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2. The lungs receive from the thoracic portion of the vagus the pulmonary or bronchial nerves, the so-called anterior branches of which, in conjunction with filaments of the sympathetic, form a plexus on the anterior wall of the bronchus, and enter with the latter the lungs, while the posterior branches, together with those coming from the four upper thoracic ganglia of the sympathetic, are distributed in the same way on the posterior surface of the bronchus. They are the motor nerves for the unstriped muscles of the bronchial tree.

The diseases of the bronchial nerves, which produce, as it seems, a faulty innervation of the circular muscles of the bronchi, give rise to the morbid condition which has lately been the subject of much controversy, and is described under the name

Bronchial Asthma, Asthma Bronchiale s. Convulsivum s. Nervosum, Spasmus Bronchialis (*Romberg*).

Pathology.—Opinions about the nature of bronchial asthma are still divided. While some (Störk, Fräntzel) maintain that it is due to an acute swelling of the bronchial mucous membrane, others (Bamberger, Wintrich) consider a tonic spasm of the diaphragm to be responsible for it; still others (Trousseau, Biermer) believe it to be a vagus neurosis, supposing, in consequence of a disturbed innervation (vagus), a tonic spasm to take place in the circular muscles of the medium-sized and fine bronchi, thus producing an acute pulmonary emphysema. After Bert had shown, in 1870, by experiment that a contraction of the medium-sized and finer bronchi could actually be produced by irritating the vagus, later Biermer worked out his theory so thoroughly, and has defended it so successfully, that, in spite of the objections recently raised by Schmidtborn (Volkmann's Samml. klin. Vorträge, 1889, No. 328), who considers a vascular spasm in the distribution of the pulmonary artery to be responsible, we are probably justified in accepting it as correct, especially as with its help all the characteristic symptoms, the sudden onset and the sudden disappearance of the attacks, the expiratory dyspnœa, the low position of the diaphragm, etc., can well be explained. It is clear that this bronchial spasm forms an impediment much more easily overcome by inspiration than by expiration, and that this difficulty in expiration must of necessity not only influence the alveoli, but also the smaller bronchi, from which the inspired air can only imperfectly be forced out; hence arise dyspnœa and emphysema during expiration. On auscultation, sibilant rhonchi are heard all over the chest. But all this does not explain the cause of the spasm. This may be sought for in an independent affection of the bronchial mucous membrane, a view which possibly may be supported by the presence in the sputa of asthmatics of the so-called "Curschmann's spirals" (spiral threads which must be looked upon as casts of the finest bronchioles), and of so-called hæmosiderin cells found by v. Noorden, which are identical with pigment cells (Zeitschr. f. klin. Med., xx, 1, 2). Or we may assume a reflex origin. Thus Leyden maintained that certain pointed octahedral crystals which he discovered in the sputa of asthmatics irritated the mucous membrane, and produced the spasm. Many observations, however, allow us to doubt the correctness of this latter view. It has been established, on the other hand, beyond doubt (Voltolini, Hack, Sommerbrodt), that certain diseases of the nasal mucous membrane (polypous growths, chronic catarrh, etc.) may give rise to asthmatic attacks—reflex neurosis; possibly some part in the production of these is played by the reflex dilatation of the vessels in the bronchial mucous membrane, which was by Störk and Weber supposed to take place in connection with the bronchial spasm, a theory which was after-

ward confirmed by Sommerbrodt. With reference to this connection I have convinced myself from long experience with such cases that the above-mentioned affections only lead to asthma in persons with a nervous predisposition; they are only the "*agents provocateurs*," not the real cause (Brissand, Revue de méd., 1890, 12). This is especially the case in children (Blache, Étude sur l'asthme chez les enfants, Paris, 1890).

Symptoms.—The characteristic features of the disease are the paroxysms of distress and dyspnœa, previous to which the patient may for days complain of general malaise, be low-spirited, and troubled with digestive disturbances, diarrhœa, etc. The attacks begin quite suddenly, usually at night, more rarely in the daytime; during them the respiration is changed, so that the breathing in inspiration, but more especially in expiration, becomes labored and accompanied by a loud wheezing. This may last only a few hours or may continue for days, and may be repeated at varying intervals. Toward the end of the attack moist râles can be heard on auscultation, and there is expectoration which contains the above-mentioned spirals and crystals. Between the attacks the patient enjoys perfect comfort.

