to the tenth and twelfth are not uncommonly affected as well. The whole of these areas may be equally sensitive, or there may be one point in front or behind far more sensitive than the rest. Occasionally the hyperæsthesia is confined to one surface of the body, either anterior or posterior, and sometimes it is equally well marked on the left side as on the right. In one case under my care both the extent and the degree of hyperæsthesia were greater on the left side than on the right, without, so far as the appendix was concerned when it was exposed by operation, there being any apparent reason for it.

This local hyperæsthesia is evidence that the corresponding spinal centre is receiving from some part of the body to which its nerves are distributed stimuli of unwonted intensity and that it is in consequence unduly sensitive to ordinary impressions. These stimuli may come from the muscles. It is well known that the skin over the insertion of muscles that have been overworked is intensely hyperæsthetic. Or they may come from branches distributed to the abdominal wall. Quite recently I have had under my care a case of retroperitoneal abscess of very large size, not connected with any of the viscera, lying behind the liver and stomach, causing, as one of its most prominent symptoms, extreme sensitiveness of the skin on the right side of the body over the region of the sixth to the tenth dorsal segmental areas. Or they may come from the abdominal viscera, travelling to the centre along its visceral branches. But, as Head has pointed out,[6] they do not come from the peritoneum. So long as the inflammation is confined to the surface of the serous membrane there is no true cutaneous hyperæsthesia in peritonitis. Like McBurney's point, therefore, cutaneous hyperæsthesia over the region of the eleventh right dorsal segmental area may occur under a variety of conditions: the stimuli that cause the morbid activity of this centre may come along any of its branches, muscular, parietal, or visceral; but when the hyperæsthesia is definitely associated with other evidence pointing to inflammation of the appendix it may be taken as a clear indication that the wall of the appendix itself is involved and that, therefore, though the inflammation may subside, it will in all probability leave some permanent alteration in the appendix which will necessitate operation later.

There is one other point about the cutaneous hyperæsthesia in inflammation of the appendix which, if borne out by further observations, is of very great importance. Sudden cessation of the hyperæsthesia, without at the same time any corresponding improvement in the general symptoms, suggests very strongly that the appendix has become gangrenous and that immediate operation is absolutely necessary to prevent septic peritonitis. I have seen this in two well-marked cases of inflammation of the appendix and I have known a similar occurrence in a case of twisted ovarian pedicle in which also cutaneous hyperæsthesia was present at the first. The exact relation between these events is not obvious at first sight. It probably means that when the inflammation is so intense as to lead to gangrene either the nerves are no longer able to convey stimuli to the centre involved or that the centre itself is exhausted and can no longer respond. But whatever may be the explanation there can be no doubt of the fact that in these three cases the cessation of the hyperæsthesia coincided with the beginning of gangrene, and in such circumstances I regard nstant operation as imperatively required.

[6] Loc. cit.

SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.—The following officers and members of the council for the session of 1903–4 were elected at the annual general meeting of this society on July 23rd. Council: Dr. J. Ford Anderson, Dr. C. W. Chapman. Mr. H. J. Curtis, Mr. H. L. Carre-Smith, Mr. Clinton T. Dent, Dr. C. N. Gwynne, Dr. E. Hobhouse, Dr. H. R. Hutton, Dr. R. Hutchison, Mr. F. Jaffrey, Dr. C. J. Macalister, Dr. A. Morison, Mr. R. H. Parry, Dr. J. P. Parkinson, Mr. George Pernet, Mr. H. Betham Robinson, Mr. Sydney Stephenson, Dr. S. B. Smyth, Mr. Harold J. Stiles, Dr. Frederick Taylor, Dr. James Taylor, Mr. J. W. Thomson Walker, Mr. R. H. A. Whitelocke, and Dr. A. W. Wills. Honorary treasurer: Mr. R. Clement Lucas. Honorary secretaries: Dr. George Carpenter, Dr. Theodore Fisher, and Dr. G. A. Sutherland.

ON THE SO-CALLED STOKES-ADAMS DISEASE (SLOW PULSE WITH SYNCOPAL ATTACKS, &c.).

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DEFINITION.

Stokes-Adams disease is a clinical condition characterised by (1) a profound disturbance in the automatic mechanism of the heart—true bradycardia, hemisystole (false bradycardia), and allorythmia; (2) nervous symptoms—vertigo, syncope, pseudo-apoplexy, and epileptiform attacks; and (3) secondary symptoms—Cheyne-Stokes breathing, cardiac asthma, angina pectoris, and the vaso-motor accompaniment of profound heart-shock. The post-mortem changes are not constant. In a few cases coarse lesions of the nervous system have been found, in a small number no lesions whatever; in a large proportion of the cases arteriosclerosis is present. The clinical picture is very variable—there are acute, rapidly fatal cases, chronic cases in which for years the patient has slow pulse with syncopal or pseudo-apoplectic attacks, and forms in which slight but well-characterised attacks occur at intervals in persons apparently well.

HISTORICAL NOTE.

Adams's description is of interest.[1] His patient, a man, aged 68 years, was of full habit and subject to oppression of breathing and cough. When seen he was just recovering from the effects of an apoplectic attack which had suddenly seized him three days before, but he was well enough to be about the house and even to go out. What attracted Adams's attention were the character of the breathing and the remarkable slowness of the pulse (30 to the minute). His regular attendant informed Adams that during seven years this patient had had not less than 20 apoplectic attacks. After a day or two of heaviness and lethargy he would fall down completely insensible and on several occasions he had hurt himself. The pulse would become slower than usual and the breathing loudly stertorous. He never had any paralysis after the attacks. Death followed an attack and the heart was found to be very fatty. The valves were sound. There was no statement about the coronary arteries. R. W. Smith[2] refers to this case, and states that he had also noted a condition of slow pulse with fatty heart.

Stokes's much more important contribution is entitled "Observations on some Cases of Permanently Slow Pulse." He describes the case of a man, aged 68 years, who had recurring fainting fits which left him without any unpleasant effects. During three years he had had at least 50 seizures. They were induced by any circumstance tending to impede or to oppress the heart's action, as sudden exertion or distended stomach. He was never convulsed. The duration of the attack was seldom more than four or five minutes and during this time he was perfectly insensible. He had never been paralysed. When admitted his general health seemed to be very good. There was an apex systolic bruit and the pulse was 28 to the minute. The arteries appeared to be in a state of permanent distension, "the temporal arteries ramifying under the scalp just as they are seen in a well-injected subject." An interesting feature in this case was that the patient could ward off attacks by a peculiar manœuvre. "As soon as he perceived symptoms of the approaching attack he directly turns on his hands and knees, keeping his head low, and by this means he says he often averts what otherwise would end in an attack." While his heart was beating only at 28 Stokes noticed that there were occasional semi-beats between the regular contractions, eight of them in the minute. On his readmission there was noted a new symptom, a remarkable pulsation in the right jugular vein, which was more than double the manifest ventricular contractions.

Beyond the occasional reports of cases not much of special importance has appeared in England on the subject. The condition is only mentioned incidentally in Allbutt's "System of Medicine." Gibson discusses it in a very good section in

[1] Dublin Hospital Reports, vol iv., 1827, p. 396.
[2] Dublin Journal of Medical Science, vol. ix., 1836.
[3] Dublin Quarterly Journal of Medical Science, 1846.

his "Diseases of the Heart." In France Charcot called attention to it in 1872, but to Huchard we owe a revival of interest, and in the various editions of his "Traité des Maladies du Cœur," under the name Stokes-Adams disease is found the best description in literature. In Germany the condition has not attracted attention, but within the past two years excellent papers have appeared by His, jun., Hoffmann, Jaquet, and Luce in the *Deutsches Archiv für Klinische Medicin.* In the United States a most comprehensive study of the slow pulse was made by the late Dr. D. W. Prentiss[4] of Washington when he presented before the Association of American Physicians a remarkable case in which the condition had persisted for a period of nearly two years in association with very frequent fainting fits. Of the 94 cases which he abstracted from the literature there were 32 which belong to the condition under consideration. In the case reported by Dr. Prentiss there was a necropsy, and it is stated that neither the aorta nor the coronary artery was atheromatous, which is somewhat remarkable considering the extreme grade of sclerosis in the peripheral arteries. There were no lesions of the pneumogastric nuclei. In the discussion which followed the above case Dr. F. P. Kinnicutt and Dr. Jacobi narrated typical cases. In 1895 I described the condition briefly in my lectures on angina pectoris. Dr. R. T. Edes[5] has reported a series of cases and given an exhaustive analysis of the literature of the subject. Babcock has a good chapter in his recently issued work on "Diseases of the Heart."

CONDITIONS IN WHICH SLOW PULSE IS MET WITH.

