a man aged 60, it is interesting to note, had always earned his living by cracking stones. He first noticed a lump forming on the extroverted bladder nine weeks prior to admission. For a fortnight it had been very prone to bleed. He complained of considerable pain, especially down the legs. With the exception of the mucous membrane of the epispadiac penis, the whole of the extroverted surface was replaced by a prominent cauliflower-like mass the size of an egg, which bled freely when touched. The openings of the ejaculatory ducts could be distinctly seen, and exuded glutinous fluid. The ureteral orifices were lost in the growths. Both testicles were completely descended. The inguinal and external iliac glands were enlarged and hard. The condition was quite inoperable, and the patient died a month after admission exhausted by pain and haemorrhage. My microscopic sections of the growth and of one of the iliac lymphatic glands proved the tumour to be a carcinoma.

In conclusion, I should like to say that abroad sufficient credit has not been given to Dr. Lendon, of this city, for having devised this new method. It is usually known as Peters's operation. It is, however, only fair to state that Dr. Lendon performed his operation two months before Peters operated upon his first case in Toronto.

REFERENCE.

¹ EPITOME, BRITISH MEDICAL JOURNAL, December 16th, 1905.

BRADYCARDIA, WITH ARRHYTHMIA AND EPILEPTIFORM SEIZURES.*

By JOHN MAGEE FINNY, M.D.DUB., F.R.C.P.I.,

PHYSICIAN TO SIR PATRICK DUN'S HOSPITAL; KING'S PROFESSOR OF PRACTICE OF MEDICINE, SCHOOL OF PHYSIC, DUBLIN.

BRADYCARDIA—"slowheart," or, as very properly insisted on by Gibson, "infrequent beating of the heart"—has of late years assumed a prominence in clinical medicine, and has awakened considerable physiological and pathological interest. The work of Gaskell¹ on the functions of the heart muscle may be considered to have revolutionized the older views of the neurogenic cardiac mechanism, as it transfers the stimulus of the cardiac muscles from the higher nerve centres through the vagi and the sympathetic nerves to the muscle of the heart itself. "The muscular fibres of the heart possess the power of rhythmically creating a stimulus, of being able to receive a stimulus, of responding to a stimulus by contracting, of conveying the stimulus from muscle fibre to muscle fibre, and of maintaining a certain ill-defined condition called tone" (Osler).

Many observers since then have detailed clinical cases based upon this theory—the "myogenic theory"—the more notable being Wenckebach, Jas. Mackenzie, Alfred Webster, George A. Gibson, and John Hay.

The term "heartblock" has been applied to the failure of an impulse, which has originated in the muscles of the auricles, to pass the auriculo-ventricular septum through the auriculo-ventricular bundle of His, and to stimulate a contraction in the muscles of the ventricle. In other words, the auricles contract regularly, but the ventricles do not respond oftener than once in two, three, or four of such auricular contractions.

When the ventricular impulses and systoles are continuously out of rhythm with the auricular contractions the term "bradycardia" is applicable; the rate of the ventricular systoles and the radial pulsations being synchronous, though infrequent—say to 40, 30, or 20 in the minute. Should the ventricular systole be occasionally still further delayed, so that there may be a cessation of ventricular contractions for 3, 5, 10, or 15 seconds in a minute, the term of "arrhythmia" is applicable.

The following case exhibits bradycardia and arrhythmia, and, as a result of the latter, epileptiform attacks of varying severity, in one of which sudden death occurred.

CASE.

Mrs. J. M'N., aged 42, married, was admitted to hospital on June 19th, 1905, under my care, on the recommendation of my colleague, Sir Arthur Macan, who had noticed the remarkable slowness or infrequency of her pulse, *circa* 22 in the minute. She has three living children, and had had a fourth, born on April 27th, 1905, but he died of convulsions at the age of 5 weeks. Since her confinement, nine weeks before admission, she has had frequent "weak turns" or fits, and complains of weakness of her heart. She is unaware of her pulse rate being unusual. She has never had any other serious illness, and denies any syphilitic disease.

When questioned closely as to the fits the patient states that

she never had had any attack before April, 1904—that is, fourteen months before admission—and that she then suddenly fell and became unconscious; that this occurred three months before she became pregnant; that these attacks continued, at varying intervals, during pregnancy; they were not worse during parturition, nor while she was nursing her baby; but that after the death of the child they returned, and they have become much more frequent of late, having had as many as twenty in one day last week.

I instituted inquiries at the maternity where she was confined, but the only note made was that the case was a breech presentation; and no comment of any convulsions, cardiac infrequency, or ventricular arrhythmia. She herself says that her heart was not affected, as at present, until the death of her baby—that is, not until about a month before admission to hospital, and that she never asked advice on the subject, although Dr. Byrne, Medical Officer of Grand Canal Street Dispensary, was in attendance at her house on the baby, during its illness, and up to the day of its death.

