Gentlemen, we may be still very far from the realization of the position which some of us think we ought to occupy in relation with the public and with the State. But I believe we are moving towards that end; progress even if slow is still progress; and the objects we have in view, whether they be purely scientific, or ethical, or political, must, on the whole, be promoted by such an association as this, heartily supported by the members of the profession, and wisely directed by its elected officers.

I will conclude with one more quotation from Faust, but the speaker is not this time the arch-Fiend:

Expression, graceful utterance, is the first
And best acquirement of the orator.
This do I feel, and feel my want of it.

An Address

ON THE

OCCURRENCE OF EPILEPTOID ATTACKS IN TACHYCARDIA AND BRADYCARDIA.

DELIVERED BEFORE THE BORDER COUNTIES BRANCH OF
THE BRITISH MEDICAL ASSOCIATION.

By FRED. HUGH CLARKE, M.B., C.M.,

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PRESIDENT OF THE BRANCH.

[AFTER thanking the members present for the honour of appointing him President of the Branch for the year, Dr. Clarke said:]

The subject I have chosen for this address is one which, from an accidental circumstance, I have been deeply interested in since the date of my graduation, now some twenty-five years ago. Till quite recently literature on the subject practically did not exist; records of a few cases, more or less carefully reported, with some theories, more or less wild, generally more rather than less, being all there was. Within the past two or three years all this is changed; admirable work is being done every day, and there are few lines of research being more carefully investigated by clinicians, physiologists, and pathologists than arrhythmia, heart-block, tachycardia, bradycardia—call it what you will. It is to two of these conditions, apparently quite the opposites of each other, but in reality very closely allied—namely, tachycardia, or quick pulse, and bradycardia, or slow pulse, with certain nervous conditions common to both—that I would ask your attention for a short time this afternoon.

"Tachycardia," according to Clifford Allbutt in his *System of Medicine*, denotes not merely any case of quick heart, but

an enormous quickening of the pulses of a heart not necessarily the seat of static disease, a quickening which attacks the patient suddenly, which does not persist indefinitely, but for a variable space, rounded off by an equally sudden reversion to the normal state, less certain phenomena of exhaustion.¹

It is to "certain phenomena of exhaustion," as Clifford Allbutt puts it, specially connected with the nervous system that I would particularly refer.

In the three or four cases I have seen there was no ascertainable heart lesion. After repeated attacks signs of hypertrophy may be noted, and are not uncommon, but this undoubtedly is an effect and not the cause of the condition. A systolic bruit may be developed during an attack, but disappears when it is over, as may the hypertrophy also.

An attack of tachycardia may last for hours, days, or even, in extreme cases, for a week or more. After a variable interval the heart tends to become exhausted, and irregularity occurs—in short, heart-block takes place. Stops are felt in the radial pulse, and concurrently nervous symptoms arise, which vary from the slightest feelings of sparks flashing before the eyes or the waving of a sheet before the patient to severe epileptiform convulsions, with loss of consciousness. Yet such attacks are certainly *not* epileptic.

I will now give the summary of a typical case of tachycardia which I was able to watch carefully throughout its course. I had the further advantage of being

associated in this case with Professor (afterwards Sir William) Gairdner and the late Dr. Finlayson of Glasgow, both of whom repeatedly saw the patient during attacks. This will be a guarantee that no point of importance was missed and of the accuracy of the observations.

Mrs. A., aged 63, mother of seven children. Her general health may be regarded as good and her organs free from disease. Heart normal, except for a somewhat diffuse apex beat, and, after her illness had continued for some weeks, a slight increase in the transverse cardiac dullness. No history of epilepsy or insanity in the family. In the summer of 1878, after a slight illness (influenza), she began to suffer from palpitation, or tumultuous action of the heart, described by her as "tumbling," and after a time irregularities in the beat. At the commencement had occasional feelings as of an electric wave passing from the heels to the head. No serious disturbance of health, however, occurred after this for two years, when on stooping to lift something she fell and cut her brow slightly. She seems to have been unconscious for a few seconds, and from this time for some three years her illness continued. At irregular intervals of a few days to one or two weeks, generally without obvious cause, the heart's action became very rapid and irregular. The irregularity continued, in the earlier stages of the illness, a few hours, later on two or three days. The longest attack was one week, during which time the heart was never regular for a minute.