1. *Physiological.*—The statement is made that dark persons and the dark races—e.g., Bretons—have a pulse rate below the average. It does not appear to be the case with the African race. I know a family most of the members of which have a pulse-rate of about 60; one son died from Stokes-Adams disease, another, perfectly healthy, has sometimes a pulse rate of only 48. Cases which can be called physiological are not very common. The slow pulse of Napoleon rests upon tradition;[6] it has been suggested that his epilepsy and attacks of apathy may have been associated features in a chronic form of Stokes-Adams disease. There are remarkable instances in the literature, many of them quoted by Dr. Edes, of persons in good health for years with a pulse-rate from 30 to 40. The slow pulse of old age may be considered normal, but the physiological limit is passed when with advancing arterio-sclerosis there are attacks of vertigo or syncope.

2. *Neurotic.*—1. Organic disease of the brain, the cord, or the nerves. In tumours, in meningitis, in injury of the spinal cord, and in pressure on the vagi bradycardia may occur. Dr. Edes gives a list of cases in the literature. 2. Functional. In nervous debility, in perverted states of the higher centres, as in melancholia and hypochondriasis, the pulse-rate may fall below 60; in neurasthenia bradycardia may be a special feature. We must recognise, too, that severe and fatal attacks of Stokes-Adams disease may occur without any discoverable lesion in the nervous system, the heart, or the arteries.

3. *Toxic.*—(1) Inorganic poisons, such as lead; (2) bacterial poisons, as in diphtheria and typhoid fever; (3) vegetable poisons, as digitalis and tobacco; and (4) metabolic poisons, as in jaundice, uræmia, the puerperal state, and after prolonged exertion.

4. *Cardiac and cardio-vascular lesions.*—Valvular, myocardial, and arterial, alone or in combination; a large majority of the cases of bradycardia with nervous symptoms belong to this group.

VARIETIES OF STOKES-ADAMS DISEASE.

What is called Stokes-Adams disease is in reality a syndrome or symptom-complex, not a disease with constant anatomical lesions and a uniform etiology. The cases may be arranged in three categories.

1. *Post-febrile group.*—Following, more rarely in the course of, an acute infection—such as typhoid fever, diphtheria, pneumonia, scarlet fever, or rheumatism—bradycardia may occur with vertigo, syncope, or epileptiform seizures. A fair number of such cases are on record, and while Huchard is not inclined to regard them as instances of Stokes-Adams disease the main features are not to be distinguished. The attacks may recur for weeks, as in Schuster's case.[7] This form seems more hopeful, though the first attack may prove fatal. The following is a good illustration.

CASE 1.—A physician, aged 35 years, had on Feb. 8th, 1901, a streptococcus pharyngitis of moderate intensity, followed by acute nephritis. He convalesced satisfactorily and went to Florida on March 12th. He felt very well and had been taking a fair amount of exercise. At 4.55 A.M. on the 21st he was awakened out of sleep with a very peculiar sensation of numbness in the fingers and he felt faint and was covered with sweat. The pulse was only 16 to the minute but very forcible at the radial artery. He fainted and the physicians found him in a condition of extreme cyanosis and collapse, the sweat pouring from him, with a pulse-rate of about 20, quite regular, and the same at the heart and wrist. He was given hypodermic injections of ether and inhalations of oxygen. At 9 o'clock his pulse was 30. In the afternoon he regained consciousness and the pulse rose to 50. He was dazed and had no recollection of the attack. The cyanosis was extreme. On the next day his pulse was 60 but it was two or three days before he recovered completely. From the statements made by his physicians it was evident that his condition must have been critical. When I examined him on April 20th he was a robust, healthy-looking man, rather stout. The pulse was regular, with no intermission, and 76 to the minute. The apex beat was within the nipple line. The sounds were clear and of normal relative intensity; there was no change in the recumbent posture or bruit after exertion. The pupils were equal. The knee-jerks were present. There were no signs of any disturbance in the nervous system. In such a case it is reasonable to suppose that myocardial changes had followed the acute infection and were responsible for the profound disturbance in the automatic action of the heart with an associated acute dilatation. It is worth noting that in Case 11 the slow pulse was first noticed after an attack of pneumonia.

2. *Neurotic group.*—(1) With coarse lesions of the nervous system, as pressure on the medulla following injury (Holberton), narrowing of the vertebral canal (Lépine, Boffard), tumour pressing on the vagus, and degeneration of the trunks of the vagi. There has been no observation showing degeneration of the nuclei of the medulla. Charcot and Huchard have laid great stress on the bulbar features of the disease, but in the cases above referred to, particularly those of Holberton, Lépine, and Boffard, in which the symptoms were most characteristic, the lesions were extrinsic. A remarkable case is reported by Neuburger and Edinger in which a neurasthenic man, aged 46 years, with obstinate constipation, had vertigo and fainting attacks when at stool. For nine days before his death the attacks were associated with deviation of the head and eyes and a pulse-rate of 18 to the minute. The necropsy showed absence of the right lobe of the cerebellum and a varix close to the accessorius nuclei, with beginning hæmorrhagic infarction. (2) Without recognisable lesions. Typical attacks of Stokes-Adams disease of maximum severity are on record in which the most careful examination has failed to find any changes in the heart, the arteries, or the nervous system. Dr. Edes's first case, that of a woman, aged 50 years, with a very neurotic history, hadfor seven or eight months recurring seizures, with loss of consciousness, &c., in which the pulse fell to 20 to the minute. She died in an attack; the necropsy made by Dr. Councilman was negative.

3. *Arterio-sclerotic group.*—In this group there are obvious changes in the circulatory system. So large is the majority of the cases in this division that Huchard's dictum is justified, "L'étiologie est cette de la sénilité artérielle.' My experience is confined almost entirely to this form. The histories, which I have condensed as much as possible, give a fair picture of the phases and forms of this remarkable condition.

CLINICAL HISTORIES.

All my cases, 12 in number, were in males; three of the patients were above 76 years of age and six were between 50 and 70 years of age. The youngest was 35 years old, the case following a streptococcus infection. Excluding this patient, my cases fall into three categories—a group of five cases with very severe and acute symptoms; a senile goup of four cases; and a milder form in younger men, two cases.

I.—SEVERE CASES.

The patients in the severe cases are men as a rule in the pre-senile stage and they present well-marked cardio-vascular lesions. In Case 2 there was extensive calcification of the coronary arteries and of the arch of the aorta, with

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Transactions of the Association of American Physicians, vol. iv.
[5] Philadelphia Medical Journal, 1901, and Transactions of the Association of American Physicians, 1901.
Ogle :THE LANCET, Jan. 30th, 1897, p. 296.
Deutsche Medicinische Wochenschrift ,1896.

hypertrophy of the heart and chronic myocarditis. Some of the attacks are very much like the syncope anginosa of the older writers. In Cases 3 and 4 the symptoms were those of myocardial disease associated with dilatation of the heart. Case 5, the most characteristic instance of the group, presented an extraordinary series of vascular and cerebral manifestations.

CASE 2. *Unusual sensations in the chest; constantly recurring vertigo with slow pulse; subsequent attacks with complete loss of consciousness; pulse-rate usually as low as 28, sometimes sinking to 20; arterio-sclerosis; duration of the symptoms for about six years; sudden death; calcification of the coronary arteries and the root of the aorta, with hypertrophy of the left ventricle.*—The case is given in full in my lectures on angina pectoris (1895). Of the subsequent history Dr. Houston of Troy, New York, writes (Jan. 5th, 1897): "Mr. V.'s pulse continues at about 30 to the minute with an occasional intermittence. He has now very marked vertigo, perhaps 25 attacks each day, each time nearly but not quite losing consciousness. He has no headache, sleeps well, and says he is very comfortable except for these sinking 'spells' and weakness." Jan. 25th, 1898: "Mr. V. has had some very bad attacks of late. In the present month he has twice fallen to the sidewalk while conversing with friends, loss of consciousness being absolute." March 13th, 1898: "The attacks of vertigo with unconsciousness have occurred at shorter intervals through January and February. After a period of complete rest he improved somewhat; pulse-rate 27 to the minute." On March 2nd, 1899, the patient died suddenly. On the 8th Dr. Houston wrote: "The day before Mr. V. died he had an attack of syncope on the street. In the evening he felt unusually well and played checkers with his son. He retired early and slept well. In the morning he got up and had his tub and went back to bed in order to have his breakfast. Just as the tray came he fell back suddenly and in a few moments died." The necropsy showed extensive calcification of the coronary arteries and the root of the aorta, with hypertrophy of the left ventricle. Dr. Houston states that at times his pulse fell to as low as 20 to the minute. The usual rate was 28.

This very remarkable case illustrates the long duration of the affection, fully six years, the syncopal type, without apoplectiform symptoms or convulsions, and the coronary artery changes.

