As regards treatment, most of the recognized principles of cardiac therapeutics apply. Some special points, however, deserve attention.

1. The exciting cause must be sought. Sometimes this is obvious—for example, when rheumatic fever has led to the vicious circle of aortic regurgitation and cardiac ischaemia, or when pneumococcic infection has led to that of pneumonia and heart failure. At other times close investigation of detail will be required. In law "*de minimis non curat lex*" may be pardonable; but "*de minimis curat medicus*" is a safer motto for the physician dealing with obscure disease. Thus an ill-ventilated gas-lit office or over-indulgence in tobacco may so depress the heart, even when organically sound, that the pulse becomes feeble and intermittent and life miserable and useless, the heart and general health depressing each other. Removal of the exciting cause may in itself ensure recovery.

2. It is frequently desirable to actively treat *each* of the several factors concerned. In pneumonia, for example, the practitioner who merely watches the lungs, and is oblivious of the more important cardiac signs, loses the best chance of helping his patient weather the storm. The finger on the pulse is as important as the stethoscope on the chest. Again, if in the rush of the out-patient room the unsound heart of a coalheaver is stimulated with digitalis and strychnine, while no steps are taken to lessen the daily toil that is hindering Nature's efforts at compensation, the remaining span of life will probably be but brief.

3. Where there is a choice of treatment, each practitioner must attack what appears to him the *locus minoris resistentiae*. Hence results a varying *modus operandi* for the same disorder, a variety which puzzles the patient, especially if several doctors are consulted in succession. An example will illustrate the point: A stockbroker of advancing years suddenly collapses, and is found to be suffering from insomnia, failing heart, crepitation over both bases, albuminuria, general anorexia and dyspepsia. One physician orders him to Switzerland, believing that complete change of scene and air will so restore his nervous system that circulation, digestion, and renal organs will rapidly get back into working order. Another prescribes a long spell of bed. No *Stock Exchange Daily List* can worry him; recumbency will give his heart less work; the warm bed will act on the skin and relieve the kidneys; the lighter diet will facilitate the work of the stomach and liver; the improved circulation and digestion will relieve the lungs and react in favour of the heart. In brief, this treatment ensures physiological rest to brain, heart, lungs, and viscera, a change of air and scene being prescribed at a later stage. As a result of these cumulative measures, the vicious circle is converted into a healthy one, all the organs improving by degrees and helping one another in an ascending scale. Here are two methods of breaking the vicious circle, and each method would at times be most successful.

4. The close dependence of the heart on the central nervous system is shown by the existence of several vicious circles in nervous introspective patients. Oft-times there is a baseless fear of sudden dissolution, or some trivial ailment is magnified into such importance that all useful altruistic life is ruined. Here a few well-chosen words from a trusted counsellor may sever the bonds and loose the sufferer from a state of despair. Where insomnia forms part of the neurosis, a dose of morphine may render great service, acting, in the words of Pridgin Teale, "like a good coxswain, who rallies a crew who had been catching crabs, and makes the men pull together."

5. Many vicious circles associated with the myocardium arise from the failure of compensatory changes, as, for instance, when a salutary hypertrophy is followed by undue dilatation, caused either by degeneration of the myocardium or by the excessive work thrown on the heart. Here treatment may do much to increase the vigour of the myocardium or to lighten its load. By either one or both courses the failing compensation may be so restored as to render the heart once more equal to its task. Above all must an ample supply of pure blood be secured to the myocardium.

CONCLUSION.

Indulgence may fairly be claimed for this the first attempt to deal systematically with the vicious circles associated with a great organ such as the heart, the capital, so to speak, of an empire with whose remotest outposts it is, for good as well as for ill, in constant communication. The subject is one which throws some fresh light on Hippocrates's aphorism:[15] Συμπαθέα πάντα· κατὰ μὲν οὐλομελίην πάντα, κάτα μέρος δὲ τὰ ἐν ἑκάστῳ μέρει μέρεα πρὸς τὸ ἔργον, that is, "the whole body sympathizes with every member, and every member with the whole throughout its structure," and emphasizes the limits that are imposed on the *vis medicatrix naturae*.

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REPORT, WITH COMMENT, OF SIX CASES OF HEART-BLOCK,

WITH TRACINGS, AND ONE POST-MORTEM EXAMINATION OF THE HEART.

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IN the *Journal of Experimental Medicine*, January, 1906, Erlanger summarizes the results of his admirable experimental investigations upon the function of the auriculo-ventricular bundle as follows:

1. In the dog, the impulse which normally causes the ventricles to contract is conducted through the auriculo-ventricular bundle of His.

2 By compression of this bundle all stages of heart-block may be obtained. These include (*a*) an increase of the inter-systolic pause; (*b*) an occasional ventricular silence; (*c*) regularly recurring ventricular silences—for example, one silence in ten, nine, eight, seven, six, five, four, three, and two auricular beats; (*d*) a two-to-one rhythm; (*e*) a three-to-one rhythm; (*f*) complete heart-block.

3. As a rule, the ventricles take on a constant slow rate at the moment complete heart-block is established. Occasionally, usually when the block becomes complete suddenly, there results a marked preliminary slowing of the ventricular rate. The auricular rate does not change.

4. When the block is complete, stimulation of the vagus nerve has no, or but a minimal, effect upon the rate or force of the ventricular beats, whereas the auricles still react normally.

5. When the block is complete, stimulation of the accelerator nerve increases the rate both of the auricles and ventricles.

6. When the block is complete, the rate of ventricular beats may not be materially affected by variations in the general blood pressure, nor by asphyxia, nor by interference with the coronary circulation.