As a rule, when the irregularity had continued for some hours, but sooner when very violent, pauses became noticeable in the radial pulse, some seconds elapsing before the next beat was felt. If the pause was not unduly prolonged, not more, say, than for four or five seconds, an expression of alarm on the patient's part was all that was noticeable. She said she felt as if "something were about to happen." If the interval were longer, say six to eight or nine seconds, the face became pale, the lips livid, there was a scream, the thumbs were drawn inwards, the head backwards, and there was convergent squint. In the milder attacks consciousness was not lost, and the patient was able to talk *immediately* afterwards about her sensations. In the more severe attacks—say, when the radial pulse disappeared for eight, ten, or twelve seconds—consciousness was lost, and the convulsive seizures were more severe. The abdominal pulsations, which were marked during the attacks of irregularity, went on after the radial pulse had ceased to be felt. So far as I could judge, slight contractions of the heart were present during the fit, but these were probably auricular only, and, if ventricular, very slight. The severity of each fit was proportional to the length of time the radial pulse was absent. At any stage of the fit a return of the pulse cut short the attack. The number of fits in each attack of irregularity varied from a few to many dozens, and were of all kinds of severity. As a rule, after a specially severe fit, the pulse within a few seconds, or after a few beats, became perfectly regular, and the patient was able to say that her heart had "settled," and that the attack was over. Unless from some exhaustion following, which was soon recovered from, the patient seemed little the worse, and would remain well till the next attack occurred—it might be days or a week or so after. This went on for three years with but little disturbance to the general health.

Three uric acid calculi were passed during these years, and from time to time some rheumatic or gouty symptoms developed in the smaller joints. Several of the attacks were attributed to fright, nervous anxiety, indigestion, etc., but as a rule no apparent cause could be found for them. Often she woke from a sleep with the heart beating violently. It seldom "settled" during a sleep.

The patient recovered perfectly, and enjoyed good health for three years, when a slight cerebral haemorrhage occurred. This was recovered from, but a second and larger took place after a year, which proved fatal. Her death occurred some six years after the first appearance of the nervous attacks. There was no *post-mortem* examination.

Another case of a somewhat similar kind I was able to watch for many years till her death early in the present year from cardiac failure following an attack of influenza.

The lady was unmarried, 65 or 66 when I first attended her, and, though neurotic, had mental powers far above the average. Following upon a prolonged and anxious illness of a near relative, there came at times marked turns of palpitation. The heart took what she described as "runs," when the beats seemed to merge into one another, and it became quite impossible to count the radial pulse. Such attacks lasted for a few hours or for a day or two, and came on at fairly regular intervals for nearly three years. The return to perfect regularity in the pulse was generally sudden. If she went to bed and stayed there during the attack nothing particular happened further than occasional sensations of waves of light passing before her eyes, and resulting exhaustion, more or less marked according to the violence of the palpitation and its duration. If she got up and moved about, however, a fit was almost certain to occur, precisely similar to an epileptic seizure. A former distinguished President of this Branch was dining at her house one evening when she fell on leaving the

dinner table. He attended to her, and told me afterwards that the attack was distinctly epileptic. Though she had many similar heart attacks for years after this, she never had another fit, as she was kept strictly in bed whenever the heart's action became irregular. She had no known heart disease, though signs of hypertrophy were noted in the later stages of the illness.