On admission the pulse was regular, 20 to 24 in the minute, compressible, and was unaccompanied by cyanosis, dyspnoea, or hurried respiration. There was no complaint made about the heart, except a sensation, when a fit came on, of the heart beating forcibly. Subsequent observation did not confirm this idea, for rather the very opposite happened, as before a "fit" the heart's beat was still less frequent, and sometimes there was an absence of any impulse, or pulse at the wrist, for 5, 8, or 10, or even 15 seconds; and as soon as the "fit" passed, there was no excitement or tumultuous action of the organ.

The heart itself was slightly enlarged; the area of cardiac dullness extended from the left sternal line, at the level of the fourth rib, to the mammary line, and the impulse was under the nipple; a shade of dullness was present at the third left costo-sternal articulation and third space near it. The impulse and radial pulse were synchronous and, as a rule, regular; and when the pulse at the wrist was noted to be absent for five or ten seconds the ventricular systole and impulse were likewise absent. The impulse was abrupt, but never heaving or forcible, and did not give the impression of hypertrophy. A second wavy impulse could at times be seen and felt above the fourth rib, and was probably auricular. The first sound was loud, and could be heard up the whole of the sternum, somewhat prolonged, owing, perhaps, to its slow rate, and was at times droning in character, and with an approach to a murmur. The second sound was sharp, and, like the first, very "deliberate," and did not instantly follow the first; it was pure and fairly loud, the pulmonary element being more pronounced. Careful examination showed the cerebro-spinal, pulmonary, hepatic, digestive, and genito-urinary systems to be free from all organic disease, and menstruation had reappeared a month ago.

Two days after admission the jugular veins, at the root of the right side of the neck, were seen to beat two or three times for each ventricular systole. This phenomenon was present each day during the first two months of her stay in hospital, and could be readily noted and counted, as the rate of the pulse was so infrequent as 20 to 24 in the minute. If these venous pulsations were to be considered as due to auricular contractions, then the auricles were contracting three times the rate of the arterial pulse at the radial; and the ventricular systoles and radial pulsations occurred after intervals of three or four seconds between them and the auricular contractions.

This very interesting question was settled by the application of the *x* rays; and on July 7th Dr. E. J. Watson, our Assistant Physician and X-rayist, kindly examined the patient's thorax with the screen. His report was "that the heart is situated very horizontally; the auricles can be seen beating between the ventricular pulsations; two beats of the auricle were counted between the beats of the ventricle. The auricles seem slightly enlarged."

This is, I believe, the first recorded case where a practical ocular demonstration was made of the mechanism of this irregular pulsation in cases of bradycardia.

Professor W. H. Thompson most kindly took transmission tracings of the right radial, the jugular vein, and the respiration on July 21st. The venous pulsations can be readily seen to exceed those of the radial by two or three to one.

On June 28th the patient had a fit, and during it her face became flushed and her hands shook. It was noticed that just before the fit came on the pulse at the wrist had ceased.

The sequence of the fit following the cessation, or arrhythmic contraction of the left ventricle, was not a matter of theory, for though it rarely happened that a fit came on while under examination, with the finger on the radial or the stethoscope over the heart, and though the vast majority of the fits were recorded by the nurses in charge, or the clinical clerks, after their onset and during their occurrence, still it did so happen that, on several occasions during the patient's stay in hospital, I myself verified the cessation of the ventricular systole and the radial pulse, and was able to call the attention of the class of students to an approaching fit when the intervals between the pulses extended to five, seven, or ten seconds. The house-surgeon and clinical clerks confirmed this fact on many occasions. I was further fortunate in securing a sphygmographic tracing on July 21st, which Professor Thompson kindly took just at the very

* Read before the Section of Medicine in the Royal Academy of Medicine in Ireland, on Friday, March 30th, 1906

moment of the occurrence of a specially long pause, the pulse-rate being about 26 to the minute.

It will be seen that the pause lasted about ten seconds, and at that moment the patient had a slight "fit"—that is, her face flushed, her eyes were turned up to the right, a slight tremor passed over her lips and fingers, and then she came to herself, looking a little frightened, and with a sort of sigh, while her eyes filled with tears.

It is not necessary, and it would be tedious, to give the

on much more frequently than the others, they were the commonest met with. Several occurred during my visits to the bedside, and while the stethoscope was being applied to the precordium and the fingers to the pulse.