CASE 3. *Attack of faintness, with a slow pulse; great cardiac weakness with pallor and syncopal attack; gradual recovery.*—The patient, a man, aged 57 years, was referred to me by Dr. J. Newton on April 4th, 1900. The patient had been a hard-working man who had lived carefully and indulged in no excesses. He had not used tobacco or alcohol. He had typhoid fever 20 years previously. His mother died from heart trouble at the age of 63 years. Two brothers had died from heart trouble at the ages of 30 and 40 years respectively. Two weeks before the onset of the present illness he had had a great deal of extra work and worry. On Nov. 14th, 1899, while walking to his place of business he felt faint, continued to walk, though slowly, and had no pain. He went to his office, walked upstairs, chatted with his foreman, then started home quietly. He felt very faint and excessively weak and was breathing very slowly. When he got home he was very pale, the left foot was particularly cold, and the left hand was very pale. He had no pain. His pulse became exceedingly slow—down to 38 and even lower. From this time on he was in bed for two months and had constantly recurring attacks of great pallor and coldness with cardiac weakness and slow pulse. During this time he had two fainting attacks in which he got very pale and cold. The heart's action gradually strengthened and he was able to get up and to move about the room. He had since then gradually improved but had had to be very careful and to go very slowly. When last seen he complained of a remarkable tickling and feeling of numbness in the left hand and the left foot, with a sensation of pulsation in the fingers. He was a very healthy-looking man, of good colour, with a good complexion. The eyes were clear; there was slight arcus in the left. The pulse was 72 and regular; the tension was plus; the artery was distinctly sclerotic. The apex beat was not visible; there was no increase in the area of transverse cardiac dulness. At the apex both sounds were clear, the first being loud and ringing. The aortic second sound was not accentuated. Altogether he seemed remarkably well and it was difficult to believe that he had had such a serious cardiac attack only a few months before.

CASE 4. *Attacks of transient vertigo with permanent slow pulse; syncopal attacks; cardiac dilatation with pulmonary œdema and enlargement of the liver; gradual improvement; persistence of the bradycardia; sudden death.*—The patient was a robust, healthy-looking man, aged 61 years, of good habits and good family history. Except mental overwork and the stress and strain associated there were none of the factors leading to arterio-sclerosis. When about 35 years of age he had severe burns of both hands and the face which had been long in healing. Many members of his family had a pulse-rate of about 60. In the spring of 1899 I saw him during convalescence from an attack of bronchitis and was then impressed with the feebleness of the heart sounds; the pulse was firm and full, not specially slow. In the autumn of the same year he had an attack of syncope and it was then noticed that the pulse was slow. He had had transient attacks of vertigo. Early in the spring of 1900 he had another very severe attack of syncope in Philadelphia with prolonged collapse and great feebleness of the heart. He recovered rapidly, but the pulse remained at about 45. In the summer he became very neurasthenic and was unable to stand slight noises and had a distressing feeling of tension in the head. In July, August, and September there were much flatulency and gastro-intestinal disturbance and on exertion a tendency to fainting. There were râles at the bases of the lungs, slight cough, swelling of the liver, and signs of cardiac insufficiency. It was impossible to feel the apex beat, the area of transverse dulness was increased, the sounds were muffled, and there was a soft apex bruit. He was under the care of Dr. Holford Walker of Toronto who had the advantage of the counsel of Dr. McPheadan and of Dr. Musser of Philadelphia. I also saw the patient early in August. Gradually the heart grew stronger but without any change in the pulse-rate, which was constantly between 30 and 40. On no occasion did I find a "coupled rhythm." From October, 1900, to January, 1902, he improved steadily and was able to take long walks and to get back much of his accustomed vigour. On the morning of Feb. 5th, while getting out of bed, he complained of not feeling very well, was nauseated, and fell back in the bed and died in a few moments.

CASE 5. *Fainting "spells" with slow pulse; slight arterio-sclerosis; epileptiform attacks; remarkable discrepancy between the heart and pulse rate; death in an attack.*—A man, aged 56 years, a farmer, was admitted to hospital on May 15th, 1900. He complained of dyspepsia and a nervous affection. There was nothing of any moment in his family history. When eight years old he had typhoid fever; as a youth he had malaria and at the age of 21 years he had measles. Twenty-five years before he had an attack of gravel. He had never had any venereal disease. His habits had been good. He used neither alcohol nor tobacco. He had been a large eater. Until five years previously he was a shoemaker. In August, 1899, he had two fainting "spells" which lasted for about 10 or 15 minutes. In November he began to notice that after exertion there was a feeling of suffocation. He had had to be extremely careful in his diet; if he took too much food or very indigestible articles he would feel sick at the stomach or everything would grow dim and misty before his eyes. He had had in all four severe fainting attacks, in each of which he was unconscious for at least ten minutes. He had had many of the mild, slight attacks. The patient was a fairly well-nourished, well-built man; the mucous membranes were a little anæmic. The tongue was coated. There were no tophi. Dorsal decubitus; there were no cyanosis and no arcus; the pupils were equal and normal. The pulse was of good volume, of about normal tension, regular, and 28 to the minute; the vessel wall was distinctly felt but was not greatly sclerosed. He had a moderate grade of funnel breast; the expansion of the chest was good; the lungs were clear throughout. The point of the maximum impulse of the heart was seen and felt in the fourth interspace, 10 centimetres from the middle line and one centimetre inside the nipple line; there was no thrill. Absolute dulness began on the fourth rib and extended from the right sternal border to the point of maximum impulse in the fourth interspace. There was a loud well-marked first sound, with which there was a soft systolic bruit, traceable as far out as the anterior axillary line. The second sound was well heard and clear. Between the clearly heard heart beats at times there was a faint systolic sound heard after the first and separated from the next by a long interval. This abortive character of the alternate heart beat, "coupled rhythm," was noted on

admission. At the aortic area the heart sounds were enfeebled and there was a soft systolic murmur. The second sound was not accentuated. The abdomen looked natural and there was no tenderness on palpation. The border of the liver was not palpable; the spleen was not enlarged. There was no œdema of the legs or feet.

The case is of sufficient interest to give in some detail. On May 16th the patient felt dizzy and at 4 P.M. he suddenly became very pale and fainted, remaining unconscious for ten minutes. The pulse fell to 15 beats per minute. Between 4 and 8 P.M. he had four such attacks, the pulse varying from 15 to 20 beats per minute. There was sweating with each attack. After the last attack of syncope he vomited. The night nurse reported that on several occasions the pulse was as low as 15 beats. He slept comfortably and in the morning seemed all right. On the 17th, at the visit at 9 A.M., I found the patient with a pulse-rate of 18. Both sounds had a very normal character at the apex. The systolic bruit had the greatest intensity over the body of the heart. The percussion note was clear over the manubrium. There was no tracheal tugging. On the 19th, while making the morning visit, I saw the patient in two attacks, each of which lasted about half a minute. The eyes were turned to the left and became fixed; the muscles of the face twitched and the hands twitched slightly. It resembled a slight epileptic attack. The pulse at the wrist was 12, and the cardiac impulses corresponding to them were strong and easily seen. In the jugular veins there were fluttering systolic impulses, quite well defined, but difficult to count, about 120 to the minute. On careful inspection of the cardiac area there were seen in the fourth and fifth interspaces small, regular, systolic impulses, exactly 100 to the minute. Corresponding to these there could be heard faint systolic sounds at the same rate. During the night the patient had a severe attack of vomiting and had frequent syncopal attacks. Dr. Futcher noted that while the pulse was beating at 12 to the minute auscultation over the heart gave 143 feeble beats to the minute, each eleventh or twelfth beat being forcible, with a loud first sound, and corresponding to the one recorded in the radials. On the 20th the patient had some hiccough and at midnight there was typical Cheyne-Stokes breathing. The heart could be seen beating at 95 to the minute but the pulse was only 19 at the wrist. On the 23rd the patient had been better for the past two or three days. The pulse had been at about 20 at the wrist and 84 at the heart. On the 26th the patient had a persistent pallor, the pupils were small and equal, the pulse was full, 19 to the minute at the wrist and 74 at the heart. On the 29th at the time of the visit the pulse was 28 at the wrist. The small beats were not visible at the point of maximum cardiac impulse, where the impulses corresponded with the pulse at the wrist; but on auscultation, between each of the forcible impulses, which shook the whole front of the chest, there was heard a little, soft, faint, systolic sound, which was not expressed at the apex as a visible or palpable impulse and was not felt at the wrist. The pulsations in the jugular vein were very evident but it could not be determined if they corresponded with the total number of ventricular beats. At 9.30 P.M. the patient had an attack of unconsciousness, lasting for several minutes, followed by dyspnœa and afterwards Cheyne-Stokes breathing. On the 30th, at 10 A.M., while the nurse was giving him his bed bath, he suddenly vomited and immediately afterwards became perfectly rigid and unconscious and cyanosed. Before chloroform could be administered the muscles relaxed, the patient gave a few gasps at rather long intervals, and died without regaining consciousness. During the attack the pulse was not perceptible at the wrist. Artificial respiration was performed without any result. There was a slight trace of albumin in the urine and there were a few hyaline and granular casts. There was no necropsy.