I have notes of several other cases of a somewhat similar kind, but the two already given illustrate sufficiently the special points it is wished to emphasize. Published reports of tachycardia are somewhat rare, but cases of bradycardia, from their greater frequency, now and then see the light. Though apparently the one condition is the extreme opposite of the other, yet the effects of tachycardia and bradycardia are often so exactly alike that you will perhaps allow me to describe one or two typical cases of slow pulse before offering an explanation of the phenomena noticed during its suspension.

The condition of bradycardia may be said to exist when the radial pulse is anything under 40 beats a minute. It may fall far below that number. Sometimes, beyond an abnormally slow pulse, there are practically no symptoms other than perhaps breathlessness on exertion; at other times a condition is set up of the most alarming and gravest nature.

A gentleman, about 70, now under my care, thinks himself pretty well if his pulse-rate is 40 per minute. It often falls below this; and at such times if he exerts himself unduly either physically or mentally his wife tells me he is apt to become suddenly pale, and lose all sense of his surroundings for a few moments. Absolute rest in bed, with some cardiac stimulant, invariably puts him right again in a few days. Though there is no valve disease, I think there is some serious deterioration of the cardiac muscle in this case. His son, aged about 30, and always delicate, showed the same peculiarity of pulse, though in a less marked degree. A few years ago I was asked to see him as he seemed listless and not well. I could find nothing wrong with him beyond a markedly slow pulse. He was ordered to remain in bed for a few days, and when sitting up for breakfast a morning or two later he fell back on his pillows and died almost immediately. Such a sudden collapse is not at all unusual in bradycardia.

Dr. Alfred Webster² gives particulars of a case which is so admirably recorded, that it requires no apology from me to give a short summary of it:

A weaver, aged 48, had never had an illness till a fortnight before his admission to the Western Infirmary. When at work, on stretching over his loom to tie a thread, he had a sensation of faintness and a feeling as though he would fall. He remembered nothing after that for some seconds apparently, but on recovering felt his face flushed, and his heart beating forcibly. He was taken home, and remained in bed for two weeks, having many similar attacks in the meantime. Sometimes the attacks followed one another in rapid succession, sometimes a day or more would elapse without any. After his admission to hospital sphygmographic tracings show that there were intervals of ten to fifteen seconds during which there was no appreciable pulse at the wrist (tracings shown). If the finger were kept on the pulse the following facts were noted: First, the pulse became suspended, and almost at the same moment the face became rapidly and extremely pale. If the stop were only of short duration, say for or five seconds, there was a gradual and crescendo return of the pulse, and a coincident flushing of the face. During these short pauses the patient appeared to be merely sleeping. Frequently, however, the pulse was suspended much longer—ten, fifteen, and twenty seconds elapsing between the beats. The pallor of the face became more and more marked, and the breathing very shallow. Later on, perhaps after fifteen seconds, spasmodic twitchings of the face were observed, going on if the pause were prolonged till the muscles of the neck, arms, and legs took part. Squint, either concomitant or conjugate deviation to either side, was observed, with widely-dilated pupils. When the convulsion was at its height respiration was noisy, with puffing out of the cheeks. The patient was quite unconscious during the greater part of the period. The return of consciousness was preceded by noisy breathing, and very marked flushing of the face. After the convulsion the patient appeared dazed, and as if just aroused from a deep sleep. The flushing of the face and return to consciousness were almost always simultaneous with the appearance of the radial pulse. Auscultation over the heart during the pauses discovered what appeared to be abortive beats of the heart, but these beats were not appreciable in the radial or superficial arteries. His radial pulse varied from 18 to 24 per minute.

This patient recovered to a great extent, had a relapse after a few months, and finally died at home about a year after his first seizure.

Napoleon Bonaparte was a sufferer from bradycardia, his normal pulse being 40 per minute. He undoubtedly had occasional epileptoid attacks in his later life, and

some years ago an elaborate article was written by Lord Wolseley accounting for the failures towards the end of his career by the fact that he became an epileptic. From all I have been able to find in various works of his life, I have no doubt his seizures were due to prolonged cardiac systole, and that he was not an epileptic at all.

