Wood (H. C.)

Reprinted from
UNIVERSITY MEDICAL MAGAZINE,
March, 1891.

CARDIAC NERVE STORMS.

Essential Tachycardia.

BY H. C. WOOD, M.D., LL.D.,

Professor of Therapeutics, and Clinical Professor of the Nervous System, University of Pennsylvania.

REPRINTED FROM
UNIVERSITY MEDICAL MAGAZINE,
March, 1891.

CARDIAC NERVE STORMS.[1]

Essential Tachycardia.

BY H. C. WOOD, M.D., LL.D.,

Professor of Therapeutics, and Clinical Professor of the Nervous System, University of Pennsylvania.

WHEN, in response to the request of the Council of the Philadelphia Neurological Society to deliver an address at this meeting, I cast about me for some subject concerning which it was possible for me to say something that might be of interest to you, the only one which presented itself with any force was "Cardiac Nerve Storms." A search through the latest and fullest treatise upon nervous diseases in the English language, that of Professor Gowers, showed that it contained no allusion to any of the several affections which it was my thought to discuss, and emboldened me to call your attention to the matter.

In the present state of physiological knowledge it does not seem possible to form a definite, clear idea of the nature of a "nerve storm." It is, indeed, uncertain whether the sensory disturbance that accompanies a locomotor ataxia crisis, or a megrim, has anything common in its nature with the no less furious motor disturbance that manifests itself in an epileptic convulsion; it may be that the one is due to a negative condition of the nerve centre, whilst the other is the outcome of a positive, active state. However this may be, it is certain that the term "nerve storm" suffices well, for the purpose of the clinician, to designate a great, sudden disturbance of the nervous system, either sensory or motor, which comes and goes like a tempest in the larger world about us.

[1] Address before the Philadelphia Neurological Society, January 18, 1891.

For the study of their clinical aspects, cardiac nerve storms may be divided into those which are sensory, and those which are motor, *i. e.*, into those in which the chief manifestation is pain, and those in which movement and not pain is the striking feature.

Pain in the region of the heart is the inseparable companion of lesions of the heart itself. The distress and agony of a failing heart muscle, an occluded coronary artery, or a diseased cardiac valve, is evidently the outcome of the local lesion. True angina pectoris may possibly exist, as a functional or organic centric nerve disorder, but, to my thinking, it is probably always the result of a more or less obscure organic disease of the heart itself. It comes not, therefore, within the horizon of our present view.

The hysterical mimicry of it needs, however, a passing notice. Rather oddly, it may be, it has been my lot to see this hysterical angina more frequently in men than in women; and sometimes the complaints of pain are so urgent that the clinician may well be deceived. Usually, however, a little care will show that the facial expression and the general acts of the patient are not in accord with the alleged intensity of suffering, that emotion plays an important *rôle* in the bringing on of individual attacks, that other evidences of hysteria are visible, that no failure of pulse occurs during the paroxysm, and that it is impossible to find an antecedent history of alcoholism, rheumatism, syphilis, excessive obesity, heart strain, advancing age, or other well-known cause of true angina pectoris. I have seen thrombosis of the coronary artery occur in a young subject and produce symptoms that might possibly be ascribed to a neurosis. In such a case, however, there must be always a peculiar expression of gravity in the face and acts of the patient which an experienced physician could hardly fail to recognize.

In 1879 Vulpian called attention to the frequency of valvular disease of the heart in locomotor ataxia, and his observations have since been confirmed by both French and German writers. It is usually, but not always, the aortic valves that are involved. I am inclined to believe that in many of these cases the lesions are *de facto* trophic, *i. e.*, secondary to nerve lesion. This seems to be especially true of cases like those reported by Teissier, in which the aortic valves were punched as with a perforating ulcer. Whether this trophic view be or be not correct, certainly in some of these cases, and I believe also in cases in which no valvular lesion can be detected, violent nerve storms occur, accompanied with excessive pain—nerve storms similar in character to those which constitute the so-called gastric crises of posterior sclerosis, and entirely different from the paroxysms of true angina pectoris.

As illustrating cardiac crises, I report the following case, made more interesting, perhaps, by the fact that the patient had been seen before he came under my observation by several of the best clinical teachers of this city without the true nature of his illness being recognized.