Another case of the same intensity may be referred to. On May 13th, 1902, I saw in the afternoon at the Union Protestant Infirmary, with Dr. Hamburger, a man, aged 45 years, who illustrated all the symptoms of this severe type of the Stokes-Adams disease. He was a stout, healthy-looking man. He shook hands with me as I went in and then gradually closed his eyes in an apathetic way. The face and general surface of the skin were a little suffused. The pulse was 20 to the minute; there was slight sclerosis of the artery; no special increase in tension was present. There were no intermediate beats of the radial pulse. He was rather stout and the apex beat was not easily visible, but the slow heart beats were palpable. Intermediate small beats could neither be seen nor felt. There was considerable increase in the transverse area of cardiac flatness. In the neck the visible pulsation of the carotids was very plain, 20 to the minute, and there were in the suprasternal notch and just along the sterno-cleido muscles on either side two or three tremulous venous impulses not so localised as in Cases 5 and 8. On auscultation at the apex both the first and second sounds were well heard. With the first there was a rather rough grating murmur. There were no intermediate sounds audible. Above the fourth costal cartilage, particularly at the mid-sternum, Dr. Hamburger had noted between the sounds one or two distant soft sounds. These I, too, could catch with distinctness. Just after we had finished the examination the patient had in succession two epileptiform paroxysms. The onset could be told at once as the beat of the heart stopped and could not be felt at the wrist or heard at the heart for from 15 to 20 and even as long as 35 seconds. The convulsion was a general tremulousness, the face became slightly cyanotic, the breath was held, the eyes twitched and rolled up, and the muscles of the face twitched. The paroxysms lasted for a few seconds only. He came out of them very quickly and the face flushed with great rapidity and he seemed a little dazed. He had as many as 150 of such attacks in the 24 hours. The patient recovered from the serious condition and returned to his home in Washington. A few months later he died during an attack.

CASE 6. *Typical pseudo-apoplectic attacks as described by Stokes; slow pulse; in some attacks convulsions; duration more than ten years.*—While attending the meeting of the British Medical Association at Montreal in 1897 I saw at a club an old friend, a man aged about 60 years, who consulted me casually about distressing attacks of transient vertigo and others of a more severe nature in which he lost consciousness completely. During the attacks he said that his pulse sank to 20 or even lower. He said to me, "It is not at all improbable that I may have an attack at some time while you are in the club and I will tell the head waiter to notify you." On the following day, while I was at dinner, I was called hurriedly to the secretary's office and there I found my friend in an attack. He had fallen unconscious, was breathing very heavily, almost stertorously, and had a very flushed face. I thought at once that it was really an apoplectic stroke. The pulse was very slow and very full, about 20 to the minute. The arteries were very sclerotic. The heart's action was very forcible and I could not hear with the unaided ear any intermediate beats or a murmur. So alarming did I think his condition that I said to his servant that I thought it might be well to send for his physician and to have him bled, to which he replied that this was an ordinary attack, that he had seen him in many quite as severe, and that he rallied in the course of from 15 or 30 minutes. I returned in the course of half an hour and found him up, feeling a little dazed and heavy in the head but quite himself. In some of the attacks he stated that he had convulsions. On March 3rd of the following year he wrote to me that he had been very much better. He stated that he had had the fainting attacks and the slow pulse for many years. Dr. James Stewart, under whose care he was, told me that his attacks were often of the character of *petit mal* and were sometimes followed by motor aphasia, mainly of nouns. The lowest pulse-rate he had ever found was 21, but it had been as low as 18. He died during an attack. The trouble had lasted for fully ten years. He had painful neuromata along the extensor surface of the left arm and two in the left shoulder. There was a curious relationship between the pain in these and the pulse-rate, the slower the pulse the greater was the pain.

II.—SENILE CASES.

Not infrequently old men have bradycardia with attacks of vertigo, or of fainting, or even of prolonged unconsciousness. There may be in addition cardiac dyspnœa and Cheyne-Stokes breathing. The cases are more common, I think, than any description in the literature would lead one to suppose, particularly the incomplete forms, *forme fruste*, with vertigo.

CASE 7. *Slow pulse; syncopal attacks; arthritis; possibly gout; pericarditis; death.*—On Jan. 16th, 1899, I saw, with Dr. Hemmeter, a man, aged 72 years. The patient, a very healthy man, had lived an active life, had been a hearty eater, and had been "fond of a glass of beer." He had had several attacks of arthritis, once or twice in his big toe. During the past three years it had been noticed that his

pulse was very slow, down to between 50 and 60, and he had had three syncopal attacks with pronounced bradycardia. In one of these he had fallen when at stool and was unconscious for some time, with a pulse-rate of 40. When I saw him he had slight arthritis, a temperature of 102°F., and a pulse of 56, full in volume. The vessel wall was moderately sclerosed. The heart sounds were clear. On the 18th I saw him again. There was then a very loud to-and-fro pericardial friction murmur. His temperature had kept up, the joint symptoms were better, but he evidently was not so well. The amount of urine was reduced and there were much albumin and numerous tube casts. He became very much worse during the next three days, became unconscious, Cheyne-Stokes breathing developed, and he died on the morning of the 23rd. There was no post-mortem examination.

CASE 8. *Attacks of vertigo and sudden weakness; slow pulse; arterio-sclerosis; sudden death.*—The patient, a medical man, aged 70 years, was seen in March, 1900. He had always been a strong, robust man; he had never been ill in his life and was unusually well and strong. He had been a devoted cigarette smoker for 40 years. He was not a heavy eater; he had drunk during and after the War of Secession, but had been a temperate man for 26 years. He had been gouty at times. He was a fairly hard worker. On Feb. 10th he had a "giddy spell" and felt a sudden weakness as though he wanted to lie down but did not faint. The attack lasted for an hour or two. He then noticed that his pulse was slow, only 40 to the minute. It did not reach the normal for a couple of days. His usual pulse had been from 72 to 76. He did not feel well for two days. Ten days later he had a second attack, felt giddy, and noticed that his pulse was again low and that it had remained low, once being as low as 38, occasionally rising to 42 and 44. When quiet he was comfortable. He staggered a little when he first got up. He had no pain about the heart. The pulse in the sitting posture after rest and quiet was just 40. There was moderate sclerosis of the arteries. The apex beat was diffusely visible just below the nipple. The beats at the apex were 40; there were no abortive heart systoles. There was an apex systolic bruit, not obliterating the first sound, and very faint at the aortic cartilage. No flatness was manifest over the manubrium. In the right side of the neck there was a pulse in the jugular vein which occurred in groups of three and which was twice as rapid as the pulse in the temporals. It looked just like a wavy venous impulse and was 80 to the minute. I saw this patient again twice. He improved for a time after the use of tobacco and the pulse-rate rose to 55. He had a return of the "sinking" attacks with bradycardia and I heard of his death during an attack in April, 1903.

It is remarkable for how long bradycardia and occasional fainting attacks may persist. In the following case the patient claimed that he had had a slow pulse for 30 years and that he had occasional fainting attacks.

CASE 9. *Stokes-Adams disease; slow pulse; syncope with anginal attack; cardiac dyspnœa; sudden death.*—The patient, a man, aged 74 years, was seen with Dr. McDowell, complaining of debility, with attacks of shortness of breath and palpitation. He was a thin, spare man and had a good colour. He had had a very healthy life without serious illnesses. Thirty years previously his son, at the time aged 12 years, had a fall and after feeling his pulse he felt his own and found that it was only 50 per minute. He consulted a physician who told him that his condition was serious and that he would not live long. He thought that he had had a slow pulse ever since until lately. For many years he had been subject to fainting attacks on extra exertion or on great mental emotion, but they had never been serious. Five years before, on the occasion of the fiftieth anniversary of his ordination, he preached and at the conclusion of his sermon he was seized with a pain of terrible severity in the region of the heart and down both arms. In the arms the pain was as intense and severe as the sharpest toothache. He was very faint and lost consciousness. He broke out into a profuse perspiration and the attack ended with severe diarrhœa. His pulse was very slow and three days after the attack the medical attendant said that his pulse was 38. Since that date he had not had any fainting attacks. He had felt on many occasions palpitation of the heart and irregularity and he had known that his pulse had been below 50. At night he would sometimes be aroused if he lay on the left side by a sudden sharp pain, as if something had grasped the heart. Within the past year he had had certain additional features. His pulse had become more rapid, having risen to above 70. He had had attacks of cardiac dyspnœa at night and was short of breath on exertion. That summer at the seaside he preached, took a great deal of interest in church matters, and seemed to have overtaxed his strength. He had become much more feeble. The patient's colour was good; there was no swelling of the feet; no arcus was present. The radials were stiff, with no calcification. The apex beat was in and just outside the nipple line. There was a soft systolic murmur and along the left sternal border a soft aortic diastolic which was heard only when the breath was held. Both sounds were rather feeble at the base. There was no tenderness over the course of the aorta. I saw this patient again with Dr. McDowell on Oct. 13th. He had been steadily failing, had much less ability to get about, and much shortness of breath, particularly at night. For the past two or three days he had been worse. On the previous evening he had an unusually severe attack of cardiac asthma and one attack was associated with a good deal of pain. I saw him at 5.30 in the afternoon. He was resting quietly, breathing easily. His pulse was 90 and of very good volume; there was no special tension. There was a soft apex systolic murmur, as at the previous examination. There was no gallop rhythm. His cardio-vascular condition seeemed to be extremely good. He was rather dull, did not care to be disturbed, and complained of feelings of great prostration and weakness. At 12.30 that night he died suddenly without awaking.