Having now considered the clinical aspects of these two conditions, the question arises whether or not we can give adequate reasons for their occurrence. Without doubt we can, so far, at least, as the convulsive seizures are concerned. Suddenly withheld nutrition of the brain is the cause, and that without reference to any sudden change of blood pressure which may take place in the cerebral vessels. Langendorff³ in 1878, published an article on *Epileptic Convulsions in Peripheral Stimulation of the Vagus*, which I may quote, as his results have been confirmed by many others but never disproved. He experimented with rabbits, previously narcotized with chloral hydrate, about 7 grains, sufficient to abolish all voluntary acts, reflex activity being completely retained. He says:

Electrical stimulation of the peripheral end of the vagus causes a prolonged stoppage of the heart, with a very remarkable phenomenon, never hitherto observed so far as we know—a complete epileptic convulsion occurs ten or fifteen seconds after the diastolic pause. The eyes roll, the pupils at first contract then dilate, the muscles of the face twitch, the head is at first drawn back, then convulsively moved forwards. Violent convulsions of the fore and hind limbs next ensue, and often the abdominal muscles are tetanically contracted. Breathing, after a brief acceleration, stops, and in fact we have exactly the picture of convulsions from profuse hæmorrhage, so thoroughly described by Kussmaul long ago. Distinct paleness of the fundus oculi makes it clear that we have to deal with the results of cerebral anaemia.

Precisely similar convulsions are produced by compression of the thoracic aorta at a point nearer the heart than where the great vessels are given off to the head.

The description of these convulsions as epileptic I can by no means accept. They are merely anaemic convulsions, and agree in their origin with convulsions from suffocation.

Rosenthal, also, in his experiments on cardiac poisons, found that continued diastole of the ventricle produced general convulsions.

When the ventricles cease to contract, or contract so slightly that circulation is arrested, anaemia of the brain results, the severity of the succeeding convulsion being proportional to the degree of anaemia present—that is, to the period of delay in the ventricular systole.

This part of the subject having now been cleared up, we come to consider whether we can account for the abnormal rapidity or slowness of the pulse in tachycardia and bradycardia, and the pauses so often noticed. Theories or surmises innumerable have been suggested to explain the former condition. The familiar and attractive weakened condition of the vagi, irritated accelerators, hysterical ganglia in the heart itself, and sudden variations of the blood pressure may only be mentioned. Sir William Broadbent, at Toronto last year, said that the small, scarcely perceptible pulse sometimes found in tachycardia, and the sense of faintness might be due, not to weakness of the systole, but to the diastole being so short that the ventricles had not time to fill. That may be so, but the ventricles having had ample time to fill, what causes the delay in their contraction?

From the fact that atropine affects the auricles only,⁴ and from other experiments, we know that the ventricles are, to a very slight extent, if at all, under the control of the vagus. We also know that the cardiac ganglia are mainly concerned in controlling the blood vessels supplying the heart muscle.⁵ We know, also, that very marked changes in the blood pressure cause no such effects as those we have been considering.

Gaskell, in 1883, from experimenting on the heart of the tortoise, came to the conclusion that there must be some muscular connexion between the auricles and ventricles, propagating stimuli to contract from the one to the other. The nature of this connexion neither he nor any one else knew for the following twenty years, till his younger, in 1903, discovered the band connecting the two—now generally known as "the band of His." Since its discovery we have undoubtedly got beyond pure hypothesis to demonstrable fact as regards cardiac contractions. Of this peculiar fibro-muscular band and its functions I would remind you that it has been found in the heart of man and all animals examined. Structurally it is like nothing else found in the heart. It

begins at the anterior edge of the coronary vein, then passes forwards on the right side of the auricular septum, below the foramen ovale, lying close on the auriculo-ventricular septum, which passes obliquely from left above to right below. Just above the place of insertion of the median flap of the tricuspid valve the bundle forms a special knot-like thickening which sends processes down on either side of the ventricular septum under the endocardium, ramifying in the walls of both ventricles.⁶ The band contains not only nerve fibres but nerve ganglia, so that conduction by muscle alone is as yet unproved.