In his book, *Das Reizleitung's System des Saugetier-Herzens*, Tawara records his wonderfully painstaking anatomical researches. He demonstrates that the A.V. bundle is the trunk of a tree-like system (comparable to the bronchial system) which has its roots in a tangled "knot" above the fibro-cartilaginous atrio-ventricular septum, that it sends backwards a short bundle to the neighbourhood of the anterior part of the coronary sinus, where this becomes connected with the auricular muscle fibres. The trunk of the tree after breaking through the auriculo-ventricular septum splits up into two prongs, one for the left, the other for the right ventricle. These run subendocardially and divide into branches, which re-divide till the ultimate twigs come into direct connexion with the ventricular muscle fibres. Throughout its course this tree is isolated by means of connective tissue, and within it blood vessels and nerves are to be found.

Through the methods of instrumental registration of the working of the cardio-vascular apparatus, as evolved and interpreted by Mackenzie[1] and as further elaborated by him and Wenckebach, physicians are gradually attaining greater accuracy in the study of cardio-vascular cases, and it is through reading their publications that we are tempted to write these reports

For recent reviews of the literature and publications of excellent tracings the reader is referred to two papers, one

by Ashton, Norris, and Lavenson,[2] the other by Gibson and Ritchie.[3]

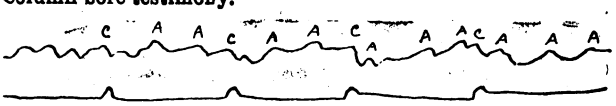
Here we propose recording in full the history of a case of acute heart-block with *post-mortem* findings, of which only a bare notice has previously appeared, reviewing from the clinical standpoint the cases which have come under our notice, and publishing some of the more interesting of the tracings taken from these patients.

CASE I.

The patient was a young man of 30, of excellent family history. Six years previously he had acquired syphilis, for which he was treated by mouth for six months. Two years later he contracted gonorrhoea, from which he never entirely recovered, a morning urethral drop being regular in its appearance. Till six weeks previous to his admission into hospital his powers of endurance were good, and his field of cardiac response wide. At that time he had an acute exacerbation of his gonorrhoea. This after three weeks led to a bilateral acute epididymitis. He developed fever, felt chilly, suffered from headache, and later experienced pain in the hamstring and wrist tendons. Three days prior to admission, whilst resting in bed, he suddenly felt faint and everything became dark. He momentarily lost consciousness; when he awoke he arose and tried to sweep the floor but he became dizzy and fell upon the bed. Five seizures occurred in quick succession, all being of the same character. During the next three days he had many fainting spells, but no new symptoms arose. When admitted his temperature was 100°, pulse 80 to 90, and it remained at about that figure for two days, during which only a few seizures occurred, the pulse at these times being very slow. Then it became permanently infrequent, and continued so till his death two weeks later. The following status was taken by one of us on the third morning after his admission:

The patient is a short, thin, wiry, muscular man. He is somewhat cyanotic, but there is no oedema. There is no paralysis, and his joint movements are free. Little petechial spots are present over the body surface. The hamstring and wrist tendons are hypersensitive and somewhat rigid, but not swollen. The lungs are normal. The heart dullness is widened, the palpable apex being 1 in. outside the mid-clavicular line. A systolic bruit is present, and is most pronounced at the mitral area, and is transmitted in all directions. The ventricular rate is 22 to the minute; over the auricles faint double sounds are heard in the intervals between the successive ventricular sounds. The radial pulse is 22 to the minute, regular in rhythm, of good tension; in the pause tiny beats can be appreciated. The neck veins are not dilated, but pulsate much more rapidly than the radial pulse, apparently about 4 to 1. There is now no urethral discharge. The prostate is enlarged and of irregular consistency. On massaging it a copious amount of muco-pus is discharged. In this no gonococci could be detected. The epididymes are hot, enlarged, and tender. The liver and spleen are normal in size. The kidneys are not palpable. The nervous system and special senses are intact.

Later investigations showed that the urine contained many threads, but was otherwise normal. The leucocytes rose to 40,000 per c.mm., the polynuclears being in relative excess. Repeated blood cultures were negative. The systolic blood pressure (Stanton apparatus) was 140 to 145 mm. of Hg, and the lower point of greatest oscillation of the mercury column was from 100 to 110 mm. At this level, between the larger oscillations due to the ventricular systoles, were smaller oscillations, probably due to the contractions of the auricles. The sphygmograph of the radial pulse also showed on the down stroke the little elevations which the finger previously could detect, and to which the movements of the mercury column bore testimony.



Tracing 1 (Case 1).—Venous and radial pulse.

The patient's temperature ran a marked septic course, at times intermittent, at times remittent, varying from 105° to 97°. *Pari passu* the pulse varied from 40 to 20, thus showing response to temperature changes. Chills occasionally occurred, and marked sweating was present. During two weeks many paroxysmal seizures happened. At first they were of the nature of fainting spells; later the pseudo-apoplectiform, and later still epileptiform attacks developed. The cyanosis became deeper, the patient often becoming very blue during a prolonged seizure. During the milder paroxysms the neck veins continued to pulsate, and the auricular sounds were still audible. During the severer attacks, the neck veins, after pulsating for a few seconds, became much distended; they no longer pulsated, and no sounds could be heard over the base of the heart. The patient continued to weaken, the heart gradually grew feebler, and death occurred in an attack fourteen days after his admission. A few hours before death the temperature fell from 105° to 95°, the pulse, however, remaining at 40, to which it had risen with the preceding temperature rise. The greatest length of absence of pulse-beat up to the time of his death was 40 sec.

The clinical diagnosis was: Acute gonorrhoeal epididymitis, acute gonorrhoeal sepsis which had led to an acute involvement of the bundle of His.

We regret that the temperature and pulse charts, and the list of the sphygmograms were burnt in the San Francisco fire. The curve (No. 1) appended is a traced copy of a sphygmogram taken with the Mackenzie instrument. The sphygmogram had become adherent to the wood on which it was placed to dry, and so escaped destruction. It shows that heart-block was present, but it is too poor to admit of a strict analysis.

