

**Vincent, S.** VAGOTONIA AND SYMPATHICOTONIA. [Endocrinology, Vol. 1, p. 459.]

An editorial writer in the *B. M. J.*, Feb. 8, 1919, commenting on this communication says a good deal has been written lately about vagotonia and sympathicotonia, terms introduced by Eppinger and Hess to account for and describe nervous symptoms caused by abnormal activity of the vegetative nervous system. These names are somewhat impressive and require some explanation; by vagotonia is meant increased tonicity of what Hess and Eppinger called the autonomic (parasympathetic) system, namely, the cranial, medullary, and sacral outflow of the vegetative nervous system. Langley originally introduced the words "autonomic system" for what was formerly spoken of as the sympathetic, and it is unfortunate that Eppinger and Hess should restrict its use to a part only of the whole, thus causing some confusion. Sympathicotonia refers to increased tonicity of the sympathetic or the remaining part of the vegetative nervous (old sympathetic) system, and is supposed to depend on an increased continuous secretion of adrenalin into the circulation, vagotonia being regarded as the result of an excessive amount of a hypothetical substance, autonomine, the counterpart of adrenalin. Normally there is an equilibrium between the autonomic and the sympathetic systems, but when from any cause this balance is disturbed, the action of one becomes predominant and symptoms result. By means of drugs which act on the two divisions of the vegetative nervous system these disturbances become more pronounced and the diagnosis of the respective conditions is facilitated; thus, the subjects of vagotonia are sensitive to pilocarpine and physostigmine, and the manifestations thus produced are counteracted by atropine, whereas the patients with sympathicotonia are made worse by adrenalin, and although they are benefited by atropine it does not act as an antidote to the same extent as in vagotonia. Two papers bearing on the subject may now be briefly referred to. In a destructive criticism of the whole theory Swale Vincent urges that as there is no proof of a continuous secretion of adrenalin into the circulation the grounds for the imaginary existence of autonomine are based on a false analogy, that Eppinger and Hess have evolved this hormone out of their inner consciousness, and that the whole conception is highly speculative and has little or no experimental or clinical evidence in its favor. Matsuo and Murakami (*Arch. Int. Med.*, 21, 1918, p. 399), of the medical clinic of the Kyoto Imperial University of Japan, accepting with some modification Eppinger and Hess's views, investigated enteric fever in which there is sometimes an unexplained slowness of the pulse. In 46 cases of bacteriologically proved typhoid fever injected with atropine under the same conditions as Marris's cases, they found that atropine was quite effective in quickening the pulse, especially those with bradycardia. Marris, as is well known, found that atropine did not quicken the pulse

in enteric fever to the same extent as in healthy persons or in patients with diseases other than enteric; and for the divergence of their results the Japanese observers cannot offer any explanation. From examination of the vegetative nervous system in 38 cases of enteric fever by means of injection of atropine, adrenalin, and pilocarpine they concluded that 14 cases corresponded to vagotonia and 11 to sympathicotonia, so that the majority of the cases showed one or other condition. In many of the cases with well marked slowing of the pulse vagotonia was present and may therefore be the cause, whereas many of the cases without a slow pulse showed sympathicotonia. The 5 fatal cases out of the 38 all showed sympathicotonia, and as this state is often accompanied by a rapid heart, which has long been recognized as a bad prognostic in typhoid, the existence of sympathicotonia may be of significance in the prognosis of the disease.

**Jackson, D. E., and Pelz, M.** THE DISTRIBUTION AND FUNCTION OF CERTAIN NERVES IN CHELONIANS. [Proceed. Am. Soc. Pharmacol., XI, 2.]

In the turtle the intrinsic muscles of the lungs are controlled by two sets of nerve fibers. One of these sets runs in the vagus nerve for at least a part of its course and perhaps throughout its entire length. Stimulation of this set of fibers causes contraction of the lung. The second set of fibers are apparently sympathetic and emerge from the cord in the region of the anterior thoracic nerves. Stimulation of these fibers with a weak tetanizing current causes relaxation of the lung. It seems probable that some constrictor fibers run in the same nerves as those which carry the dilator fibers. We have not been able to follow the course of the dilator fibers completely but have obtained evidence of their existence and action by stimulating the main sympathetic trunk in the anterior thoracic region. Stimulation with a weak current at this point causes the lungs to relax. In many specimens we have failed to obtain this result, and in these cases we have considered our failure to be due either to poor technic, seasonal variations in the response of the nerves and tissues of the animal, or to anatomical variations in different species and specimens. This last feature is especially noticeable, and in snapping turtles we have not been able to identify some of the nerve fibers in the other common species. In box turtles the sympathetic fibers emerging from the thorax give off a branch which passes forward and joins the vagus in a large ganglion. If this branch of sympathetic fibers be stimulated with a *strong current* below the ganglion, *i.e.*, before it has joined the vagus, a contraction of the lung will be produced. It seems probable in this case that some constrictor fibers pass through the sympathetic to the ganglion on the vagus and thence through the vagus to the lungs. The records obtained in these experiments were produced as follows: The animal is