Mr. B., brewer, aged 71. In spite of long-continued military exposure and hardships, and of excessive use of alcohol over many years, Mr. B. had been a very powerful and vigorous man until about 67 to 68 years of age, when he began to suffer from vague rheumatic pains about the head and thorax, and occasional evidences of vertigo when getting up from

a chair, or, more especially, when rising from a horizontal position. These symptoms increased, and after a year or two were followed by attacks of excessive thoracic pain. These spells had increased in frequency and violence until I saw him. There was also, during this time, great complaint of a peculiar acrid, "coppery," bitter taste, recurring at intervals, and so annoying as to throw the patient into paroxysms of rage. Even before the first of the thoracic attacks the patient had noticed that he had great difficulty in buttoning his clothes, and at the time I saw him he was not able to dress himself. There were numbness and paræsthesia in the arms and hands, with great sensitiveness to temperature, any slightly warm substance producing violent burning pain, so that it would be dropped at once. Tinnitus aurium and slight progressive deafness were also present.

At the date of my visit the paroxysms were very frequent; they would come on suddenly, the pain becoming excessive in a few minutes. A spot of intense, horrible burning would develop over the middle of the right axillary region, accompanied by a sensation described as like a tight band constricting the thorax even unto death, and also by frightful shooting pains through the chest into the abdomen. In a little while the abdominal pain also would grow into an agony, with violent retching and futile attempts at vomiting. The heart's action was rapid and arhythmic; the pulse feeble, hobbling, irregular, intermittent. During the agony there was great mental excitement, and in the acme of the paroxysm an overwhelming oppression and feeling "that the room was too small" would force the patient into the open air; when in the open air unrelieved, he would exclaim "that the whole world was too small to contain him." During the height of the attack the excitement was so great that it was almost impossible to restrain the patient from getting out of bed, walking the floor, and even rushing into the streets. Shudderings, furious cursings, wild ejaculatory cries, screams of frenzy, threats of suicide, were but the manifestations of a despairing fury of agony, which in a number of instances ended in determined attempts at self-violence and at suicide. One very violent crisis terminated in a convulsion, from which the patient passed into a condition of stuporous melancholy, with strong suicidal impulses.

There was, when I saw Mr. B., a distinct systolic mitral murmur, and without doubt a valvular insufficiency, which had led to the diagnosis of organic lesion with secondary anginose attacks. The character and seat of the pain—the facts that its chief centre was axillary, that it was burning, that it radiated so forcibly into the abdomen, that it lasted for many hours, that it was accompanied by vomiting and often a perfect frenzy of restlessness and rage—made, however, to my mind, highly improbable the theory that the pain was truly cardiac, which improbability was greatly increased by the circumstance that there was no shortness of breath or distinct failure of circulation between the paroxysms. This doubt was changed into conviction by finding, first, disordered sensation and co-ordination in the hands, though the only signs of ataxia in the legs or gait were a curious inability of the patient properly to define the exact position of his legs when his eyes were shut, and some sluggishness of the knee-jerks; second, distinct Argyll Robertson pupils. The diagnosis of a posterior sclerosis commencing in the upper segments of the cord was confirmed by the after history of the case.

Slowly the pain centre worked downward, until it became distinctly abdominal, and the manifestations were chiefly those of a gastric crisis. Still later there developed bladder crises, paroxysms of burning, lacerating agony in the region of the bladder, with violent straining, and with screaming and incessant attempts at urination. There was the wildest mental excitement, ending almost invariably, unless morphine were given hypodermically, in frenzied attempts at suicide. Still later in the case lancinating pains, with marked paræsthesia, appeared in the legs. Finally, loss of co-ordination, absent knee-jerks, and ataxic gait completed the picture of locomotor ataxia.

The case which I have just recorded illustrates so clearly the diagnostic rounds by which the clinician must climb to a knowledge of the true nature of a cardiac crisis, that it seems hardly necessary to say more upon the subject, and I pass, therefore, at once to the second portion of my address, namely, a study of those cardiac nerve storms in which the disturbance is purely motor. In

doing this it seems proper to say a few words concerning some rare and curious cases which I suppose must be known as epileptic, although not only are the symptoms peculiar, but also the best results are obtained by treatment different from that adapted to ordinary cases of epilepsy.