Ætiology.—The ætiology of the disease is but little known. No doubt hereditary predisposition does exist, and persons with a neuropathic family history fall, *cæteris paribus*, more easy victims to asthma than others. Just of what nature the exciting causes of the actual outbreak are we are as yet unable to say. We have repeatedly observed that hysterical persons suffer from asthmatic conditions, which, on examination of the respiratory organs, prove to be of a nervous origin. In these instances the patients are for days troubled with paroxysmal dyspnœa, their expiration is difficult and wheezing, while nothing abnormal is found on auscultation and percussion. We shall later on have more to say about this hysterical asthma.

That the inhalation of certain kinds of dust may give rise to asthma, while not a frequent, is certainly a well-authenticated observation. We may especially find this connection when the same obnoxious causes have been acting frequently and through a rather prolonged period of time, as is the case in those who follow certain occupations (millers, bakers, etc.); in the same way it is well known that repeatedly druggists have been affected regularly with asthmatic attacks while occupied with the pulverization of ipecacuanha root, and that the dust of certain kinds of grain—for instance, of oats—causes such disturbances

in those engaged in thrashing (cf. Hirt, Krankheiten der Arbeiter, 1871, Bd. i, p. 12).

The asthma which develops under the influence of certain poisons has to be classed among these cases, and in this connection the so-called lead asthma (asthma saturninum) is deserving of special mention. This is a very peculiar disease, which sometimes sets in very acutely only a few minutes after the work has been taken up. Though to the highest degree distressing to the patient, a fatal outcome in it has never been noted (cf. Hirt, *op. cit.*, Bd. iii, p. 40). This trouble is, however, even among lead-workers, quite rare, so that we may assume that among one hundred affections due to working in lead two instances at most of this above-described asthma occur. As to the mode of origin, we do not know whether to refer it to the action of the poison on the central nervous system, or on the peripheral nerve-endings of the vagus.

Treatment.—We are not acquainted with any specific for bronchial asthma; the much-recommended iodide of potassium (2.0–5.0 (xxx to lxxv grs.) a day) often fails, and, as a rule, we do not accomplish much with the usual nervines, arsenic, quinine, bromide, etc. From the use of electricity we have never seen any lasting benefit. Well-conducted hydrotherapeutic measures may produce a decided decrease in the frequency and the severity of the attacks. For the treatment of the attack itself we can foremost recommend pyridin, which was suggested by Sée. It is a product obtained in the dry distillation of organic substances, a colorless fluid which easily evaporates in the air. For the therapeutic use half a teaspoonful of it has to be poured on a shallow dish, and this inhaled three to four times daily in a closed room. The smell is horrible, and often disgusting, but in many instances the action was found extremely beneficial. As soon as the pyridin evaporates the patient becomes easier, the feelings of distress are relieved, the heart's action is more regular. The effect is not always lasting; still, I have seen cases in which daily regular inhalations used for several weeks have not only cut short the individual attacks, but have also decreased their frequency. Of course, with this, as with all other remedies, we may be disappointed. From the inhalation of the fumes of burning saltpetre paper, which has recently again been recommended by Kochs, I have only seen transient, never any lasting effects. The same holds for the well-known stramonium cigarettes, for amyl nitrite, and the vapors of tur-

pentine. More good may be expected from the administration of tinct. lobeliæ, which often works like a charm (tinct. lobel., 5.0 (ℳ lxxv); aquæ laurocer., 15.0 (℥ iv). Sig.: 15 to 20 drops every two hours). An alkaloid "lobelin" has been used by Nunes (Rio de Janeiro, 1889). With the extract of quebracho, which has been recommended by Penzoldt, I have no large experience of my own. Hyoscyamine, together with small doses of strychnine, given several times a day, has been used by Walker (Lancet, August 20, 1887, p. 368).

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B. Cardiac Affections due to Lesions of the Vagus.

The superior and inferior cardiac branches are given off from the cervical and thoracic portions of the vagus; they join the cardiac branches of the sympathetic to form the cardiac plexus (superficial and deep). It has not yet been determined of what character these fibres are; there is, however, no doubt but that we have to distinguish inhibitory fibres, the stimulation of which diminishes, and accelerator fibres, the stimulation of which increases the number of heart beats. The sensory nerves of the heart are also furnished by the vagus.

Angina Pectoris.