CASE 10. *Attacks of dyspnœa; slow pulse; two attacks of loss of consciousness; arterio-sclerosis; hypertrophy of the left ventricle.*—The patient, a man, aged 65 years, was seen on Dec. 14th, 1896, complaining of dyspnœa. On entering the room the patient was so short of breath that he had to wait several minutes before he could say what was the matter. With the exception of dyspepsia, which he had had at intervals for 30 years, he had been a very healthy man. He had not used alcohol or tobacco and had not had syphilis. He had diphtheria 10 or 12 years ago; he had not had rheumatic fever or gout. He had worked hard and recently had had business troubles and cares. For the past two or three months he had been getting short of breath, particularly on exertion. After proceeding thus far in the history I felt his pulse and was surprised to find it very slow, 40 to the minute. I then asked him about attacks of unconsciousness, of which he had made no mention, and got a history of two very remarkable seizures. In June, 1895, in Washington, after a couple of days of anxious business, he returned home one Thursday and just after dinner had a fainting attack. He did not know how long it lasted, but his family was very much alarmed and sent for a medical man; he was "off his head" for some time after the attack and talked foolishly. He was not able to leave the house for ten days. He had a second attack in June of this year. He was in the Custom House attending to some business and fell in a faint. He was unconscious for more than 20 minutes and was a good deal dazed after coming to. These were the only two attacks he had had. The patient was a tall man, with iron-grey hair and beard; he talked very deliberately and the expression was rather heavy and dull. The pulse when first counted was 40 per minute, full, slow, incompressible, anastomotic; the vessel wall was considerably thickened. The apex beat was in the fifth interspace, just below and a little outside the nipple line, forcible and punctuate. There was no thrill. On palpation in the second right interspace and second right costal cartilage there was to be felt an unusually loud diastolic snap. The limit of cardiac flatness was on the fourth rib at the right sternal margin and just at the apex beat. At the apex both sounds were heard, with the first a soft systolic murmur. At the second right interspace there were a soft systolic murmur and a second sound of most unusual and remarkable intensity, without any amphoric quality, sometimes single, sometimes distinctly reduplicated. I saw the patient again on June 27th, 1897. He had been much better; the slow pulse was not permanent but at times it was as low as 40.

III.—CASES OF SLOW PULSE WITH OCCASIONAL SYNCOPAL ATTACKS IN YOUNGER, HEALTHY MEN.

CASE 11. *Attacks of fainting for five years; slow pulse; arterio-sclerosis; good general health; appendicitis; operation; recovery.*—The patient, a man, aged 40 years, was seen on May 20th, 1901, complaining of attacks in which he fell and lost consciousness. The patient had been a healthy man, very active and vigorous. For many years he had had at times rheumatic pains. After an attack of pneumonia, nearly

20 years previously, it was noticed that he had a slow pulse but he did not remember the rate. Five years previously he had his first attack of fainting. He felt nauseated and faint but recovered very rapidly. A few days later he had a second attack. During that summer he had a good many and he had had them at intervals ever since. He felt in them as though everything stopped and then he would fall. There were no movements in the attacks. He was more apt to have them when he was tired or after any special excitement. In the attacks he either fainted away completely or could with difficulty keep himself from fainting by rubbing his wrists violently. The attacks rarely lasted more than a few minutes. They were accompanied with a very slow pulse, usually below 40. His apex beat was in the normal position. The heart was not enlarged. The sounds were clear; the aortic second sound was a little accentuated. The radials were distinctly sclerotic. The patient was admitted to the hospital, where he had an attack of appendicitis, of which he had had several previously, and he was transferred to the surgical side for operation. Very many observations were made upon his pulse and heart. The rate was usually about 50. The day before operation it was 40; just after the operation it rose to 90. There was no "coupled rhythm." He remained in hospital until June 3rd, during which time the range of the pulse was from 40 to 65. There was no hemi-systole. On the occasion of his visit to me I made him go out and walk briskly and when he came in his pulse rose to 110. He was a very healthy, active man and said that he had got accustomed to the fainting attacks.

CASE 12. *Syphilis at 23 years of age; for six months recurring attacks of vertigo; two attacks of great severity; permanent slow pulse of 28.*—A robust, healthy-looking man, aged 45 years, of good habits, had consulted me on March 15th, 1902, for what he called fainting attacks. They had begun six months before without any cause and were of two varieties. The first was a slight transient giddiness, in which he felt for a moment or two as though he was about to fall, but quickly gained control without struggling or swaying; while he was undressing he flushed a moment and said, "There, that was a slight one." He had many of these, often three or four in the day, and any excitement was apt to induce them. Twice he had had more serious attacks in which he had become faint and had had to hold on to something to keep from falling. He did not lose consciousness but felt a sensation of "utter goneness," as he expressed it, and then broke out into a profuse sweat. There were a sensation, too, of stuffiness in the throat and a wheezing in the tubes. Shortly after his attack of faintness the medical attendant noticed that his pulse was very slow and it had ranged ever since from 28 to 36. He had no abnormal sensations about the heart itself. He felt well and was vigorous but he had become much alarmed since the onset of the severer attacks. He had been a vigorous, muscular man; he had used much tobacco and alcohol in moderation. He had had syphilis but was thoroughly treated for two years. I dictated the following note:—"Healthy-looking man of good colour. Pulse 28, full; radials easily felt. Visible pulsation in cardials and a fluttering venous impulse difficult to count but about double the rate in the carotid. Apex beat not visible, not palpable; no increase in the area of transverse cardiac flatness. Both sounds audible at apex; no intervening beat to be heard at apex or base. Aortic second sound accentuated. After exertion the pulse-rate rose to 40. Examination of the other organs was negative."

I have permission to quote the following remarkable description of his own case by an army officer who has consulted me by letter on several occasions and who has been under the care of Dr. R. H. Babcock and Dr. E. F. Wells of Chicago:—

January 5th, 1903.

DEAR SIR,—While in Germany in the summer and fall of 1900, taking a course of the Schott bath treatment for an affection of the heart, I took advantage of the opportunity thus afforded to consult several German professors regarding my malady. Among these men was Professor Dr. Rosenbach of Berlin, an author of a work on "Diseases of the Circulatory System." The professor, although he spent an hour or more in his examination of me, was very non-committal in his diagnosis as well as prognosis, but stated that he hoped the condition then found would readjust itself and that improvement of my health would result, in which case I was to inform him of the fact, as he would distinctly remember my very unusual case. He stated further that upon my return to this country I should make it a point to consult you. This I was unable to do owing to the illness of the person accompanying me to Europe upon our landing in New York. Since my return to the West I have been continuously under the care of physicians, and until July last under the observation and care of Dr. Robert H. Babcock of Chicago. It was he who in April of 1902 first informed me that mine was a case of Stokes-Adams disease, although the original examination of me by Dr. Babcock had been made as early as October, 1899. Dr. Babcock informed me that he was inclined to make that diagnosis after mature consideration of an article by Dr. August Hoffmann appearing in the *Zeitschrift für Klinische Medicin*, vol. xli., 1900. Later the article on the Stokes-Adams Disease by Professor A. Jaquet in *Deutsches Archiv für Klinische Medicin*, Band ii., confirmed him in his opinion.