A "weak turn" was ushered in and preceded by a delay in the heart's beat and radial pulse. Thus, when a pause lasted seven to ten seconds the patient's face became pale, the eyes had a very pained and frightened look, and some little restlessness of the head and hands ensued; and it ended by a slight flushing of the cheeks, some tears in the eyes, and the patient would give a slight sigh, or little hem or cough, and the weakness would be over. The "slight fit" came on with a soreness or sharp feeling in the stomach and left side of the thorax, which would then extend up the right side of the chest into the head and eyes. The pallor of the face would be more marked, the eyes would turn up to the right side, and the hands would fidget and the fingers catch at the sheets. The patient always came out of this fit in a more frightened and slightly dazed manner, and the eyes had a strained and somewhat blind look for some minutes. The "severe fit"

Fig. 1.—Simultaneous tracings from the lung, right jugular vein, and radial artery. a. Respiration. b. Jugular pulse. c. Seconds in minute. d. Radial pulse.

notes of the daily observations. Each day was like the other as a general rule, and I shall therefore summarize the progress of the case.

From the day of admission, June 19th, to the end of the month the "bradycardia" was generally at the same rate, and varied little between 22 and 28—several observations being made each day—and on the 28th it touched 32 for one counting. In July the same was the record, though it was as low as 18 on the evening of the 1st and the morning of the 5th, and was 16 in the evening of the 17th.

It showed variations sometimes, but very rarely. For example, at my visit, 10 o'clock a.m. on the 23rd, I found the pulse, which was 25 at 7 a.m., beating regularly at 56, and then, in a minute or two, it fell to 22, and that evening it reached 58—the highest since admission. On the 24th it was 25 at 7 a.m., 21 at 9 a.m., 56 at 10 a.m., and 36 at 8 p.m.

The patient, who had been kept recumbent, was allowed to sit up in bed on and after July 28th, and seemingly this was not attended by harm, though the pulse-rate rose to 52 on the 28th, and 54 on the 30th. Fish and eggs were given on the 31st, and on August 3rd she was allowed out of bed to sit in a chair, and to move about the ward. At the same time meat was added to her dietary. This extra dietary and change of posture seemed to have no other effect than to make the heart more regularly infrequent, and its rate for the fortnight following was between 24 and 20—the same whether lying down or sitting—and it fell to 18 on the evenings of the 9th and 11th, and to 16 on the 10th. From August until she left hospital on October 12th, the pulse rate was much the same, while her general health and strength improved, she had had fewer of the slighter "weak turns," but no bad fits.

Naturally, an examination of the heart was being constantly and frequently made by myself, the house surgeon, the resident staff and my clinical clerks, and nothing different from what I have described above was noticeable until July 26th. On this day, between the ventricular contractions, which were 22 in the minute, four to six single sounds could be heard in the minute. These sounds were not accompanied by any pulse at the wrist. Their chief situation was over the right ventricle, at the fourth costal space in the left parasternal line, and they were faintly audible above the fourth rib. This observation was not again met with; but on July 29th and 30th the heart was noticed to beat between 52 and 56, regularly, and the pulse at the wrist agreed thereto; the first sound was of a deeper tone and accompanied with almost a murmur.

On August 27th another and a new feature was noted by the house-surgeon. On listening to the heart, one sound was to be heard in addition to and between the regular heart sounds: the patient while under observation had a "weakness," and nine of these intermediary sounds were counted before the heart beat again, then nine sounds and again a heart beat, then six intermediary sounds, and these sounds gradually became fewer until only one was heard between the ordinary heart sounds.

I must now refer more fully to another feature of this case which I have incidentally recorded, and that is, the attacks of the so-called "fits." Of these there were three kinds, and though probably of the same nature, they became spoken of as "weak turns," "slight fits," and "severe fits." The association of these nervous attacks with the marked arrhythmic retardation of the pulse was more than a matter of fancy. As the "weak turns" came

was more distinctly epileptic with sudden onset, the patient falling back if sitting up in bed, great pallor, convulsive movements of hands and arms, and also slightly so of feet, unconsciousness for five to ten seconds, and then great flushing of the face, and the patient came out of it very confused, and she was dull and nervous the whole day following. She never bit her tongue or passed urine in them.

On July 2nd six fits occurred before noon, and at noon, as she was turning round in bed to take her dinner, she had a fit of unusual severity. The pulse stopped for ten seconds, she became dizzy and weak, her head fell down, the face flushed,

Fig. 2.—Tracings from the radial artery with arrhythmia and syncopal attack. a. Seconds in minute. b. Radial pulse.

and the limbs shook. Five minutes later another fit followed of less severity, and three more fits occurred at intervals of a few minutes. The patient had twelve fits in the course of the day. She was quite unconscious in the attacks, but by her subsequent feelings could say if they were mild or severe; the latter being preceded by the gastric aura referred to.

I was never able, during her first sojourn in hospital, to take the pulse at the moment preceding or following one of these "severe fits."