Autopsy Report of the Heart (only part obtainable).—Mr. K., patient of Drs. Jellinek and Cooper, January 12th, 1906.

Clinical Diagnosis.—Gonorrhoeal sepsis, death from acute Adams-Stokes's disease with heart-block.

Heart of normal size. Muscle of normal appearance everywhere, except directly underneath the membranous septum near the large flap of the mitral valve. At this point on the left side of the septum the endocardium shows several ragged perforations, the largest one about 2 mm. in diameter, which lead into deep defects in the myocardium. The muscle near the lesion in the endocardium is quite opaque and soft. On the corresponding side on the right side of the septum the space between the median flap of the tricuspid valve and the upper part of the muscular septum is filled with solid mixed thrombus.

Smears from the thrombus show much granular material, few red and white corpuscles. No bacteria found, in spite of careful search. Sections of the diseased area show a complete necrosis of the heart muscle, which begins at the membranous septum and extends into the muscular septum on the right side for a distance of 10 mm., on the left for 8 mm. In the middle of the section it does not go quite so far. On the right the necrotic area is covered with an ordinary mixed thrombus. The necrotic mass contains some nuclear débris in places and also quite a little crystalline pigment (haemotoidin). Along the edge of the necrosis one finds some leucocytes (polynuclears and lymphocytes), and also some new-formed connective tissue cells. The adjoining muscle is normal. Serial sections show that a medium-sized artery entering the necrotic part from the membranous septum and two of its branches, of which one runs towards the left and one towards the right side of the muscular septum, are filled with thrombus consisting of granular material and fragments of leucocytes.

No bacteria found in the thrombus in the arteries, in the necrotic area, or in the thrombus on the outside. Specimens were stained with Weigert's modifications of Gram's method, methylene blue, eosin, dilute carbo-fuchsin. Anaemic necrosis of muscular septum in the region of the His bundle.—W. OPHULS.

CASE II.—*Incomplete Heart block but no Paroxysmal Seizures.*

H. Y., a farm labourer, aged 31, was admitted into the German Hospital, July 7th, 1906, complaining of occasional shooting pains in the cardiac region. Father and mother were both dead, the former from cancer at 68, the latter at 54, cause unknown. When a child he had measles. He knew of no other illness, and denied all venereal infection. He drank but little, but smoked and chewed considerably. Till a week previous he had worked hard and felt well; since then he had experienced shooting pains in the lower left mammary region. These travelled towards the margin of the left chest, were of momentary duration, and after them he felt somewhat breathless. He had never fainted or had any kind of a seizure.

Examination showed a thin, tall individual whose face and hands were much tanned. His nails, lips, and gums were pale. Both supraclavicular and infraclavicular regions were retracted, and had been apparently the seats of old tuberculous trouble; there were, however, no indications of recent activity. A few hard small glands were palpable above both clavicles. There was no anaemia, and none of the systems gave any further indication of disease except the cardio-vascular.

The heart was of normal size; the rate was 36 to the minute with regular rhythm. The apex beat was weak but localized. The heart sounds were clear and distinct. Over the base faint sounds were at times audible during the ventricular silence, at other times not; the point of greatest intensity of these faint sounds was in the second right interspace, 1 to 1½ in. to the left of the sternum. At the root of the neck much more rapid vibrations were apparent in the neck veins, the latter not being distended. The ratio of the venous pulse to the carotid beat seemed about 3 to 1. The pulse-rate was 38 to the minute, apparently regular in rhythm; the tension was fair, both radial pulses were equal. The walls were thickened, but the arteries were not tortuous.

At times the venous pulse was not visible, and deep respiratory efforts or muscular movements were required before the vein waves could be demonstrated to observers. On standing, the pulse-rate was 42 to the minute. After exercise the pulse-rate was 24 during the first succeeding half-minute.

The patient was kept for a week in bed and suitably fed. The heart became somewhat dilated, but the pulse-rate remained the same. Then iodide of potassium was administered, 10 grains t.i.d., increasing 1 grain every day. The pulse-rate then rose to 55–60 at rest; after walking, to 72. The

patient's condition much improved, and after a month he left the hospital feeling quite well. The heart was then of normal size. The tracings showed that the patient had incomplete heart-block with a 3-1 rhythm (auricular rate 112, ventricular 36-38). The *a-c* interval was never more than ⅕ second. This, however, we regard as no absolute index of the conductivity of the bundle at the times when the ventricular systole failed to occur, and consequently do not feel justified in regarding the condition as being due to diminished excitability or contractility of the ventricular muscle. Later the auricular rate was halved, and a ventricular systole followed each auricular contraction. Under atropine (1/50 grain subcutaneously) the pulse registered 90-100. To save space these tracings are not inserted.

CASE III.

A. H., male, aged 27, a labourer by occupation, was admitted into the German Hospital in June, 1904, complaining of dizzy spells and breathlessness. His family history was irrelevant.

As a child he had developed whooping-cough, measles, and mumps. He denied all venereal infection, drank but little, and neither chewed nor smoked.

Up to four years before he felt quite well. His powers of endurance were good, and his field of cardiac response wide. Then one day, whilst ploughing, he strained to lift his plough from a rut. He suddenly felt pain in the precordium, and his heart "just jumped." He did not fall, but at once went home feeling faint. Since then his field of cardiac response has been much narrowed. A year ago he began to be afflicted with frequent attacks of dizziness, and during the last few months he has had many fainting spells, in which he frequently lost consciousness.

Status.—All the systems, with the exception of the cardio-vascular, were healthy. The heart was greatly enlarged. A soft systolic murmur was present at the apex and was transmitted into the axilla. Toward the base the murmur became rougher and louder, was more pronounced over the pulmonary region, and was not transmitted into the neck vessels. The heart-beat was twenty to the minute. The second pulmonic sound was louder than the second aortic. The peripheral arteries did not pulsate unduly. The jugular veins were not distended, and no abnormal pulsations were noted in the neck. The radial pulse was twenty to the minute, regular in rhythm, moderate in size and tension, equal on both sides. The walls were not thickened.