Personally I have made a thorough study of my case, so far as a layman can, and although I have had most divergent views and diagnoses given me by the various physicians who have examined and treated me I am convinced that mine is a case of Stokes-Adams disease. If there are any methods of treatment or medication that you may have used, with even moderate success, in your practice I should very much like to try them; in that case I would be able to give you a complete medical history of my case which might enable you to treat me at a distance. I would only say at this point that, so far as I know, no specific cause can be pointed out as having produced the condition which now exists. I am now 29 years of age. At 18 I was supposed to be entirely well, for I was examined at that time for life insurance and reported entirely sound by the examining physician, who yet maintains that that report was correct, and the policy was issued. At 20 I was first informed by a physician whom I consulted as to whether or not I was physically sound, contemplating as I did entering the Military Academy at West Point, that I had heart disease. I was nevertheless admitted to the Military Academy and graduated and commissioned an officer in the army four years later. One year after entrance "aortic stenosis" was diagnosed and subsequently at each annual physical examination. At these examinations such additional notes as "heart slow and strong" were also reported. When I was graduated (or at any rate not more than six months afterwards) my radial pulse was 38, regular, and I was apparently well. It is now, and has been since May, 1899, 26 per minute. Frequently stops entirely, causing me great distress and lack of consciousness. At times there is delirium cordis. Occasionally the pulse goes up to from 40 to 70 or 75 and has been at practically a normal rate for a period of three weeks on one occasion. I cannot say whether the heart took up this higher rhythm of its own accord or not since I have been using medicine almost continuously.

I am now compelled to use morphine in moderately large quantities, it being the only thing that seems to steady my heart; eserine, too, has been tried; this just before and during the time my pulse was at a normal rate for three weeks. The use of oxygen, too, seemed to cause my pulse to become normal but would not sustain it for more than one week. The sphygmographic tracing which I send is a fair indication of the usual heart activity. My skin is almost bloodless and I suffer from the cold very severely. Since February, 1902, I have been confined to my bed almost continuously and since July absolutely. This has reduced my weight, which ordinarily is 140 pounds, to about 115 pounds. But I have begun to gain since I resumed a general diet about four weeks ago. There is a marked systolic murmur over the aortic and mitral areas, both of which murmurs become very much less marked when heart action is normal. Careful auscultation reveals usually two small heart contractions of which the latter produces the radial pulse. The former (imperfect) contraction does not produce a pulse-wave either in carotids or in the radials. None of the ordinary secondary symptoms of valvular disease of the heart, such as dyspnœa, dropsy, cyanosis, &c., are present in my case.

Yours, &c.,

DIAGNOSIS.

The severe form (Cases 2 to 6) presents such a clear-cut picture that there is no question as to the nature of the trouble. The senile form is also very well marked. The post-febrile variety may be very severe but is as a rule more hopeful and recovery, as in Schuster's case, may follow after weeks of recurring attacks. The toxic forms of bradycardia are sometimes associated with vertigo but rarely with syncope or epilepsy. The tobacco bradycardia may sometimes cause alarm. I was consulted on Jan. 7th, 1896, by a man, aged 48 years, who said that he had had heart disease for three years. He had smoked and chewed from boyhood. He complained of irregular action of the heart and a dull aching sensation in the chest. He then had attacks in which the pulse fell to 45 and 48 and which caused him great alarm. He had transient vertigo but never syncope. There were no signs of heart disease. I urged him to stop using tobacco. Within three months the pulse-rate was above 70. I have seen him on several occasions since and he has remained quite well.

A difficulty may arise in the diagnosis of the neurotic cases in women or in young men. Dr. Edes's first case illustrates the intensity of this form. Aggravated neurasthenia may be associated with bradycardia and vertigo of which the following cases may be given in illustration.

CASE 13.—A man, aged 44 years, was admitted to the Johns Hopkins Hospital on Oct. 29th, 1899, complaining of pain in the epigastrium, headache, and of swaying of the body while walking. He had used tobacco to excess, both smoking and chewing. He used whisky in moderation. He had not had syphilis but had had gonorrhœa twice. He had led a very active athletic life. Six months before, after severe exertion and exposure, he had "queer sensations" rising upwards from the thighs which made the act of breathing uncomfortable. He worried over this and had in a short time so much difficulty in getting his breath that he thought he had heart disease and was going to die. He stopped work for a few days but on resuming he had a similar attack and complained of a peculiar swaying of the body which he could

not control. The patient worked for two months but the swaying and pains in the crown of the head and in the epigastrium compelled him to give up. He was very nervous and complained of flushing, cold feet, and insomnia. He had lived a life of continued "high tension." He was a stout well-built man; the lips and the mucous membranes were of good colour; the tongue was slightly furred. The pupils were equal but not contracted; they reacted well to light and to accommodation. Well-marked dermatographia was present. There was some swaying on standing with the eyes closed and feet together. The pulse was of good volume and normal tension, regular in force and rhythm, but slow, being only 48 to the minute. The point of maximum cardiac impulse was neither visible nor palpable. The sounds were best heard in the fourth interspace nine and a half centimetres from the midsternal line; there was no thrill. The sounds were rather enfeebled at the apex and were clear and of normal relative intensity; they were clear at the base; the aortic second sound was slightly accentuated. There were no abortive beats. According to the patient's wife he had since childhood had an aversion to water, the drinking of which would cause him to have a "smothering" feeling at night. At the onset of the present illness the patient went to bed feeling perfectly well; he woke up in the night with a feeling of being smothered, so he got up and went outside. He was convinced that he was dying and after a time could not walk and had to crawl home. He was put to bed; he went to work as usual on the next morning. One week later he had a similar attack. His wife said that since the onset of the attacks he would often get up at night wringing his hands and in great fear of death, but was usually easily quieted. The patient improved very much after a short stay in the hospital, but the pulse was still slow, from 48 to 50 to the minute.

The following is a still more remarkable instance of a very slow pulse with nervous attacks.

CASE 14.—The patient, a man, aged 26 years, was seen on Oct. 4th, 1901, with Dr. Harry Thomas, complaining of attacks of nervous weakness. The family history was excellent. He had good health as a young man. In January, 1895, he fell about 20 feet, hit his back, and was severely hurt in the muscles of the back; no paralysis followed. He was in bed for three months on account of the pain in the back. This was his first year at college. He got well, except that on cold damp days his back would hurt him. His present trouble came on before the fall, in March, 1894. He had been feeling well and went to bed. In the morning he found that he was "giddy-headed" and everything in the room seemed to be turning round; he felt a little sick at the stomach. When he tried to get up he was very unsteady and if he moved suddenly he would fall; he had to support himself by chairs, &c. There were no ringing in the ears and no vomiting. He became nervous and went back to bed. The pulse sank to 42; there was no fever. The bowels were in good condition; he had a good appetite. He was in bed for three or four weeks. Every time he tried to stand he found that he was so weak that he had to go back to bed. If he did get up his heart would go from 80 to 100. He could not read on account of the headache that it caused. When he did get about he was weak. He improved slowly and in about six weeks was as usual. At the time of the first attack he was working on a farm and had had nothing to disturb him. The second attack was in about a year after his fall. Since then these attacks had recurred from eight to 12 months apart. They had all been of the same character, except that some were longer and some shorter. The shortest one was about three and a half weeks. This was in 1898. The last attack began on the afternoon of August 5th. He was taken with giddiness and vomiting and had to be taken home in a carriage and practically carried upstairs and put to bed. It was not weakness at first that kept him from walking but giddiness. The temperature was normal and remained so. On the next afternoon the pulse was 48. He was in bed for eight days and then went to his home 28 miles away with assistance. He had stayed more or less in bed every day since. The giddiness had lasted much longer in this attack than usual. He tried to go back to work a week before but was not strong enough. In going about the pulse increased to 93. The pulse when he was well was about 74 when he was going about. He was a healthy, robust-looking fellow. The pupils were active. The radials were soft; there was no sclerosis. The pulse was 80. The chest was rather long and narrow. The apex beat was in the fifth interspace, well inside the nipple line. There was no increase in the area of cardiac flatness. The aorta was not palpable in the sternal notch. The sounds at the apex and base were loud and clear; the aortic second sound was a little accentuated. In the carotids and the subclavians there was visible, possibly neurotic, throbbing. Marked and quick vaso-motor reaction was present.

These are certainly very remarkable attacks. They rather suggest a form of migraine and yet the ears were normal and the patient had no ringing in the ears with the attacks. He gave one the impression of being an excessively nervous man. On a course of hydrotherapy he had improved very much. Dehio suggests the use of atropia to determine in a given case whether the bradycardia is due to changes in the heart itself or in the nervous centres. In normal persons small doses paralyse the peripheral ends of the vagi and the action of the heart is hastened. In old people, in chronic myocarditis, and in one case of Stokes-Adams disease Dehio states that this effect did not follow. I do not know if this observation has been confirmed.