In ordinary epilepsy disturbances of the circulation appear to be secondary and subordinate, but in the cases of which I am now speaking they are exceedingly prominent, and, indeed, dominant. I have also obtained the best results in treatment by the use of cardiac remedies, so that one is half disposed to believe that the epileptic convulsions are, in fact, due to the existing cardiac hypertrophy, and are not simply true epileptic nerve storms which have been modified by co-existent heart disease. The following case is a typical one:

Mr. R. S. was first brought to me in the latter part of 1886 by Dr. R. S. McCombs, of this city. In 1880 he had suffered from a violent attack of cardiac inflammation, probably endo-pericarditis, but had since been free from marked cardiac symptoms, except occasional palpitation. He gave no history of habitual shortness of breath, although he was not able to take violent exertion.

In 1883, he began to have epileptiform attacks. These attacks were stated to come on without aura, and in those seen by Dr. McComb without any primary pallor at the moment of seizure. During the attack there is excessively violent heart action, with intense flushing of the face, and a rapid formation of punctate ecchymoses over the face, and a general oozing of blood from the surface of the face, so that it can be wiped off. Sometimes there is hemorrhage from the nose. The conjunctiva is always intensely congested, swollen, and not rarely ruptures during the attack, so that the blood runs down over the face. When the attacks come on, according to the father's statement, Mr. S. always pitches forward, and usually is quiet, though on several occasions there have been violent general convulsions, and the tongue has at various times been badly bitten. No severe hemorrhage has been noted from the tongue, but if the head be cut in the fall it bleeds furiously; and on one occasion, when the conjunctiva was scratched by a broken spectacle-glass, half an ounce of blood is said to have been lost from the scratch in fifteen minutes, and the bleeding to have continued for two days before it could be arrested. After the attack Mr. S. usually goes to sleep and remains very dull and heavy for hours. Ten to twelve hours after the paroxysm a second one is very apt to occur. The unconsciousness is, during a paroxysm, always complete: the onset is absolutely abrupt.

An examination of the heart showed great enlargement of the area of percussion dulness; a diffused, very powerful impulse, most concentrated about two and a-half inches below the nipple, and a loud, blowing, second-sound murmur heard all over the cardiac region, as far to the right as the right nipple; very pronounced at the sternal notch, but loudest and strongest at the ensiform cartilage, with a distinct blowing murmur in the carotid and also at the back.

There had been a number of the attacks, and the free, long-continued use of the bromides had failed to modify either their frequency or severity. He was placed upon the use of large doses of aconite in addition to the bromides which he had been taking, with the result that no paroxysm occurred for three months, during which time his general health much improved. The next paroxysm was attended with general convulsion, extraordinary facial ecchymoses and free bleeding from the conjunctiva.

It may be interesting to note that the continued use of aconite and bromide has kept the symptoms in check until the present time, and that the only attack during the present year occurred whilst Mr. S. was ill in bed with "*la grippe.*"

The term *tachycardia* signifies excessively quick heart action, and of course is, in accordance with its etymology, applicable to any case with rapid pulse. Fortunately, the word has not yet been used with sufficient frequency or con-

cord to affix upon it, by popular acclaim or habit, any distinctive meaning. Some term is certainly needed in modern medicine for use as the generic name of diseased states in which violent action of the heart exists without being dependent upon organic disease of the heart itself, or upon acute febrile disturbance, or upon a general constitutional disease, such as exophthalmic goitre; and I propose the restriction of the name tachycardia to those cases in which very violent heart action occurs without obvious reason. Most of these cases belong to one of three classes: First—Those in which there is paralysis of the pneumogastric or inhibitory nerve. Second—Those in which the cardiac disturbances are reflex. Third—Those in which the affection may be strictly considered to be a neurosis.

First.—Paralysis of the pneumogastric nerve is a rare disorder, of which, however, medical literature contains numerous illustrations. It may be centric or peripheral. Thus Dr. H. Doelger, in a Wurzburg Thesis, 1883. recorded a case of apoplexy in which the glosso-pharyngeal, the hypo-glossal and the facial nerves were involved, and in which there was also a pronounced acceleration of the pulse, which rose to 168, with other symptoms indicative of pneumogastric palsy. In this case there can be scarcely any doubt but that the inhibitory centre in the medulla was involved in a small clot or embolus. More frequently the pneumogastric paralysis is peripheral rather than centric, and it is commonly due to pressure by cancerous or other tumors; whilst the records of diphtheria seem to prove that it may be the result of a peripheral neuritis involving the pneumogastric nerve.