Whilst in hospital he had many seizures, these varying much in severity. An aura as of something "ascending from the abdomen and cutting off his wind" preceded these attacks. He would then lift his finger to warn us that an attack was coming, and the seizures uniformly followed. The patient finally died during a seizure. No necropsy.

The attacks were mostly pseudo-apoplectic or epileptiform in character and accompanied by rotation of head and eyes to the right. The longest duration of absence of pulse was one minute forty seconds. During the longer seizures involuntary movement of rectum and bladder frequently happened. On recovery from the seizures four pulse beats about four seconds apart would occur, then the pulse suddenly became very rapid, the pulse exhibiting the staircase effect, then systoles would drop out irregularly, the pulse falling to 50-60 per minute, and later to 20.

We briefly refer to this case, notwithstanding the loss of our detailed record and tracings during the fire, because of the age of the patient (27) and the peculiar behaviour of the pulse following an attack. We clinically regard it as a case of myocarditis due to causes unknown, in which dilatation of the heart had suddenly occurred whilst he was lifting a plough, the degenerative process having either before that time or later involved the bundle of His.

CASE IV.

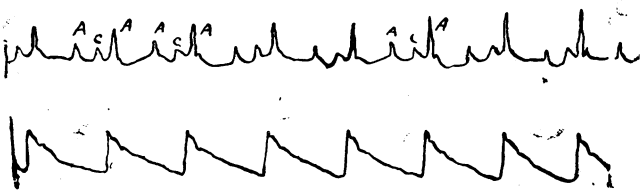
Mr. D., aged 68, a stockholder by profession, was seen by one of us in consultation because of the happening of peculiar cerebral phenomena following an operation for cataract. He became unconscious and comatose, and seemed as though he would die. Artificial respiration had been performed, greatly to the annoyance of the patient, who, having passed through many such unconscious spells, strongly objected to the disturbing artificial respiratory movements.

He had had gonorrhoea and syphilis; had used alcohol moderately; smoked but did not chew.

Nine years previously he had a slight fainting spell, and such attacks had recurred at intervals for the next five years. Four years ago they began to happen more frequently, twenty-five sometimes occurring in a week. Of late these seizures had become of a graver type, they, however, occurring at irregular intervals. His field of cardiac response had been narrow for some years.

Status.—The liver is congested and large. With that exception all the systems other than the cardio-vascular are healthy. Cardio-vascular: The heart is much enlarged, being 19 cm. from right border to apex. The precordium heaves at each ventricular systole. Between the main beats little ones are occasionally appreciable below and outside the nipple. A loud soft systolic murmur is audible at all areas and is transmitted in all directions. The second sound is loud at all areas. Following it one can sometimes hear faint tones like faint echoes, these being best heard at the apex. The ventricular rate is 28 in all postures. The jugular veins are not distended but distinctly pulsate, there being apparently two vein between successive carotid pulsations. The radial pulse is 28 to the minute, regular in rhythm, of high systolic tension, equal on both sides, the walls are fibrously thickened. In severe attacks the pulse ceases to beat, the patient breathes stertorously for ten seconds, then breathing stops, his tongue is thrust between his teeth, is bitten and bleeds, the head is jerked over to the right; the eyes open, the corneal reflex is absent, the face is quite blue. Then, fifty seconds after the breathing ceased, a peculiar "chortling" breath is taken which leads one to think that he is breathing his last, the face becomes bluer, the hands and feet tremble; after another fifty seconds of apnoea another choking breath is taken and the face becomes blue-black. Then two and a half minutes after the onset of the period of pulse absence, one loud stertorous respiration occurs, a slight wave is felt in the pulse, ordinary breathing is resumed, and the patient awakes. During the seizure involuntary movements of bladder and bowels occur. For fifteen seconds the pulse is very rapid, then it suddenly drops to 28 Many similar seizures were noted, and to those unfamiliar with the previous attacks it seemed that the one witnessed must inevitably be the last. Nevertheless he gradually improved, was able later to leave his bed, and for the last two years has been able to get about, having had no attack. The systolic blood pressure = 200 mm. J.Hg. The diastolic (?) 150 (Stanton).

A tracing (No. 2, slow speed; time interval, one-fifth second) of this case is appended. Two auricular con-



Tracing 2.—Simultaneous record of jugular and radial pulses. Note the alternation of the height of the auricular waves. This is due to the auricular systole taking place every other beat during the period of closure of the auriculo-ventricular valves. The block was a complete one. No v waves are evident, which is not uncommon when auricles and ventricles contract synchronously. Time beat ⅕ sec. Tracing 3, which showed three auricular systoles at one place between two successive c waves is purposely omitted.

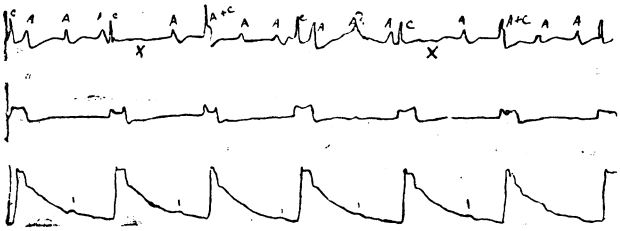
tractions are seen between two successive c waves. One occurs always during the period of closure of the auriculo-ventricular valves and therefore is always a high wave. The time interval between the beginning of this high elevation and the succeeding auricular contraction is constantly greater than the time interval following in the proportion of 4⅘ to 4⅕, and this though we would expect the auricle to fill quicker after the big wave. The rhythm in this tracing is 2 to 1, but we are dealing with a case of complete heart-block. This was suggested clinically by the constancy of the pulse-rate in all postures, and by taking many tracings we obtained curves on which c and the following A became very close together, three auricular contractions then occurring between two successive c waves. We were not fortunate enough to obtain tracings of the rapid pulse immediately supervening on an attack.