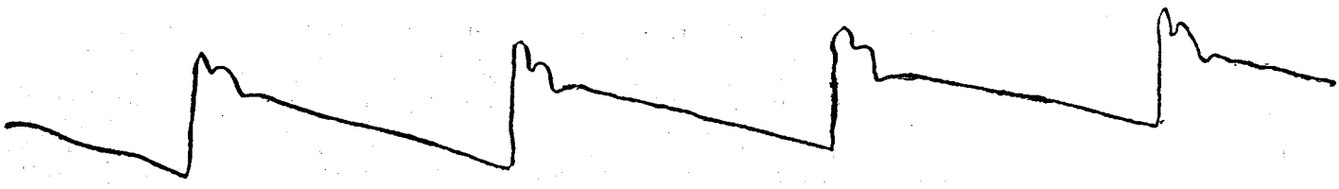
CARDIO-VASCULAR FEATURES.

All of the patients except Case 1 with an acute post-febrile attack presented arterio-sclerosis; in Cases 5, 11, and 12 it was of moderate grade. In no case was the heart greatly enlarged. In Cases 2, 4, 5, and 10 there was a soft mitral systolic murmur; in Case 9 an aortic diastolic murmur was present. In Cases 4 and 5 the sounds were weak and muffled. In the only necropsy of the series (Case 2) the arteries were found to be sclerotic, the root of the aorta and the coronary arteries were calcified, and the left ventricle was hypertrophied. The condition is not associated with the ordinary forms of valvular lesions, as in a majority of the cases the valves are normal, the heart is not enlarged, and the sounds are clear. In Dr. Edes's series collected from the literature in 31 of the 35 cases in which a necropsy was held there was a definite statement as to the heart and arteries and in 26 there were sclerotic and myocardial changes. Certain special features may be considered.

True bradycardia.—One of the first lessons a student has to learn in the wards is that infrequent slow pulse and bradycardia are not the same and that with an infrequent pulse there may be a normal or an increased number of heart beats. In lesions of the mitral valve, in chronic myocarditis, and as a result of the action of digitalis, the radial pulse may be 50 or even 40 and the heart beats exactly double, the intervening small beat (often visible at the apex in thin-chested persons and audible as a faint systolic sound) not reaching the finger at the wrist. True bradycardia with heart beats and pulse beats of equal number occurred in most of the cases, only in Cases 5, 8, and 12 did it alternate with false bradycardia. In eight cases the bradycardia was a permanent condition; in one case the beats fell to 12 per minute; in Case 9 the slow pulse had been observed for 30 years, in Case 2 for six years, and in Case 6 for at least ten years. In Cases 1 and 2 the slow pulse was not permanent but only occurred during the attacks. In Dr. Hamburger's case with a heart-rate at 20 prior to a convulsive attack I counted intervals of 20, 30, and once 35 seconds in which there was no heart beat to be seen, heard, or felt. The pulse in true bradycardia is usually full, strong, and regular. This last feature is marked and is apt to deceive the inexperienced as it may be present with intervening abortive systoles—the "coupled rhythm." There may be a very strong pulse with an indistinct cardiac impulse and feeble sounds, or just the opposite. The infrequency in true bradycardia seems to be due to prolongation of the diastole; the systole as a rule is sharp and quick. The tracing which is here presented shows this very clearly. By far the best sphygmogram I have seen is the one here given which was taken by Dr. Wells of Chicago. It is a normal tracing, except in the extraordinary prolongation of the line of descent, the period of diastole.

False bradycardia.—Stokes noted in his patient that the pulse-rate and the heart-rate were not the same and that there were semi-beats of the heart. In three of my series these abortive beats were present. They may not be evident at the radials or in the carotids where the pulsations may be in orderly sequence and of exactly equal size—so that at the wrist there is no indication whatever of arrhythmia; even the sphygmographic tracing may not show the interpolated beats. They may sometimes be seen and felt at the apex as in Case 5; more often they may be heard with distinctness. The number of abortive systoles varies; usually they are alternate and in orderly sequence, so that with a pulse of 40 at the

wrist 80 heart beats may be seen and 80 first sounds may be heard—the "coupled rhythm." In the severe attacks with very infrequent pulse there may be a number of abortive beats. In Case 5 in which this feature was studied with great care there were remarkable variations. On one occasion, with a pulse-rate at the wrist of 12, the heart beats were 100; on another they were 143. On May 26th the pulse was 19 to the minute at the wrist, with 74 palpable, visible, and audible cardiac beats. On the 29th the pulse-rate and heart-rate were the same (28), no intervening beats could be seen, but on auscultation there were feeble, only just audible, systolic sounds intervening between the forcible thumping pulsations which shook the whole chest.



Pulse-tracing of the patient whose description of his own case is given above. Pulse-rate at 40.

The question has been raised whether in all cases there are not these abortive beats, too feeble to be seen, felt, or heard. Case 5 was a very favourable one in which to study this problem. All the abortive systoles were usually visible on the thin chest wall and palpable. One day with the pulse at 28 and the same apparently at the heart and the wrist and without any visible or palpable abortive systoles such as were usually present, on auscultation one could hear distinctly soft systolic sounds intervening between the loud booming shocks. In other cases I have listened with the greatest care but in vain for these abortive sounds.

The "heart-block," or independent auricular systole, without corresponding ventricular contractions.—Stokes noted on the readmission of his patient a new symptom—a remarkable pulsation in the right jugular vein, more than double the rate of the ventricular contractions. This feature has been studied by Chauveau, by Quincke, by His, jun., and others, who are of opinion that the jugular pulsations correspond to independent auricular contractions which are not propagated to the ventricles—a state of "heart-block," as Gaskell terms it. In Case 5 the jugular pulsations never corresponded in number with the carotid pulsations or with the total number of ventricular beats. Cardiac, radial, and jugular tracings were taken simultaneously, from which, unfortunately, nothing very definite could be determined.

Jacquet has recently called in question the existence of these independent auricular contractions,[8] the existence of which his tracings do not support. It is quite possible that there are ventricular contractions too feeble to be heard and too feeble to open the sigmoid valves, yet which might in a dilated heart cause a pulse-wave in the right jugular vein. In Case 5 as a rule the interpolated beats were palpable, visible, and audible, but they continued to be *heard* after they had ceased to be felt or seen, and still feebler contractions might cause no sound and yet transmit a wave to the veins through an insufficient tricuspid valve. We cannot dismiss the question of independent auricular contractions as settled. In Dr. Hamburger's case slight but definite jugular pulsations were visible in the prolonged periods of systole with complete silence over the cardiac area.

Cardiac arrest.—This phenomenon is one of the most remarkable witnessed at the bedside. In the case just referred to the apparent stoppage always preceded the slight convulsive attack. The eyes were set, the face was flushed at first, then pale, and the patient appeared to be in the article of death. In such circumstances to wait 35 seconds, watch in hand, without a heart beat seems like the final "count-out"; but this man, after scores of such attacks, recovered sufficiently to go to his home. A precisely similar condition is met with in some cases of angina pectoris. It always suggests to me the heart state of fibrillary contraction produced experimentally by the Kronecker puncture.

Vaso-motor changes.—As in angina pectoris the vaso-motor system is profoundly involved in the severe attacks, as shown by the pallor, the sweating, and the vomiting, and in some cases by the marked paræsthesia, the numbness, and the tingling.

[8] Deutsches Archiv für Klinische Medicin, Band lxxii.

NERVOUS FEATURES.

It is for these the patient seeks relief, naturally much alarmed. Four forms of attack occur:—

Vertigo.—While walking in the streets or engaged in conversation the patient feels giddy and staggers or may fall unless he catches hold of something. As in Case 2, the attack may be of great severity and very distressing, incapacitating the patient. The attacks may precede for years the onset of severer forms. In very old men with a permanent slow pulse this may be the only manifestation. It is a well-recognised and common symptom of arterio-sclerosis.

Syncope.—Much more alarming are the fainting attacks with complete loss of consciousness. Without warning the patient becomes pale and may fall instantly in a deep faint, with feeble, imperceptible pulse and the general features of cardio-vascular collapse. The respirations are shallow, a clammy sweat breaks out, and in severe attacks death may seem imminent; indeed, the patient may pass away without recovering consciousness. As a rule the attack does not last more than a few minutes. Many attacks may occur in a day and, as in Case 2, they may alternate with vertigo. They may resemble in character *petit mal*. Syncope is the most frequent of the severe nervous symptoms and occurred in eight of my cases.

Pseudo-apoplexy.—The patient falls in a deep coma, with loud stertorous breathing, deeply congested face, and all the features of an apoplectic stroke. Both Adams and Stokes describe the condition most accurately and the latter comments on the remarkable circumstance that there is no consecutive paralysis, as after an ordinary stroke. There are rarely convulsive movements and in four or five minutes consciousness is restored and the patient may get up and go about his work (Case 6). An extraordinary feature is the frequency of the attacks as noted by Adams and Stokes. In Case 6 they had recurred for many years and had been associated with transient attacks of motor aphasia, such as are met with not infrequently in advanced arterio-sclerosis.