Second.—Irritation of a sensitive nerve is apt to cause slowing of the heart action by stimulating the pneumogastric centre. It would seem, however, that there may be a reflex tachycardia, for although such cases must be rare, and I cannot remember to have seen one, Rommelaere reports one in which he asserts that an extremely rapid cardiac action was caused by the irritation of a biliary calculus; and in the paper of Proebsting may be found details of three cases in which a pulse of from 200 to 250 was believed to be reflexly caused by irritation of the female sexual organs.

Third.—Violent heart action, due to hysteria and allied conditions. This is so common that it is not necessary to do more than to allude to it, and to call attention to the fact that a violent emotion may, without causing distinct hysteria, produce at once an excessive tachycardia, which is liable to recur afterwards without obvious cause. As an illustration may be related a case reported by Prof. Gerhart, in which a violent fright produced in a young woman a pulse of 120, which persisted for some time, and was accompanied with a slight cardiac murmur, the paroxysm breaking off rather abruptly and recurring afterwards. Carafy reports a somewhat similar case to this, as happening during the Paris siege. The pulse in this instance reached 228, but exposure and privation, and not simple emotion, appear to have been active causes, so that probably exhaustion was a large etiological factor. Under the present heading, also, may be considered such a case as that reported by Traube, in which tachycardia developed after excessive exertion in running quickly up long flights of stairs.

The cases of tachycardia which I have grouped together under these headings can scarcely be considered to represent true nerve storms, and are only related to cardiac motor nerve storms by the similarity of symptoms, and the fact that a few cases of habitual cardiac nerve storms of the character spoken of have had their origin in acute attacks produced in one of the ways mentioned.

Such causation is parallel to what occurs in the history of nerve storms not connected with the heart. Thus, an epilepsy in its first paroxysm may indeed be a true essential epilepsy, but, on the other hand, the so-to-speak, accidental epileptic attack, due to the abuse of alcohol, recurring several times from the direct effects of the poison, may have for its outcome an essential epilepsy, which continues, although the poison is no longer taken into the system.

In order to distinctly separate in nomenclature the cases of rapid heart action just spoken of from true cardiac motor-nerve storms, the latter may be called "*Paroxysmal Tachycardia*," though there is reason to suspect that even paroxysmal tachycardia, as thus defined, would represent two or more affections. At least it is certain that in some of these cases obvious cause has existed before the primary attack, whilst in others it has been wanting. Hysteria, also, may closely mimic the true paroxysmal tachycardia, but what affection does it not simulate?

Cases in which a paroxysmal tachycardia commences in a definite cause are illustrated by one recorded by Prof. W. Winternitz, in which a woman, aged 40, after excessive emotion had a violent attack of tachycardia, and henceforward suffered almost daily from paroxysms which commenced with pallor in the face, a sense of anguish in the heart, a pulse from 230 to 260 a minute, with vibratile motion over the heart and distinct increase of the percussion dulness, the attack going off with a few very powerful and slow contractions of the heart, followed by a long, slow, dicrotic pulse, which in a few minutes became natural. No treatment was of any avail, but the attacks ceased spontaneously when the woman became pregnant. Zunker records an instance of paroxysmal tachycardia in which the primary paroxysm had been produced by excessive exertion, but recovered without special cause. These attacks were very long, and the pulse reached 220. There were also contraction of the pupils, cyanosis and some œdema of the lungs.

It is probable that these cases, and especially the last one, differ from that affection for which I would propose the name of "*Essential Paroxysmal Tachycardia.*"

The definition of the disease would be a recurrent paroxysmal neurosis, in which attacks of excessively rapid heart action occur without obvious immediate or predisposing cause, and without pronounced pain or excessive cardiac distress, the pulse rising to 160 and upward, the sounds of the heart remaining normal, and it being frequently possible to arrest the attacks by drinking a glass of cold water and certain other procedures of apparently trifling import, the disease having apparently no tendency to shorten life or to develop organic disease, and being entirely compatible with great mental and physical activity.