CASE V.

H., a shoemaker, aged 61, was seen by one of us three years ago. He was a big, powerful-looking man, with an excellent family history. He himself had been exceptionally healthy, had never acquired any venereal disease, drank but moderately, and never used tobacco. His seizures had begun as "dizzy spells" a year and a half previously. During these spells he said he reeled like a drunken man. Later, "fainting fits" supervened, and whilst bending at work he would tumble over "just as though he had been shot" He was taken to hospital, and remained in bed for six months. During that time the attacks were very numerous, twenty sometimes occurring in a day. When they occurred whilst he was lying in bed they frequently were preceded by a peculiar sensation travelling up from the middle of his neck, and accompanied by ringing in the ears. He thought he could sometimes avert the threatened seizures by widely opening his mouth and taking a deep breath. The more severe attacks were pseudo-apoplectic in nature, and not infrequently he passed his discharges during the attack. After six months' rest in bed he began to improve. The attacks became less severe and less frequent. He gradually was able to get about, and for the last two years he has led a useful life, though he still has attacks of dizziness from time to time, particularly, he thinks, if he becomes excited, or if his digestion be faulty.

Status during this Period of Activity.—All his systems are healthy except the cardio-vascular. The heart is much enlarged, the whole precordium heaving at each systole. Following the apex beat is a second slighter tap, apparently synchronous with the second sound. No murmurs are present. The sounds as heard over the base are muffled and faint. The ventricular rate is 28. The systolic blood pressure equals 200-210 mm. Hg, the diastolic 160-170.

The pulse-rate is 28 to the minute, regular in rhythm; the left radial is stronger than the right, but both occur simultaneously. The walls of all the palpable arteries are thickened. The tracing (No. 4), which was taken when the patient was



Tracing 4.—Vein above, apex in the middle, radial artery below. Note the complete block and the dropping out of the auricular systoles at X, and the little waves in the radial pulse which are due to the auricular systoles.

able to get about, shows a complete block. Sometimes the A and C waves coincide, sometimes A occurs independently but during the closure of the auriculo-ventricular valves. The auricular rhythm is itself slightly irregular, and it will be seen that at two places an auricular contraction has dropped out. On the theory that the stimulus which sets the auricular pace arises in the region of the mouths of the great veins (by no means proven), such a phlebogram is extremely suggestive of a block between sinus and auricle at the so-called sino-auricular junction. It is true that the time relationships do not exactly correspond with those instances in which Wenckebach so astutely reasoned out the existence of delays of conduction, but it is probable that many departures from the typical occur. On such a hypothesis Fig. 1 is constructed.

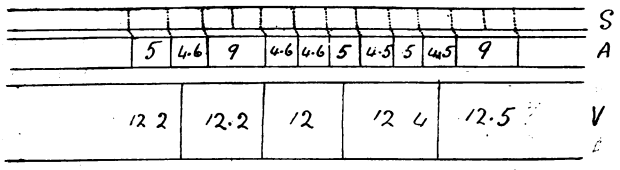


Fig. 1.—The dotted straight lines S represent the imaginary sinus waves; A the auricle waves; V the ventricular contractions. The space between A and V represents the A–V bridge, which is blocked. The space between S and A represents the imagined conduction from sinus to auricle, which fails on two occasions.

CASE VI.

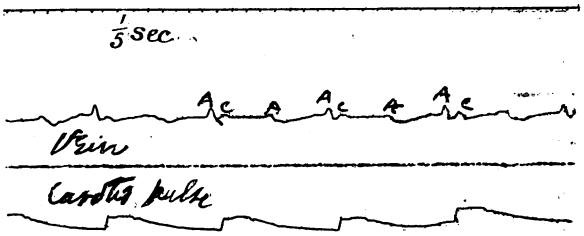
The patient, a carpenter by trade, was admitted into hospital July 4th, 1906. He was 58 years old, and came of a family whose tenure of life was exceptionally long. He himself had enjoyed excellent health. He drank little, but was an inveterate tobacco chewer. He had never contracted any venereal disease, and gave no history of any infections.

Two years previously, whilst working, he had a slight fainting spell. Since that time his field of cardiac response had been considerably narrowed. During the last few weeks he had fainted on several occasions. He would lose consciousness and often fall, and he bore on his forehead the traces of bruises so acquired. He thought these fainting seizures were of only momentary duration. He felt quite well between the attacks, and had continued to follow his occupation.

Examination showed a tall, strongly built, ruddy, healthy looking man, and the only abnormalities apart from his cardio-vascular system detected were:

1. The left pupil was somewhat sluggish in its reaction to light.
2. The tongue was unduly fissured.
3. The liver was enlarged, and the spleen was palpable ½ in. below the left thoracic margin.
4. A few râles were audible in the region of the angle of the left scapula (these, however, soon disappearing).

Cardio-vascular System.—The heart was enlarged, the right border being ½ in. to the right of the right sternal edge. The apex was sometimes in the fifth, sometimes in the sixth interspace 1 in. outside the left mid-clavicular line. The impulse was diffuse, slow, and heaving, and the most noteworthy fact concerning it was its apparent rapid shifting independent of respiration or of change in posture. A loud, long, blowing systolic bruit was audible, most pronounced at the mitral area, and carried for some distance towards the left axilla. The aortic and pulmonic second sounds were faint, muffled, and lacked sharpness. Occasionally faint sounds were audible during diastole over the base of the heart. The heart-rate was 36 to the minute and apparently regular in rhythm. The neck veins were not distended, but pulsated at a more rapid rate than the carotids; apparently about two to one. All the peripheral arteries were thickened and pulsated unduly. The radial pulses were equal; 36 to the minute, of regular rhythm. A capillary pulse was present. When standing the pulse was 42 to the minute; after exercise it registered during the first succeeding half-minute 24 beats. Three months later it was 42 in all postures, and the injection of $\frac{1}{100}$ grain of atropine had no influence upon the rhythm; the tracings still continued to show a partial heart-block with a 2–1 ratio (vagus influence at a minimum).