The number of the "fits" of all kinds—for I look upon them as epileptic in nature, though in varying degrees of intensity—varied from day to day—six on July 1st, twelve on July 2nd, three on 13th, several on 6th, six on 15th, and four on 17th. All through July the patient considered herself very much better, as the attacks were of the mildest form—"weak turns." In the second week of August, however, the more severe fits returned, and the patient became more alarmed, as she was afraid to sit up or turn to the left, as she said these positions would induce a fit, and, for the same reason, if sitting up she dreaded lying back. I looked upon this apprehension, however, as probably nervous, inasmuch that when I induced her in my presence to do as I wished her, as to change of posture, she had no attack. Probably the cessation of the catamenial period was provocative of their recurrence, and of her increased nervous dread of them, as she had not been unwell since June.

The treatment which suggested itself to me at first was directed to combat the "fits," and I gave her bromide of potassium with iodide of potassium; and, from analogy with the good results derivable from the bromides in tachycardia (as Dr. Travers Smith² has shown), I hoped they might also command the bradycardia. But after a week's trial I discontinued it, as it tended rather to

increase than diminish the heart attacks of bradycardia and arrhythmia, and the character of the pulse became more compressible, and the patient looked more depressed.

After further study of the case, and noting that the heart failure preceded the nervous attacks, and that in all probability the cardiac failure to drive the blood into the cerebral capillaries was the cause of the epileptiform attacks, I gave the following prescription with good results, and this was taken without interruption till August 18th: *R. Tinct. strophanthi, liq. strychninae hydrochl. aa mv, acidi hydrochlor. dil. mxx, syrapi limonis 3i, aquae ad 3i; Ft. dosis, mitte 3x; 3i t.d.s.* After August 28th she took "Easton's syrup" until she left the hospital on October 12th, 1905, and she continued the tonic off and on at home, always attributing good to its use. Subsequently she occasionally attended at the hospital, when she stated she was fit for light domestic work and the care of her children, but was never able to do heavy housework.

Though the "weak turns" were still present, she had had only two severe fits, so far as could be ascertained, between October, 1905, and February, 1906. At the time of her visits the pulse was still infrequent, but regular, and about 24 in the minute. Dr. Watson examined her on February 25th by *x* rays, and demonstrated that the heart beat regularly at 28, and that the remarkable want of agreement between the contraction of the auricles and of the ventricles, which had occurred in July, 1905, no longer existed, and there were as many contractions of the ventricles as there were contractions of the auricles. It was also noticeable that the jugular pulsations were very indistinct, and could not be well counted.

With the intention of making a series of observations on this point, and for the purpose of having fresh and extended sphygmographic tracings, so as to enable me to speak more accurately as to the nature and the frequency of the epileptiform attacks, I readmitted the woman to hospital on February 28th, 1906, six months after her first admission to hospital. The following notes were made by Mr. William Pearson, clinical clerk, to whom I am indebted for the accuracy of its report and for his graphic description. Owing to the terribly sudden and fatal termination by a syncopal attack on the sixth day after her readmission I give the notes in full:

The pulse, taken at the wrist, on March 1st, at 10 a.m., was regular, full, of good tension, and numbered 26 beats to the minute. It corresponded with the frequency of the heart. The heart's impulse was seen to be widely diffused; its action was slow and deliberate in character, being first apparent over the auricular region to the left of the sternum, and gradually passing downwards, over the ventricles, to the position of the normal apex beat. This slow movement was reminiscent of a wave of peristalsis crossing along the bowel beneath a thin abdominal wall. The ventricular impulse was distinct, and promised to make a good cardiograph tracing, such as Professor Thompson hoped to make in a day or two. Pulsations were to be seen in the root of the neck, apparently three for each ventricular beat. The urine was clear, amber colour, acid in reaction, specific gravity 1020, and it contained a faint trace of albumen, but no sugar. The patient asked for, and was given, permission to get up out of bed.

After her dinner, at 12.30, she was proceeding with a nurse from one ward to another, for the purpose of having her weight registered, when she suddenly—without warning or cry—collapsed on her knees, and fell to the ground in a heap. Her muscles were completely relaxed. The house surgeon, Dr. Powell, was instantly summoned, and found her lying deeply cyanosed, pulseless at the wrist, and showing no evidence of respiration, except one feeble gasp. He thought she was dead. He immediately injected *m* vij of liq. strychninae and used artificial respiration, leaving her lying with a pillow under her head, and having hot bottles placed round her. She did not make any effort to breathe for four minutes, and he would have given her up as dead, except that on auscultation the heart gave a feeble flutter. Oxygen inhalations were tried also. After some time, the artificial respirations being persevered in, the heart began to beat feebly, and respiration, somewhat stertorous in character, was resumed. After seven minutes the pulse at the wrist became palpable, and numbered about 40 to the minute. She was now carried back to her bed, and when I saw her, forty minutes later, she was still deeply unconscious, with the eyes turned up and the pupils most widely dilated. She then commenced to moan and cry. Her arms were folded across her chest in a tonic spasm, her body arched in opisthotonos, the face in trismus, and urine was passed under her; the tongue was slightly cut, although a gag had been employed from the moment this tonic spasm set in. Chloroform inhalations calmed the convulsions and under its use the pupils became small—to a pinhole size; the heart reached 56 to the minute, while the respirations assumed a modified Cheyne-Stokes respiration ("Biot's respiration").

