscular relaxation." He further states that the closing of the glottis is equally due to a general contraction of all the muscles; so that the glottis is closed, "not because the postici crico-arytenoidei muscles and the other dilating muscles * do not act with the rest, but because they are overpowered by the constricting muscles (*a*). The situation thus depicted becomes quite remarkable and full of interest, when it is remembered that the sole motor nervous supply to both these sets of muscles passes through the inferior laryngeal (or recurrent) nerves, a branch of the pneumogastric, and that when this nerve is cut or paralyzed, the closure of the glottis takes place, as a result of spasm of both of the antagonizing muscles, as just stated. On page 318 of the Hand-book the same eminent physiologist, describing the effects of section of the vagi, says: "The glottis is partially closed, just as it is in death." How the glottis is closed in death will appear from the fact, vouched for by Dr. Austin Flint, in the 5th edition of his "Practice of Medicine," when he says, the operation of passing a probang within the larynx, "is extremely difficult, if it be practicable, on the cadaver" (*b*).

There can be no doubt about the effect of the section referred to being of a paralyzing character, so far as the nerve is concerned, seeing that the simple section of the nerve during life, and the extinction of all nerve force in death, lead to precisely the same results as regards the closure of the glottal aperture. Dr. Burdon Sanderson adds that, "in animals with divided vagi, life may be prolonged by tracheotomy," showing how complete and fatal is the spasm thus produced. Other evidence of similar import is not lacking. Thus, Dr. Austin Flint, discussing the "danger of death from suffocation" in the "obstructed inspiration" occurring in nervous aphonia, says: "The condition is analogous to that after the physiological experiment of dividing both recurrent laryngeal nerves" (*c*). The same author has "reported a case in which the left recurrent nerve being situated betwen a calcareous deposit and an aneurismal tumor, spasm of the glottis occurred so frequently and to such an extent as to prove fatal" (*d*).

Now, since the recurrent nerve is the only motor nerve supplying these muscles, and since section or pressure on a nerve trunk cannot increase nerve activity—the nerve trunks being mere carriers and not producers of nerve force—it is evident that no other conclusion is possible than that the spasm here referred to is due to the absence of nerve force, and not to a stimulus from excited nerve action. And since nerve paralysis is thus shown to be directly the cause of spasm of the glottis, is it not necessary to infer that whatever is done by reflex action to cause spasm of the glottis must be of a paralyzing character to the nerve also? Thus, what is vaguely called "irritation," by which is usually meant an excitation or exaltation of nerve power, and which consists really in a perturbation of nerve force, must necessarily be an influence of a paralyzing character to the nerves it traverses. Such reflex "irritations" are usually attributed to brain lesions, to indigestible food, and other causes of a more or less debilitating character which may well arrest, rather than develop, the flow of nervous activity.

If it be true, that pain is "an expression of impeded and imperfect nerve energy, not of heightened nerve function," for which there is high authority (*e*), how much more is the perturbation of the nerve molecules, which constitutes "irritation," a disturbance of normal activities which is equivalent to paralysis.

RELATION OF VASO-MOTOR NERVES TO THE ARTERIAL MUSCLES.

I propose to show here, on the very best physiological authority, that what is known as "paralytic hyperæmia" is—contrary to the accepted opinion—venous and not arterial.

I need not delay to offer proof that the middle muscular coat of the arteries is under the control of the vaso-motor nerves of the sympathetic, which regulate the calibre of these tubes; or that the

* There are no "other dilating muscles" than the crico-arytenoidei postici.

(*a*) Hand-book, p. 308. (*b*) Ib., p. 294.

(*c*) Prac. of Med., 5th Ed., p. 309. (*d*) Ib., p. 371.

(*e*) Anstie, "Neuralgia," pp. 12 and 163.