Epileptic seizures.—As mentioned, the transient vertigo and syncope may resemble *petit mal*. The convulsive attacks are either slight spasms of the muscles of the face and hands with the loss of consciousness, as in Case 5, or more rarely they may be more general, resembling true epilepsy. In Case 5 and in Dr. Hamburger's case the convulsive attacks were very frequent. In the latter we could tell at once when the attack was coming on. The pulse would stop for 20 or more seconds, the face flushed, the breath was held, the eyes rolled up, and twitching of the face and hands began. Sometimes the attacks may not last for more than a few seconds and the patient may go on with a conversation; even a 100 or more of such attacks may occur in a day.

PULMONARY FEATURES.

The cases present the respiratory symptoms so often met with in chronic myocarditis. Attacks of cardiac asthma are common, occurring chiefly at night (Case 9). These may be the features of angina sine dolore, the distress, the pallor, the sweating, the shortness of breath, or the state called by Goodhart acute emphysema, with universal wheezing, the *Lungen-starheit* and *Lungenschwellung* of von Basch—which, I take it, is nothing more than acute œdema of the lungs. Cheyne-Stokes breathing is more common and was met with in four cases of my series. In the pseudo-apoplectic attacks the stertor, with deep laboured respiration and expiratory puffing of the cheeks, may have all the intensity of the genuine stroke. Prior to the attacks of epilepsy there may be transient arrest of respiration with flushing of the face.

PATHOLOGY.

It is by no means easy to discuss intelligently the pathology of these remarkable attacks. We have two great groups of bradycardia—the one associated with lesions in the heart itself, the other with disturbances in the nervous system,

organic (as in many well-known cases) or functional, without any obvious changes. It is not the infrequent pulse, transient or permanent, which is so difficult to understand but the phenomena of the attacks of Stokes-Adams disease. What has happened so to disturb the rhythm of the heart that to one perfect there are four or five abortive systoles, or that the rate is reduced to 40, 30, or even 20 to the minute, or that the auricular wave should not be propagated to the ventricles, or that there should be prolonged periods of 15, 20, or even 35 seconds in which the heart actually stops? Is the essential factor central in the medulla, or in the ganglia of the heart, or in the automatic mechanism of the muscle itself, or in the auriculo-ventricular bundle of His, jun., or in Kronecker's coördination centre? I do not think that we know. The key to an explanation of the cerebral features of the attack is the well-known Kussmaul and Tenner experiment. Consciousness and control of the muscles depend upon a uniform blood-supply in the nerve centres. Even transient pressure upon the carotids, in a suitable subject, may cause syncope, a knowledge of which Kussmaul in describing his original work attributes to Galen.[9] More prolonged pressure may be followed by convulsions. Transient anæmia of the nerve centres is sufficient to explain the vertigo and syncope, and it may be of cardiac origin or in many cases, as Huchard insists, it may be due to local changes in the vessels of the medulla. In any case these cerebral features may be brought into line with recurring attacks of transient aphasia, with or without loss of consciousness, of monoplegia, and of hemiplegia which are not uncommon in advanced arterio-sclerosis.

PROGNOSIS.

In all cases and in all forms the outlook is bad. In a few instances in young persons recovery has taken place. The disease may last for many years. I could see no reason to doubt the statement of the patient in Case 9 that he had had a permanently slow pulse for 30 years, and there are undoubtedly cases of true bradycardia in which good health has been maintained for years. Once the severer nervous symptoms have begun there is very little prospect of complete relief, though, as in Case 2, the patient may live for six years. Even after the most aggravated seizures temporary improvement may follow. The extreme gravity of the condition may be gathered from the cases here reported—seven are dead. Sudden death is the most common and occurred in six cases of the series.

TREATMENT.

In younger patients with arterio-sclerosis it may be worth while to try the remedies which some think may have an influence on the sclerosis; certainly, if there is a history of syphilis, iodide of potassium should be used. With high tension nitrites are indicated. In the senile form with vertigo I doubt if it is expedient to do more than to keep the bowels open and to see that too much food is not taken. The nitrites seem to be helpful in some instances when given freely, in others they are useless. The histories of cases in the literature and of those which I have given speak only too plainly of a condition not much within the scope of our art. So far as I know we have no remedy at our command which will accelerate a permanently slow pulse. Atropine may be tried, as Dehio suggests. A quiet, well-regulated life helps to ward off the attacks of vertigo and syncope as in angina pectoris. Emotional disturbances and over-exertion are to be avoided. In spite of the utmost care and most persistent treatment a patient's life (Case 2) may become a burden with the recurring seizures. For the syncope nitrite of amyl and strong ammonia may be used. When there is a warning, as sometimes is the case, their use may prevent an attack. Brandy, ether and the strong cardiac stimulants may be necessary to revive a patient in a protracted attack. The epileptiform and pseudo-apoplectic attacks may sometimes be prevented by posture. Stokes's patient could ward them off by hanging down the head and in Case 9 rubbing the wrists violently would ward off an attack. Nothing seems to control the recurring attempts at death as in Case 5 and in Dr. Hamburger's patient oxygen inhalations are said to have given relief. With signs of dilatation of the heart and many abortive systoles and infiltration of the bases of the lungs digitalis may be cautiously tried.

Baltimore.

[9] Aus meiner Docentenzeit in Heidelberg, 1903, p. 28. Kussmaul describes one of his experiments on a friend who had boasted of his strength. "His carotids were most favourably placed. Scarcely had I compressed them with my fingers when he turned pale and collapsed off the stool. I had just time to catch him. He recovered consciousness immediately and said 'Where am I?'" This is an exact counterpart of some of the transient Stokes-Adams attacks.

INTESTINAL ANASTOMOSIS FOR PROLAPSED SMALL INTESTINE.

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As the state of affairs in the following case was somewhat exceptional and presents several points of interest I think it worth recording. The patient was 12 years of age when I first saw him and his mother told me that he was quite well up to three and a half years ago when he had a sudden attack of pain in the abdomen and stoppage of the bowels. Poultices were applied but the abdomen became swollen and the child so ill that an operation was performed. This operation apparently consisted in opening the abdomen in the middle line, bringing a coil of small intestine to the surface, and opening it. Relief was thus afforded for about two weeks when the abdomen again became as distended as before. A second operation was performed, another coil of small intestine being brought to the surface to the right of the middle line. After this he obtained permanent relief so far as the intestinal obstruction was concerned, but subsequently the small intestine prolapsed through both wounds to some considerable extent, rendering the condition of the child a very miserable one. The fæces escaped entirely through one of the prolapsed pieces on the right side, nothing but clear fluid escaping from the prolapsed gut in the middle line. The skin in the neighbourhood of these openings became raw and for nine months the child passed his existence lying upon his abdomen with the prolapsed intestine in a receptacle. To add to his trouble he one day passed about a teacupful of pus by the urethra, the supposition being that an abscess had burst into his bladder. However, he had no further urinary trouble and for two years before I saw him he had been getting about carrying his prolapsed intestine in a bag attached to his waist. When I first saw him in May, 1901, his condition was extremely pitiable and is well represented in the accompanying illustration (Fig. 1). He walked into the out-patient room leaning forward and looking very ill. When he undressed and lowered the bag containing his prolapsed intestine, which with its constant wriggling appeared like two large worms attached to the abdominal wall, it was at first sight difficult to say what had happened. After a more careful examination I made out that these coils of gut were prolapsed intestine, the mucous surface being exterior. Fæces only escaped from the point A and nothing but clear fluid from the points B, C, D. His bowels had not acted naturally for three and a half years. On further investigating the case I ascertained that on injecting water at A it escaped freely at B; on injecting at B it escaped after a little while at C; from C it passed freely to D and from D to the rectum. These experimental injections were painful and caused him to vomit, more especially when water was injected with some little difficulty from B to C. What the true explanation was of the boy's intestinal arrangements I did not know but considered the site of his original trouble to be somewhere in the coil of gut between the points B and C.

The question was, Could we do anything to help the poor lad? At first sight it might appear from our injection experiments that all that was necessary was to join A to B and C to D, but as water could only be made to pass with pain and difficulty from B to C I thought that if these several points were successfully joined it would only result in a return of the symptoms of three and a half years ago. What I decided to try to do was to join the small intestine at the point A to the ascending or transverse colon if I could find it. With this object in view on May 22nd, 1901, I in the first place cut away the prolapsed portion of gut, C, D, which was serving no useful purpose. The boy stood the operation well. On August 30th I opened the abdomen above the A, B loop and without much difficulty found the portion of small intestines passing to A, but I hunted for some time before I could find any sign of the large bowel. However, by injecting air per rectum by means of