The respiratory rhythm showed a period of 70 seconds duration in which respirations occurred, followed by a period of 26 seconds of complete apnoea, but the onset and cessation of respiration were quite sudden—the first and last breaths taken being of equal amplitude with the others. The pulse gradually sank to 40. After an hour the knee-jerk and conjunctival reflex were obtainable, though the pupils were again widely dilated. At 2.30 p.m. (that is, one hour and forty minutes from onset) the attack passed off, and the patient fell asleep. She awoke at 4 p.m., and was given 25 gr. brom. pot., and had three attacks of vomiting. Pulse and respirations were then noticed to be synchronous—namely, 28 in the minute.

The following morning (March 2nd) the patient was quite calm, but was totally oblivious of the entire of yesterday's attack, and, when asked, stated she had not got up at all, and was not sick in her stomach. Her pulse was regular, 36 in the wrist, and two tracings were made (Dudgeon's sphygmograph), one in the recumbent, the other in the sitting posture. They are the same in frequency, and exhibit a sharp up-stroke, which is short, followed by a prolonged line of gradual descent, which shows faint vibrations.

March 3rd. A complaint of headache in the right temporo-parietal region. The tongue was a little sore from the effects of the fit of the 1st. The pulse was 22, and in a few minutes later it counted 32. It was weaker than usual, and somewhat irregular. Temperature, which had gone up to 99° on the 1st after the severe fit, had fallen to its usual subnormal standard of 97°. The tonic mixture of iron, quinine, and strychnine, which was resumed on the 2nd, was continued, as the patient felt it did her good; and, though very anxious to be allowed up, it was thought safer to keep her in bed—much to her chagrin.

March 4th. Two rather severe attacks occurred between midnight and 1.10 a.m., then several minor epileptiform attacks occurred this morning, and in the afternoon an almost continuous series of seizures. At noon, during a short attack, the heart beat 140 per minute for some time. At one period, towards the end of a fit, on auscultation, the usual infrequency of 24 beats were first heard, and suddenly it increased to about 150, and maintained this high rate for just 20 seconds, when it again suddenly fell to its usual, 24. In the afternoon a severe seizure came on at 4 p.m., and lasted four and a quarter hours. Cries and shrieks were emitted, convulsive spasms and opisthotonos occurred, while at times strabismus and Cheyne-Stokes or Biot's types of respirations were present; the pupils were widely dilated. Chloroform was freely administered, and at times artificial respiration had to be employed. The whole attack, which fortunately occurred while she lay in bed, closely resembled the bad attack of the 1st instant, and death seemed momentarily imminent. The pulse at 2 p.m. was 24, at 5 p.m. it was 150, at 6 p.m. 22, and after the attack, at 10 p.m., it was 32. The attack might be considered of a somewhat milder type, but it lasted much longer. It was succeeded by other shorter attacks at 9 p.m., 9.30, 10.30, and 11 p.m. Next day, March 5th, was marked for vomiting twice and six mild fits in the evening; the pulse at six observations being 26 to 32. Three fits occurred between 8 and 9.30 a.m. on the morning of the 6th, and in the last of these she died. The pulse at 2 and 6 a.m. being, as usual, about 28.

In the final syncopal attack of 9.30 every effort was made by the resident staff to restore life; and artificial respiration was kept up by the resident pupil, Mr. Gibbon, for fully twenty minutes, but all to no purpose, as she never breathed. No sign of life returned, and she was practically dead the instant of its onset.

This sudden termination of life was not unexpected, as the alarming symptoms and cardiac failure of March 1st prepared us for the possibility of their recurrence and of death under these circumstances. Most unfortunately permission to make an autopsy could not be obtained, and thereby much pathological information has been lost.

REMARKS.