Erlanger and others have found that pinching this bundle in dogs increases the ventricular pause. A greater tightening of the clamp lengthens the pause till three or four or more auricular contractions occur for every one in the ventricle. A cut through the bundle is followed by complete diastole of the ventricle, and no stimulus can be made to pass from the auricle to the ventricle. What interests us more as physicians is that all cases of true bradycardia which have come to an autopsy since the band of His was discovered have shown extensive degenerative lesions in its structure.⁷

I would suggest, then, that tachycardia is a pure neurosis, characterized by a great increase in auricular contractions from disturbed nervous influences. After a time, from over-exertion, fatigue in the conductivity of the band of His occurs; missed impulses result, causing longer or shorter delays in the ventricular contraction. When these delays are sufficiently prolonged, cerebral anaemia is caused, followed in due course by the nervous symptoms described. Tachycardia being a neurosis, it is, like other neuroses, commonly recovered from. The condition may persist for years, and recovery may be complete, or relapses may take place from time to time.

In bradycardia we cannot be so hopeful, the ventricular pauses being due to serious structural changes in the band of His. Sclerosis of the vessels, fatty degeneration of the fibres, and an abnormal increase of fibrous tissue in the bundle have all been found. The condition is never recovered from, and the chances of sudden death from prolonged cessation of the ventricular contraction should not be forgotten. Life in true bradycardia, according to Balfour, is rarely prolonged for more than three or four years.

As regards treatment, *rest*, both physical and mental, so far as possible, is of greatest importance. In my case, strict confinement to bed prevented convulsive attacks. In another recorded case the patient "dodged" these when threatening by getting up in bed on his hands and knees, and keeping his head down. The head should be kept low, and the foot of the bed raised. Any deviation from health, such as indigestion, constipation, gout, rheumatism, etc., should be attended to. Massage also may be employed if the conditions allow.

In tachycardia I have pushed bromides till bromidism was marked, without good results—rather the opposite. They certainly have no effect in controlling the fits. Nerve tonics, such as valerianate or oxide of zinc, seemed to do good. The glycerophosphates or glyceroformates might be tried, but I have no experience of these in this connexion. Strychnine or nux vomica, atropine or belladonna, and alcohol are better avoided.

In bradycardia strychnine hypodermically, with heart tonics such as digitalis or strophanthus, seem to have been of use. Osler records a case of recovery after treatment with iodide of potassium.

With a better knowledge of the causes at work in producing both conditions, it is not too much to hope for more successful methods of treatment in the near future.

REFERENCES.

- ¹ Albutt's *System of Medicine*, p. 825. ² *Glasgow Hospital Reports*, 1900. ³ *Epileptische Krämpfe bei peripherer Vagus-Reizung*, O. Langendorff: R. Zander, Stud. Med., *Centralblatt für medizinischen Wissenschaften*, January 26th, 1878. ⁴ *Johns Hopkins Hospital Bulletin*, Baltimore, 1905, vol. xvi, p. 202. ⁵ Gaskell, *Lancet*, vol. ii, 1897, p. 827. ⁶ Aschoff, *BRITISH MEDICAL JOURNAL*, October 27th, 1906, p. 1105. ⁷ Erlanger, *ibid.*, p. 1113.

THE fourth Congress of Climatotherapy and Urban Hygiene will be held at Biarritz in Easter week (April 20th to 25th), 1908, under the presidency of Professor Pitres, President of the Medical Faculty of Bordeaux. Information relative to the Congress may be obtained from the General Secretary, Dr. P. Gallard of Biarritz.