Among the neuroses of the heart which probably are caused by a disturbance in the vagus, we shall first consider angina hysterica or angina pectoris (stenocardia, cardiac neuralgia, nervous heart pain), a disease of the true nature of which our knowledge is as yet quite imperfect, though its symptoms have been recognized for more than one hundred years (Heberden, 1772). Its cardinal symptom is a piercing, burning, paroxysmal pain in the region of the left nipple, attended with a sensation of impending death; it often radiates into the left arm, and even down to the finger tips, and may continue for minutes or hours. It usually begins without any premonition, and surprises the patient by day at his work, or wakes him up at night out of his sleep. The severity of the pain differs; in some cases it is moderate, in others it reaches an insupportable degree. Dyspnœa is not always present; the respiration remains sometimes regular and quiet, although the patient suffers from a distressing feeling of anxiety, and his skin is covered with a cold sweat. During the intervals, the patient feels perfectly well, unless there is a co-existing lesion of the heart muscle or valves.

The diagnosis may present some difficulties, since intermediate conditions between angina pectoris and bronchial asthma are met with, or a combination of the two conditions may occur.

The prognosis depends mainly upon the question whether we have to deal with a vagus neurosis, or whether some complication co-exists. If the myocardium, owing to disturbed intracardial circulation (caused, for instance, by atheroma of the coronary arteries and insufficient blood supply to the myocar-

dium, or by syphilis), has undergone pathological changes, death may occur during an attack. Such cases are not rare, and I have recently again had occasion to observe an instance of this kind, in a man of robust appearance who suffered from stenocardia, and who, while in apparently good health, died suddenly in an attack within two minutes after its onset; the arteriosclerosis was very pronounced. Sudden death, however, is never to be feared unless the heart is organically diseased. It is impossible to give an absolutely favorable prognosis with regard to recovery, because here also we do not possess any remedy which is capable of doing away with the attacks entirely. But the same suggestions as have been made for the treatment of bronchial asthma apply to cases of angina pectoris, and about the same results have been obtained in both. If internal treatment can not be dispensed with, digitalis may in the first place be tried, then strophanthus, and finally arsenic, which latter may with advantage be combined with strychnine. With the tinct. piscidiæ erythrinæ, which is supposed to lower arterial tension and which has been recommended by Liégeois, I have no personal experience. It is prescribed as follows: Tinct. pisc. erythr., 60.0 (℥ xv); tinct. veratr. virid., 10.0 (℥ ijss.); tinct. aconiti, 15.0 (♏ 225). Sig.: 15 to 20 gtt. t. i. d.

For the attacks, freshly prepared amyl nitrite, a few drops (5 to 10), to be carefully inhaled by the patient, is the most useful treatment; besides this, inhalations of chloroform and hypodermic injections of morphine deserve recommendation, as they relieve the patient at once from the intolerable torments of his condition. The severe states of collapse following these measures, observed by Bamberger, are probably, after all, quite exceptional.

Murrell recommends a systematic treatment with nitroglycerine (Therap. Monatshefte, 1890, iv, 11), beginning with 0.0001, increased gradually to 0.003 p. die. From external measures, such as the application of hot-water bags or ice-bags over the heart, as well as from hot baths, I have seen no good result.

The ætiology of the disease is as obscure as its nature: here we must again carefully discriminate between the cases where the angina pectoris is merely a symptom of some organic heart disease (disease of the coronary arteries, fatty heart, valvular disease), and where it appears as an independent affection—i. e., where no heart lesion can be demonstrated. The latter form is disproportionately less frequent (Gauthier). Males and those

advanced in age seem especially predisposed to the disease (Gauthier); yet the author has also seen cases where displacement of the uterus was accompanied by stenocardia, as well as cases of undoubted angina pectoris in children thirteen to fifteen years of age. Psychical disturbances, such as are found in hysterical patients, also the influence of certain poisons—e. g., tobacco—deserve some consideration. Peyer (Zürich) claims to have observed a connection between stenocardia and spermatorrhœa (Wiener med. Presse, 1892, 25). That angina pectoris is a vagus neurosis can reasonably be accepted, as the sensory fibres of the heart are furnished by the vagus, and as pain is the most prominent symptom of the trouble. Presumably the sympathetic is, however, also concerned, and some are even inclined with Lancereaux, who several times found this nerve vascularized, to regard the cardiac plexus of the sympathetic as the chief seat of the disease; but even were this so, we could not exclude some participation of the vagus. A publication of Leroux, who found at the autopsy a bronchial gland and the right vagus grown together in a case where anginal seizures had existed until just before death, appears also to speak in favor of an implication of the latter nerve. Frequently no anatomical lesion can be found.

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