Of the many interesting clinical features presented by the foregoing case the first and most striking is the infrequency of the pulse—so-called "bradycardia." This symptom is in itself not very uncommon, and every physician must occasionally meet with cases where the pulse-rate is slow, either temporarily or permanently. Indeed, during part of the time this patient was in hospital I had under my care two instances of this condition, though due to very diverse causes—one in a boy, aged 6, who was unconscious, suffering from the pressure signs of tuberculous meningitis, and the other in a man, aged 72, presenting atheromatous arteries and fatty heart, in whom the pulse was 30 in the minute, and syncopal attacks not unfrequently occurred. The bulk of the recorded cases of slowly beating hearts belong to this latter group, and it was in such conditions occurring in old men that Dr. Stokes³ and Dr. Adams published cases of slowly beating hearts with paroxysmal syncopal, or

pseudo-apoplectic attacks—a group of symptoms now spoken of as the Stokes-Adams syndrome. It will be in the recollection of the Academy that last December Dr. Parsons⁴ submitted a very full communication and recorded the clinical features of a typical example of this condition in a male patient, aged 68 years, who exhibited paroxysms of this symptom, with a pulse-rate of 30 to 40 in a minute, and who was afflicted with general arteriosclerosis.

Thirteen cases of bradycardia are collected and reported by Dr. Alfred Webster,⁵ in his valuable article on Cardiac Arrhythmia in relation to Cerebral Anaemia and Epileptiform Crisis. Of these, 7 were over 50 years of age, and all except two were males; and the 3 cases which illustrated Dr. G. A. Gibson's paper⁶ on the same subject were in males aged 80, 68, and 50 respectively.

The case of Mrs. M'N., which I have recorded, makes, then, a marked exception, in that the regular infrequency of pulse, at the rate of 22 in the minute, was present in a woman, aged 42, and who was free from all cardiac, valvular, and arterial disease, and whose kidneys and other organs were apparently quite healthy. As her case does not come under the class of cases due to either senility, with myocardial degeneration, or to arteriosclerosis, the cause, therefore, must be sought for elsewhere.

The second important feature of my case was the arrhythmia. The pulse-rate, as a rule, day by day and month by month, was between 21 and 28, and, as can be seen by the sphygmogram [exhibited] taken during 120 seconds, it was perfectly regular. Yet at varying intervals, the pulse became irregular, and a state of pulselessness—lasting 5, 8, 12, or 15 seconds—would occur without any cause or influence that could be explained, and which culminated in the terrible attacks of pulselessness (lasting seven minutes) which occurred a few days before death, and in the last of such cardiac arrests she died suddenly.

During the shorter cessations of the pulse, careful observations of the heart by stethoscope and hand failed to detect any ventricular contraction by sound or impulse, and the solitary sphygmogram Professor Thompson was able to make during one of the attacks of arrhythmia, shows an absence of pulse wave for 4 seconds, and again for 10 seconds (see Fig. 2).

The idea of independent auricular contraction was forced on my mind by the observation that for every ventricular systole the jugular veins at the root of the neck beat three times. I do not wish to imply I made a discovery, but I found a novelty in my clinical experience which I was able to study at my leisure, and under peculiarly advantageous circumstances, for not only was the phenomenon constant day by day, but the rate of the pulse was always so slow or infrequent that it was easy to note these prominent features. This relative want of synchronicity of the jugular pulsation and the heart's beat was not unknown to the great Stokes,⁷ and was recorded by him as having come on in the course of one of his cases of fatty degeneration of the heart with pseudo-apoplectic attacks in a man aged 68.

Since his time many observers have recorded the same, although all do not take the same view as to its mechanism.

The tracing of the pulsation at the root of the neck—kindly made for me by Professor Thompson—shows this condition graphically, and it can be readily observed that two or three elevations occur in the jugular tracing during the diastole of the ventricle (see Fig. 1).

In this connexion I would again emphasize the most valuable and, as far as I know, unique confirmation in the fact that the *x* rays demonstrated a distinct double movement of the auricles for each ventricular movement—that is to say, there were three cardiac movements, in two of which the ventricles took no part. It also showed that both ventricles acted together, and that there was no "hemisystole."

It is not my intention in this clinical record, even if it were in my power—which it certainly is not—to further discuss this most interesting and fascinating physiological and pathological subject, and I would refer to the works of Wenckebach,¹⁰ Alfred Webster,⁵ Mackenzie⁷ and Gibson,⁸ with their series of pulse tracings and other evidences, to prove the reality of this cardiac arrhythmia. I would adopt the words of Dr. John Hay¹¹: "Heartblock

is no longer a theory; it is a fact, and has been recognized as such ever since Gaskell published his classical research on the contraction of cardiac muscle."

Of the many contributions to the literature of the subject that have lately appeared in the medical journals, one in particular struck me as so very remarkable that I cannot refrain from referring shortly to it. It is a paper by Henry Harford of Nottingham, under the title of Remarks on a Case of Gummata of the Heart.¹² He bases his remarks on a case of a married woman, aged 32, in which death occurred from heartblock after many attacks of long pauses, in each of which death seemed imminent; and a syphilitic gumma was discovered in the anterior wall of the right ventricle, involving the auriculo-ventricular septum. "The prominent symptoms were, on several occasions on many different days, that the heart was observed to beat 84 per minute for a variable time, and then to stop for five to fifteen seconds. During this interval, on auscultation over the right heart and over the pulmonary area, the auricles could be heard to continue beating. There was no second sound, no pulse could be felt at the wrist, and no cardiac impulse at the praecordia. The face became very pale; the patient had a slight convulsion affecting the face, arms, and legs; the eyes were turned upwards and to the left; a few inspirations (four or five) were taken, and the whole heart resumed regular and somewhat violent action." In one of these attacks the patient died.