BILATERAL LESION OF THE AUDITORY CORTICAL CENTRE: COMPLETE DEAFNESS AND APHASIA.*

By F. W. MOTT, M.D., F.R.C.P., F.R.S.

A FULL clinical account of this case is published in the *Archives of Neurology*, vol. iii, p. 401, but since then I have had the opportunity of making a more complete examination of the extent and limits of the lesion by means of serial sections through the right hemisphere in the region of the lesion and by a separation of the left hemisphere into two portions, so that the exact condition of the convolutions lying within the Sylvian fissure could be studied. Before proceeding to give a full description of the anatomical findings I will give a short account of the clinical notes of the case:

E. M. H., admitted to Claybury March 18th, 1897, married; age on first attack, 22; now aged 25.

Family History.—Father was a drunkard, and lived a fast life. Sister and brother were said to be hysterical. Some time prior to her marriage in 1894 she had an attack of hemiplegia and aphasia. The practitioner who attended her was communicated with and wrote as follows: I cannot remember the year I saw her, but I can the principal features of the case. When called to her she presented the ordinary symptoms of a hemiplegia with aphasia, but as I could not find any cause for the condition. I put it down to hysteria, and I think the subsequent course proved I was correct. If I remember rightly she had complete hemianaesthesia. The paralytic symptoms disappeared rapidly, but the aphasia remained complete for a week or more, and partial for something like a month. She completely recovered under tonics and valerian. She was married in July, 1894, and remained in good health, mentally and bodily, until the first miscarriage, February, 1895 (about six months fetus), in Beckenham Cottage Hospital, when she suddenly lost her hearing, and three or four days later developed paralysis in right leg and arm, had convulsions, and lapsed into unconsciousness. Later on she became a patient at the National Hospital, Queen Square. The following notes were sent from the National Hospital, Queen Square:

Notes from National Hospital, Queen Square.

E. H.—In November, 1894, had a fright followed by some fairly differences; seemed unnaturally worried. A fortnight later was vacant and absent-minded and stupid in the evening. Next morning, at 7 a.m., her husband found her quite helpless; had passed her water in the bed, could not understand what was said to her, but nodded appropriately in answer to written questions. During the day had another (?) fit, cried violently, but had no definite convulsions, but her husband noticed the bed shaking violently.

She was admitted to Beckenham Cottage Hospital completely paralysed on right side. After about a month began to recover her speech. The arm recovered rather rapidly and completely. On coming home the husband noticed she could not feel a prick or a pinch on the right leg (which was oedematous) but she could one on her arm. She was able to read throughout her illness. Her leg has rapidly recovered power; complains of pains in right shin. Memory unaffected, no alteration in disposition. Said not to be emotional.

Personal History.—Married in July, 1894. Had premature birth two months after onset of hemiplegia.

Family History.—No history of paralysis. One brother died of fright, aged 27; said not to be insane.

In 1893 patient had a similar attack after a fright, supposed ghost; was unconscious for three hours, lost her speech for two weeks, but there was no paralysis, and she rapidly recovered.

On admission, May, 1895. Right leg weak. Can only say "Yes" and "No"; understands all written commands; cannot write spontaneously. Apparently has attacks of giddiness. Nystagmus. Jaw deviates to R. Left side of mouth acts better than right. Deaf. Slight anaesthesia in right leg. Left knee-jerk more than right.

Notes of an Examination shortly after Admission to Claybury Asylum by Myself.—She is quite unable to comprehend a single word spoken to her, and is apparently stone-deaf. I was unable to convince myself of this, although it is probable, for a tuning-fork applied to the skull on both sides did not attract the slightest attention; but she was then absorbed in watching her observers, therefore it may have been due to inattention. The patient can understand written or printed language, and this is the only means of communicating with her. She appears to understand simple sentences—for example, Question I: "How long have

* This paper was on the list of papers to be read at the Medical Section of the meeting of the British Medical Association at Exeter.