Prof. Nothnagel[1] puts on record cases of tachycardia, several of which certainly represent the disorder now under consideration. In the most characteristic case the man was subject to violent heart action, which would come on abruptly, even during absolute quiet, without pain, but with some feeling of anxiety; in a moment the pulse would rise to 180, the sounds of the heart remaining normal.

The length of the attacks was from a few minutes to several hours. Sometimes they would be shortened by the patient taking a long breath, and whilst still holding his breath, drinking a glass of cold water. When about half a glass was taken the heart would suddenly quiet down, three long slow beats would follow one another and the normal beat be attained. Sometimes, however, the attack, instead of being shortened by this procedure, would be only momentarily interrupted. There was no evidence of valvular or other heart disease during or between the paroxysms.

I myself have seen one typical case of essential paroxysmal tachycardia in the person of a physician who has long enjoyed the reputation and the twofold work of an exceedingly active country practitioner and of an aggressive reformer.

A very vigorous man, Dr. H. C., aged 37, received a sharp shock or jerk by stepping from a piazza to the ground which was further off than he thought. This was followed by a strange feeling of fulness in the chest, which was relieved by lying down on the left side. Two or three months after this there was a second, and a few months later a third attack. These came on without any apparent cause, with a slight jerk under the breast bone, and were accompanied by some sense of fulness of the chest and mild pains diffused down both shoulder blades. In the third paroxysm he first discovered that the pulse was so frequent and so indistinct that it could not be counted. After this the attacks recurred every few months, but were always quickly arrested by lying down upon the left side, and did not in any way interfere with the general health or business activity.

No change occurred for two or three years, when it was found that lying upon the left side no longer gave very distinct relief, and the paroxysms increased in length and severity. During their height, while the patient was lying or sitting still, the pulse would be 160 a minute, but upon any effort, such as walking, it would rise to 200 a minute. During absolute rest the only disagreeable sensations were fulness of the chest and slight pains under the right clavicle, but in a number of instances the attacks developed under such circumstances that continued walking or buggy-riding was a necessity, and on these occasions pains would come on in the back of the neck, associated with an almost complete inability to prevent the head from dropping forward. The onset of the paroxysm was habitually so abrupt that it has been compared by Dr. H. C. to the effect of the passage of a bullet, and the cessation was no less instantaneous than the coming on. The attacks frequently continued for hours, and the only remedy which was found to afford relief was morphia, until one day the patient by chance drank a full tumbler of cold water rapidly, when instantly the pulse dropped to its normal rate. Acting on this idea, for a time, whenever the attacks came on, a glass of ice-water was gulped down; at once the attacks ceased. Finally, however, this measure failed to give relief; then it was found that a cup of strong coffee, taken as hot and as rapidly as it could be, would produce instant arrest of the symptoms. This continued to serve until more than twenty years had elapsed from the coming on of the attacks, when it too became abortive.

Dr. H. C. is now over eighty, and since about his sixtieth year he has had these attacks on an average every three weeks; at times they have recurred three or four times a month. In 1886 Dr. H. C. suffered between March 25 and October 1, inclusive, from twenty-four attacks, some of them lasting fifteen hours. He was then eighty-two years old. About this

[1] Wiener Med. Blt., 1887.

time he heard that an attack could be arrested by holding the breath and bearing down as in defecation. On trial, the result was immediate, but after a few paroxysms the controlling power was lost.

Up to 1888, in their main points, the paroxysms had not materially changed, but they had grown longer, often lasting from ten to twenty-six hours. They had always commenced with a jerk under the sternum, followed by marked giddiness and a sense of warmth over the whole body. If the recumbent position upon the left side were assumed the giddiness ceased, but occasionally recurred during the attack, and could, at almost any moment, be produced by exercise. Dr. H. C. discovered that if he should rise suddenly and run or walk a few yards, the giddiness would come on with such force as to cause nausea, followed by abrupt vomiting, which in turn would put an end to the attack. This led to the habit of jumping up quickly and running a short distance during the paroxysm, with the result of giddiness, vomiting and immediate relief. After a time, however, this failed.