In my case another and most interesting point in connexion with the arrhythmia is suggested by the occurrence of the small sounds which have been heard in the intervals between the regular systolic ventricular contractions.

During most of these pauses perfect silence of all heart sounds existed, and this was the rule. Yet, as I have recorded on a very few occasions, one, two, or more distant sounds could be heard over the base of the heart; and these sounds were single sounds, not as if the heart sounds were reduplicated.

These feeble distant sounds seemed to occur between the strong ventricular systolic sounds, to be unaccompanied by a pulsation over the ventricle, either the right or left, though sometimes a wavy pulsation could be seen above the fourth rib.

Three questions arise bearing on these sounds:

1. Are the sounds due to a hemisystole of the heart—the right ventricle contracting while the left does not?—a view hard to understand, though propounded by Sir William Broadbent,¹³ and supported by J. Seymour Maynard,¹⁴ and accepted, with great reserve, by Wenckebach. On this point I give no opinion; but the theory gains no support, nay, rather is refuted, by the observations made by the aid of the *x* rays.

2. Are these sounds produced by the rhythmic contractions of the auricles, acting alone, and unaccompanied by ventricular contractions?—a view favoured by Wenckebach, and supported by G. Gibson and Malet's observations.⁹

3. Can they be produced by feeble ventricular contractions which are not strong enough to empty the ventricular contents into the aorta in sufficient volume to cause the closure of the aortic valves and so produce a diastolic second sound?

The last feature of interest presented by my case is the occurrence of paroxysmal unconsciousness and epileptiform attacks. These attacks came on at irregular periods, and in varying degrees of severity, sometimes several in a few hours—six to twelve in a day—and, again, not for some days; and in severity they varied from a sudden pallor, followed by a flushing of the face, to slight convulsive movements of hands and legs, with the eyes turned up and fixed; and again, to a more severe type, which caused a fall and more general convulsion, lasted longer, and out of it the patient came to more slowly and feeling strange and frightened; and the last attacks were signalized by prolonged unconsciousness and coma, with absolute failure of heart, and sudden death.

The question presses—Were these earlier fits examples of epilepsy mitior (*petit mal*) occurring in a woman, in whom were present cardiac infrequency and cardiac arrhythmia, simply as a coincidence? or were the cardiac affection and the fits in direct relation to one another as cause and effect, and, if so, did the fits cause the cardiac phenomena, as claimed by Tripiër, or vice versa?

When the patient came under my care I was at first inclined to think she was suffering from epilepsy—and this opinion I based on her history—namely, that fourteen months before she had suddenly fallen in a fit, to which succeeded four others the same day; and that up to the day of admission to hospital she had had recurrence of fits. I accordingly gave her a course of treatment by the bromides, and had her under close observation. In a short time, however, I saw that the more likely connexion between the fits and the slow pulse lay in the direction of the cardiac arrhythmia being the direct cause of the fits. Later on I could foretell by two or three seconds that a fit was imminent, and I was able to observe that the fits were not due to simply the infrequency of the pulse, for they did not occur even while the pulse-rate was as slow as 18 to 20 in the minute, but that they were sure to occur when arrhythmia occurred and when pauses of 8 to 10 to 15 seconds were noticed. It was the more prolonged cessation of circulation, and presumably the cerebral anaemia rather than venous stasis, which caused the syncopal and convulsive attacks.

That cerebral anaemia is a cause of convulsion is well known and daily verified, and the experimental work of Kussmaul and Tenner, by inducing cerebral anaemia and causing epileptic convulsions, is still uncontroverted.

For a full account of this subject in relation to arrhythmia I would again refer to the work of Dr. Alfred Webster. The conclusion he arrived at after analysing the clinical features of 13 cases of slow pulse and epileptiform attacks, and after making many hundreds of observations and pulse tracings on his own case—included in the 13—seems to commend itself as the true explanation. "Our own experience," he writes,¹⁵ "tends to show a slow pulse, 18 to 24 in the minute, with even a moderate degree of irregularity, is not associated with convulsions. For example, a pulse-rate of 20 in the minute, which means an asphyxial state of 3 seconds each, or even a pulse-rate of 12, which equals 5 seconds in each pause, is not enough, so long as it is fairly regular; but when the pulse is irregular, then convulsions will be induced."