Tracing 5.—Vein above, carotid below. Note 2-1 rhythm, incomplete block and normal length of the A-C period. (Time beat ⅕ second.)

No. 5 is a tracing taken at this time. The auricular waves are marked A, the carotid peak C. The interval A–C is never more than one-fifth of a second, and is generally a little less. It is possible that the dropping out of the alternate systoles is due to a diminution of the ventricular excitability, but this is regarded as undetermined (see Case II). Though no history of luetic infection was obtainable, the unilateral pupillary sluggishness, the fissured tongue, and the enlarged liver and spleen suggested antisyphilitic treatment, but this had no influence on the pulse rhythm.

One year later the patient again came under our observation, complaining of dyspnœa. He had had many seizures of the nature of fainting spells. His respiration was of the Cheyne-Stokes type without actual pauses.

Examination revealed bronchitis. The heart apex at this time was double, a very distinct second tap following the first one, occurring internal to the first and suggesting to the finger a rolling movement.

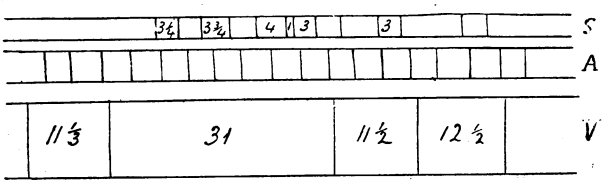
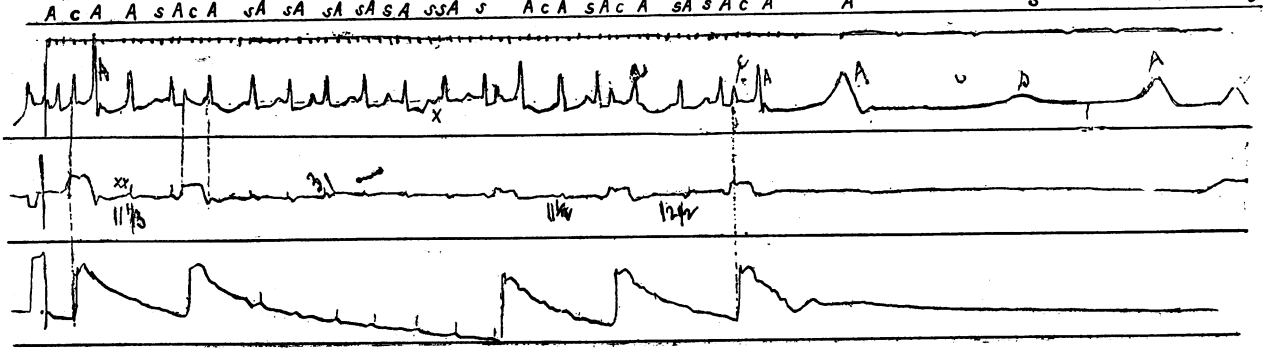
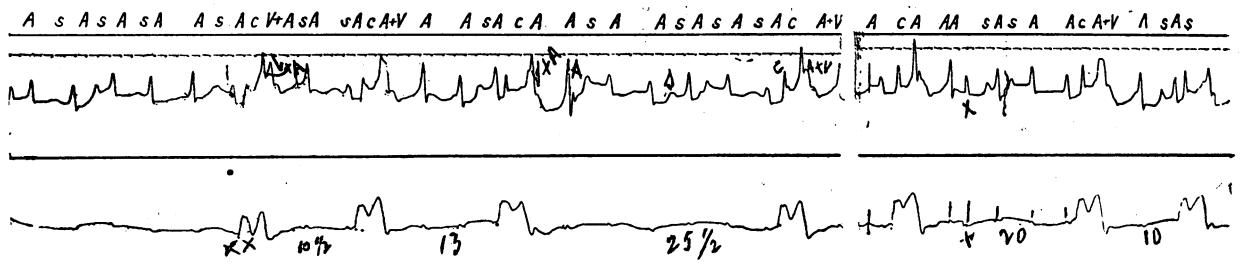


Fig. 2 is a diagrammatic representation of Tracing 6. S equals the "so-called sinus" waves, which are evidently arrhythmic; A equals the auricular systoles, V the ventricular. The clear space between represents the A–V bridge through which no waves are conducted.

Tracing No. 6 was taken at this time, and includes the period of one of his fainting spells. It presents many interesting features. Analysing first the venous pulse, we note



Tracing 6.—Note the S waves, the absence of the ventricular beat during an attack, the continued auricular pulsation evident in vein, apex, and radial curves. Time ½ second (Cf. Fig. 2).



Tracing 8 of vein above, apex below. Note the s waves, the cessation of the ventricular action during slight attacks, the continued auricular pulsation, and the auricular extra systole at x. Time beat ⅕ second.

that the second auricular contraction occurred during the closure of the auriculo-ventricular valves. The resultant wave is a very high one. The auricular contractions are unequally spaced, there being present a slight auricular arrhythmia. The auricles continue to contract during the attack.

Equally interesting are the little elevations which are evident between the auricular systoles (the s waves of Gibson and Ritchie). As their occurrence in auriculo-ventricular heart-block is concomitant rather than essential, we will not here discuss the cause of their origin, but will simply point out concerning them these facts, which can be readily gleaned from a perusal of the various tracings:

(*a*) They present a curious serial relationship to the auricular systoles, this relationship, however, apparently depending upon the preceding ventricular contraction. This is always the case in many tracings that we have.