During these years the heart has been frequently examined by various physicians of high rank, both during and between the attacks, and at no time was there found a cardiac murmur. The paroxysms have occurred by day and by night, during exertion and during quiet, and have rarely if ever been produced by emotion or any discoverable cause. They have never been followed by any signs of fatigue or exhaustion, and after being kept awake by a nocturnal paroxysm for hours, the subject would go to sleep as soon as the pulse would become quiet, and awake the next morning without any sense of fatigue or disinclination to work. The health has continued perfect; and almost always, even during the height of the attack, it has been possible by an effort of the will and by interesting himself in reading, conversing or writing, for the subject to forget entirely the sense of fulness in the chest, or indeed the existence of the paroxysm, although the pulse continues unchanged. Lying upon the back or right side has always produced a slight sensation of suffocation, and even when the peculiar glow of heat over the whole body has been most pronounced there has been no rise of temperature that could be noted with the thermometer. Usually there is no excessive discharge of urine after the attacks, although sometimes it has been increased, which increase has been thought to have been caused by the large quantities of water taken to relieve the sense of thirst, and with the hope of relieving the paroxysm. Various medicines have been tried, but, as already stated, the only one which has ever afforded relief is morphia. On one occasion twenty drops of the fluid extract of veratrum viride were taken inside of four hours without producing any effect whatever. In another paroxysm, sixty drops of the tincture of digitalis were administered at a single dose; two hours after ten drops more were given, and again in another two hours ten drops more, without result.

In 1888 the power of arresting the spells by rapidly drinking ice-cold water was recovered. Early in 1889, Dr. H. C., after a great sorrow, began to suffer with pains which would begin under the right shoulder-blade, extending through to the right nipple and from this through to the right side of the chest. Never was the left side affected. After these pains had lasted for a few minutes the heart beat would become slow, dropping to eighteen a minute. There was with this no distress, no sense or feeling of threatened death, but occasionally complete momentary intermission of the heart's action. These attacks came back at intervals during three months, during which time there was no attack of rapid cardiac action. They then ceased, and the old attacks reappeared.

In October, 1890, Dr. H. C. entered his eighty-seventh year. He is still a vigorous man, able to ride about in his carriage over the country for many hours, and even to walk considerable distances. For a year or two, when he is absolutely at rest physically and mentally, his pulse rate, which in his younger days was seventy, has been fifty-two. The attacks are exceedingly frequent, occurring during the last year almost every day, but are very frequently put an end to by drinking cold water. The pulse rarely rises above 135, there is little or no distress, and only rarely do the attacks last more than a few moments, except that about every two or three weeks there comes one that will yield to nothing in the way of treatment until it has gone on for from four to eight hours.

The little effect that the attack has upon Dr. H. C. is shown by the fact that I have recently received from him a letter, written in the midst of one of his most severe paroxysms, in which the handwriting shows no tremulousness, and indeed in no way gives

evidence that it is the work of an old man. The handwriting and the diction are alike clear, rapid and firm.

When we come to consider the nature of the curious affection I have been describing, we find ourselves in the face of a difficult and perplexing problem. It seems to me, however, that certain elements of the problem which can be fairly made out ought to be stated. In the first place the attacks may be considered to be of the character of a nerve storm ; in the second place, they produce extraordinarily little effect upon the heart, the bloodvessel system and the general organization ; in the third place, they are probably not associated with, or dependent upon, any so-called organic lesion, but represent a functional neurosis ; (it being understood that most, if not all, of the so-called functional neuroses really depend upon organic change, but that we employ the term "functional" simply to mean that the organic change is not sufficiently pronounced to be recognized by our crude methods of investigation, so that in our terms, at least, it is supposed not to exist.)

It is only necessary to point out the curious parallelism which exists between the epileptiform convulsion and the tachycardiac nerve storm. Precisely as you may have an hysterical, a toxic, or a reflex epileptiform attack, so may you have an hysterical, a toxic or a reflex tachycardiac paroxysm ; and precisely as there remains after the separation of hysterical, toxic and reflex epilepsies a set of cases (essential epilepsy, so-called) in which the paroxysms come on without known cause, so also do there remain cases of tachycardia, which are properly classed as essential paroxysmal tachycardia.