Accepting these conclusions—to which many recorded cases of "heart-block" conform—I would say, therefore, the arrhythmia, and not simply the bradycardia, should be looked upon as the causal factor in the production of the convulsions.

When, however, we look for the cause of the arrhythmia we must acknowledge we are not much nearer its discovery, as the only point of agreement between various observers, on the Continent and America as well as in this country, seems to be the absence of any gross lesion by which the symptoms of Stokes-Adams syndrome might be accounted for, if we except arterio-sclerosis of the basilar artery and the circle of Willis, and of the vessels of the medulla. Granting these pathological changes to be the cause, how are they applicable in such a case as that of Mrs. M.N.?

I shall not, therefore, take up more of your time in discussing the many theories propounded to explain it, and in recording my case—devoid, as it unfortunately is, of the results of a *post-mortem* examination—I am sadly conscious I have added little to the etiology, and nothing to the pathology, of the condition. But if I have awakened an interest in the clinical record of a very unusual and rare case, and if the full details of its course to a fatal termination may stimulate others to a clearer conception, and the unravelling of the mysteries attending bradycardia, I shall feel my claim upon your patience and time has not been unwarranted nor misplaced.

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THE late Mr. Joseph Hinks of Edgbaston, whose will has now been proved, left £100 to the Birmingham General Hospital, and £50 each to the General Dispensary, the Queen's Hospital, the Birmingham and Midland Free Hospital for Sick Children, and the Deaf and Dumb Asylum—all at Birmingham.

RICKETS IN RELATION TO THE DURATION OF LACTATION.

By A. DINGWALL-FORDYCE, M.D. EDIN., F.R.C.P. EDIN.,

PHYSICIAN FOR DISEASES OF CHILDREN, NEW TOWN DISPENSARY, EDINBURGH; MEDICAL REGISTRAR, ROYAL INFIRMARY, EDINBURGH.

THE clinical observer of disease in childhood is continually confronted by an immense number of patients exhibiting the immediate or more remote symptoms of rickets, and in a study of the disease he is forced to consider the influence of the various methods of infant feeding on its incidence, maternal nursing being naturally the first condition to come under consideration.

In a former paper I have discussed the occurrence of the return of menstruation during lactation and also the overlapping of pregnancy and lactation, and have shown that whereas the former condition by no means necessitates weaning, the latter undoubtedly does, and owing to its comparatively frequent occurrence and the usual date of its onset, I was led to recommend a lactation of eight months' duration. I have since sought to determine the relation of the incidence of rickets to the varying conditions under which the function of lactation is exercised and to the duration of the exercise of this function.

With this object in view I have examined, irrespective of the cause which led the mother to seek advice, 200 children of different families and aged from 6 months to 3 years. These children I have divided according to their early feeding into the following four categories:

(a) "Breast feeding," that is, children reared wholly or mainly on the breast for from 8 to 12 months, and also children aged from 6 to 8 months at the date of examination, who had up to that period been nursed wholly or mainly on the breast.

(b) "Hyperlactation," that is, children reared wholly or mainly on the breast for over twelve months.

(c) "Mixed feeding," that is, children reared partly on the bottle and partly on the breast for from two to eight months, and also children reared wholly on the breast for from two to eight months.

(d) "Bottle-feeding," that is, children reared entirely on the bottle, or where breast nursing lasted less than two months.

This classification was adopted as being the most practical, although by using it one suffers under the obvious disadvantage of being unable to consider the conditions existing where the infants are strictly speaking breast-fed.

Marked physical signs alone were considered as definite evidences of rickets, and as such were taken marked beading of the ribs, enlarged epiphysis, wide fontanelle, and deformities of the thorax, limbs, and spine; while cases with slight beading of the ribs, late teething, marked head sweating, recurrent bronchitis, contracted chest, etc., were considered as slight cases of the disease.

Of these 200 cases rickets was marked in 61

" " rickets was slight in 55

" " there was no rickets in 84

rickets accordingly occurring in 116 cases or 58 per cent.

A classification according to age shows:

Of infants aged—

from 6 to 12 months rickets was present in 55 per cent.

" 12 to 18 " " " 63 "

" 18 to 24 " " " 65 "

" 24 to 36 " " " 52 "

TABLE I.—Showing the Occurrence of Rickets in the Cases Classified according to the Nature of the Early Feeding.

	Rickets.		Total Rickets.	No Rickets.	Total Number of Cases.
	Marked.	Slight.			
(a) "Breast" ...	18	27	45	45	90
(b) "Hyperlactation" ...	10	8	18	13	31
(c) "Mixed" ...	5	5	10	5	15
(d) "Bottle" ...	28	15	43	21	64

From Table I it is seen that the amount of rickets among cases mixed-fed is approximately the same as among those bottle-fed, but differs in this respect, that whereas among the former the percentage number of