(*b*) They rise well above Wenckebach's niveau line.

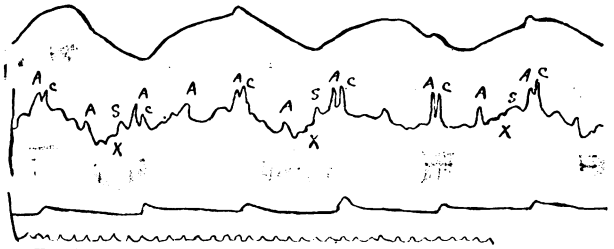
(*c*) They are arrhythmic, and sometimes multiple between successive auricular systoles (see Tracing 6 at x, Fig. 2, Tracing 7A at x x x).

(*d*) They are at places separated from the succeeding auricular wave by the comparatively big time interval of two-fifths of a second, but no auricular systole drops out.

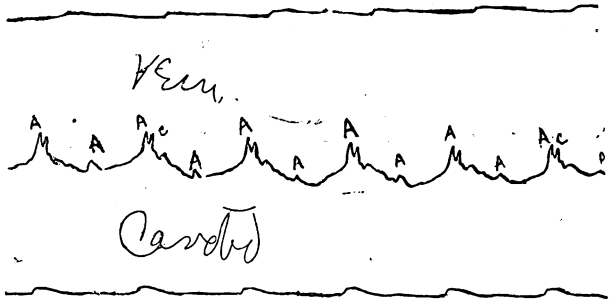
(*e*) In Tracing 7A they can be seen to bear no special relationship to any respiratory phase; in this tracing, at x x x, multiple undulations are evident. In Tracing 7B, taken with the breath held in deep expiration, they are hardly recognizable, and the same occurred with the breath held in deep inspiration.

(*f*) In Tracing 11, at x x, the c elevation is superimposed upon these little s waves.

In the cardiogram (Tracing 6) little depressions catch the eye between the ventricular systoles. Careful inspection, however, reveals at xx a small elevation preceding the depression, and it is this that corresponds to the

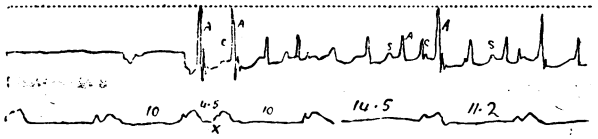


Tracing 7A.—Respiratory curve above, pneumograph over manubrium. Note the occasional little elevations due to the aorta (?) beneath. Vein in the middle, A = auricular, C the carotid waves. Note the s waves, the short A–C interval, the undulations between successive A waves, particularly at x.



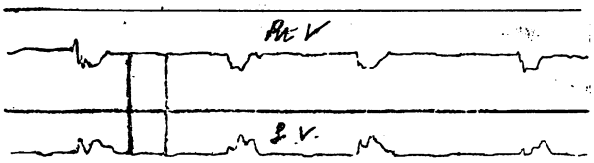
Tracing 7B is a continuation of 7A, the breath being held in deep expiration, consequently there is no respiratory curve in the pneumograph, but the waves spoken of in 7A are more distinct. The so-called s waves have disappeared or become so small as to be unrecognizable.

auricular systole, the depression representing the recession of the chest wall following the real wave. This is better seen in Tracing 8. If the ventricular interspaces be measured, it will be noted that the long silences form an approximate multiple of the shorter ones in their immediate neighbourhood, thus suggesting that the stimulus continues to be rhythmically formed throughout the attacks, but owing to faulty excitability, contractility, or conductivity of the ventricular muscle, no systole results. In Tracing 9 a ventricular extra-systole is present whose period compares exactly with the preceding full ventricular period. If the single ventricular periods be measured in Tracings 6, 7, 8, 9, it will be found that they vary from ten fifths of a second to fourteen and a half. Such a difference is unusually wide, and to maintain the



Tracing No. 9.—Vein and apex beat. Note the ventricular extra-systole at x. Time beat ⅕ of a second.

theory of rhythmical production of stimuli we must assume that in the longer period extra-systoles have dropped out—for example, in the fourteen and a half fifths period an extra-systole of the same period length as the extra-systole period in the tracing. In the tracing of the radial pulse (No. 6) little elevations marked with an upstroke represent the auricular systoles, they presenting the same time relationship as the auricular venous waves. At x in Tracing 8 an auricular extra-systole is to be seen both in the vein and apex curves. In Tracing 10 cardiograms of the right and left heart are



Tracing No. 10.—Tracing from right heart above, from left below. The ventricles contract synchronously, also the auricles.

shown. In that of the left heart little auricular negative waves correspond to the positive elevations in the curve of the right heart. Evidently the receiver was not over the part of the left ventricle which the left auricular systole projected against the chest wall, but in its immediate neighbourhood, hence the depressions instead of the usual elevations. In this compound cardiogram, then, we have evidence of the working of all four chambers of the heart.

With rest in bed and simple food, the patient's condition improved. The block became incomplete with at times a 3–1, at others a 4–1 rhythm. A tracing taken during a slight attack at this time showed ten auricular systoles occurring between two ventricular contractions. Then a regular 3–1 rhythm developed (A–C interval not more than one-fifth of a second). Further improvement led to a 2–1 rhythm, the A–C time being slightly under one-fifth of a second (a reversion to the rhythm existing fifteen months previously). In this condition he left the hospital three months after his admission. Three months later he was readmitted to the surgical side, for an injury to his hip, due to a fall from a ladder during a fainting spell. The block was still incomplete, the rhythm 2–1.

In briefly reviewing these cases clinically we note that in two there was a history of syphilis, in one other the clinical findings raised suspicion, in a fourth case improvement occurred while the patient was taking potassium iodide. In one of the cases, however, which gave a syphilitic history (Case I) this disease seems to have had

nothing to do with the onset of the condition. All six patients were males.