It is a matter of considerable interest to decide what is the mechanism through which the excessive heart action is developed. Extraordinarily rapid cardiac action may be the outcome of pneumogastric paralysis, or of irritation of the accelerator nerves. Whether it can occur in a third way is, I think, in the present state of our physiological knowledge, uncertain. It is conceivable, but not, I think, proved, that changes in the nerve apparatus of the heart itself may bring about an exceedingly rapid heart action, since the original impulse of rhythmical movement occurs in the heart itself.

What, then, is the probable mechanism of tachycardia ? As stated in the beginning of this paper, the essential nature or organic mechanism of a nerve storm still remains obscure. Whether, however, we believe a sensory nerve storm like a migraine to be due to negative conditions of the sensory centre or not, the general consensus of professional opinion at present seems to be that the epileptic or motor paroxysm is due to a discharging lesion and is not the outcome of a temporary palsy ; so that analogy indicates that the tachycardia is due not to a paralysis of inhibition, but to a discharge of nerve force. Further, the researches of Gaskell have, I think, proved that the pneumogastric nerve has trophic relations with the heart muscle. This conclusion is also in accord with the discovery made by Eichhorst, and confirmed by Von Anrep, that in birds section of both vagi is followed by rapid fatty degeneration of the heart. In view of these facts, the lack of distress, the absence of increased arterial pressure or general disturbance of the circulation, the failure to effect the nutrition of the heart, of the bloodvessels, or of the general system (which are so

characteristic of essential paroxysmal tachycardia), all seem to me to negative the idea that the paroxysms are the outcome of pneumogastric paralysis. Again, the slight influence which the affection has upon the heart or the general nutrition makes it, to my thinking, at least improbable that the paroxysms are due to changes in the structure of the nerve centres in the heart itself. Although I confess that at present we have not proof that the cardiac ganglia are not diseased in tachycardia, the aggregated facts seem to me to strongly indicate that the tachycardiac paroxysm is caused by a discharging lesion affecting the centres of the accelerator nerve. Some of the reasons for this belief are so obvious that I shall not take time to point them out, but it may not be wasting your minutes to call attention to the fact that, in lectures published in 1879, Francois-Franck asserted that any great increase of the pulse, which in the animal is produced by stimulating the accelerator, is not accompanied by any increase of the arterial pressure, or by any augmentation of the work done by the heart. Francois-Franck believes that he has shown that this is because the acceleration of the heart's action is primarily due to the shortening of the diastole, and that, therefore, during the heart's systolic contraction, so little blood is expelled from the heart that the aggregate amount which passes through the ventricle during a minute is not increased. The conclusions of Francois-Franck are, I believe, generally adopted by physiologists; certainly Prof. Foster, in his text-book, gives them the stamp of established truths. If they be correct, and if, as is almost certain, the accelerators have no trophic relations to the heart, it is evident why rapidity of pulse, solely due to accelerator irritation, should produce little effect upon the heart and general system.

One of the most curious phenomena of tachycardia is the arrest of the paroxysms by the swallowing of cold water, which is probably a characteristic feature of the disease, since it has been noted in almost all the elaborately observed and reported cases. It affords a plausible argument against the probability of the rapid pulse being due to pneumogastric paralysis, since Dr. S. Meltzer has shown that swallowing is attended with a loss of tone in the vagi centre, and consequent weakening of cardiac inhibition and increased frequency of the cardiac beats. It is, however, probable that the arrest of the tachycardiac paroxysms by the swallowing of hot or cold liquids, is due to stimulation of the inhibitory cardiac centre, produced by irritation of the peripheral nerve filaments of the stomach. It is a well-known physiological fact that irritation of the abdominal nerves is capable of reflexly inhibiting the heart, and Dr. H. C. has found that the best method is to swallow the water as rapidly as possible and as cold or as hot as he can take it; if the water is only moderately cold, much larger quantities of it are required.

You no doubt, gentlemen, will agree with me that at present the physiological explanation of the tachycardiac paroxysms remains uncertain, but this is no reason for refusing to acknowledge the affection as a distinct neurosis, for are we certain of our theories in regard even to such a familiar matter as the epileptic convulsion? And who has yet established beyond cavil the explanation of the "white fingers" of Reynaud's disease? Assuredly, positive knowledge as to the nature of very many neuroses still remains but the hoped-for fruit of future research.