Three patients were comparatively young, 27, 30, 31 respectively, and two of these died, the third being in the pre-paroxysmal stage and recovering. The other three patients are still alive and are apparently as well as when they first came under our observation. One of these had his first seizure eleven years ago. All came complaining of the attacks. The dizzy spells complained of did not consist of a rotation of the patient, or of objects relative to the patient, but seemed to be of the nature of semi-faints, the patient not quite losing consciousness. One individual, however, stated that he reeled like a drunken man.

One patient frequently experienced an aura, which always, when tested, did precede the attack. A second patient sometimes felt an aura, but only when lying down, and he believed that he could sometimes prevent the attack by opening his mouth and taking a deep breath—a manœuvre which we have known patients exhibiting extra-systoles perform with a similar intent.

We would suggest that these pre-seizure sensations may be due:

1. To extra systoles which, coincidently in some cases, in others perhaps regularly, precede the seizure, the vague description given by some patients of extra-systoles being not unlike the so-called "aurae" in Adams-Stokes's disease.

2. To slight, short seizures preceding more prolonged ones.

3. To the pumping in of blood into comparatively empty blood vessels after an extra-systole has failed to open the aortic valves; this, for instance, causing an additional pulsation in some of the vessels. Unfortunately, we have no tracing of the period of the sensations.

The attacks seemed often to occur in spells, and we have come to regard such clinically as an indication of the temporary breaking-down of the ventricular compensation. Such would naturally be more apt to occur in acutely-developing cases, and at periods in the chronic ones when the rhythm changed in an incomplete block, or, when the block became complete, the ventricle, with its commonly diseased musculature, needing time to adjust itself to the new condition of things, and it seems possible that the adoption of certain attitudes or breathing exercises might at certain times aid in a temporary adjustment, and so be the foundation of some patients' belief that they can avert attacks.

It is truly remarkable from what seizures—or, indeed, series of seizures—people can recover and afterwards live a useful life for years. Thus, one of our patients during an attack in which the pulse was absent two and a half minutes exhibited periods of apnoea of fifty seconds each, he being at these times to all appearances dead. Such attacks occurred repeatedly, yet he improved, and has not had a seizure for two years, he in the meantime being able to climb stairs, etc.

In the shorter attacks the veins in the neck can be seen to pulsate, as, indeed, our tracings show. In the longer paroxysms in our patients pulsation could no longer be detected on the swollen veins, nor could any auricular sounds be heard during such long attacks, not even in Case 1, in which they were commonly distinctly audible. Still it would seem that the auricles must continue to contract, otherwise what sustains life in a person so long pulseless and apnoeic?

The sounds as heard over the auricles seemed to us as shortened miniature toneless imitation of the normal first and second sounds as commonly heard at the heart apex; in other instances only one sound was audible corresponding to each contraction. The seat of greatest intensity of these sounds varied, being generally in the second and third interspaces, a little out from the sternum on each side, occasionally, however, at the heart apex.

In no case was there any oedema, and, strange to say, the neck veins were never distended except during an attack. In fact, frequently no intercarotid pulsation could be detected in two or three of these patients as long as they lay quiet in bed, and it was necessary to enjoin some muscular exercise in order to demonstrate their existence to onlookers.

Three different modes of behaviour of the pulse immediately following an attack were noted in our cases.

In one patient four beats, four seconds apart, would uniformly occur, then the pulse would become rapid, and the tracing invariably showed the staircase effect; then systoles would drop out occasionally, then more frequently, and finally the pulse-rate became 20 per minute (tracings burnt in the fire). We have regarded this as an instance of a clinical reproduction, immediately following a long rest, of the experimental results obtained by Erlanger.

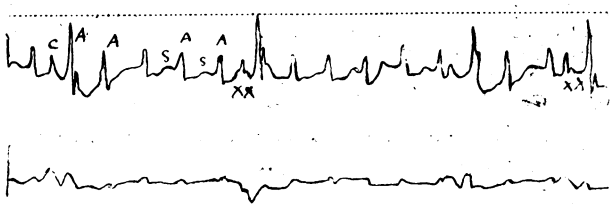
In another patient the pulse was very rapid immediately following a seizure, and then after fifteen seconds it suddenly dropped to its present infrequent rate. In view of Wenckebach's work, it seems probable that such was due to extra-systoles.

In the other cases approximately the same ventricular rate immediately followed as preceded the attacks.

In all the attacks witnesssed the pulse ceased before the seizure began, and returned after it had ended.

One patient—a thick-chested man—was examined with the fluoroscope; the result was unsatisfactory, and the information in no way compares, in the writers' opinion, with that given by multiple tracings.

Clinically, the hearts of all these patients, with the exception of the individual who developed the disease acutely, and the man who had, apparently for just a week, been in the pre-paroxysmal stage, were greatly dilated and hypertrophied. The heart examined *post mortem* (first case) was of normal size, but during life it gave every evidence of dilatation. Quite worthy of note in patient No. 6 was the shifting of the position of the apparent apex independent of movement and respiration. Thus we had to move our receiver between the fifth, sixth, and seventh interspaces at quite short intervals. This is suggested in Tracing 11, where at one period the movement of the right ventricle is recorded instead of the left We would suggest that this shifting was due to the varying quantity of blood driven into the ventricle by the varying number of interventricular auricular systoles. We have alluded to the palpable double apex beat. At x x in Tracing 8 the M shaped cardiogram shows the instrumental record of this. Tracing 10 demonstrates that it was not due to asynchronous contraction of the right and left ventricles. From this, and the peculiar rolling palpatory sensation, we ascribed the second tap to the rotatory movement of the heart.



Tracing No. 11.—Note the combination of the S and C waves at x x, and the cardiogram, in which the second ventricular wave is evidently due to the right ventricle.

In three of our patients the systolic blood pressure was high—170, 200, 210 mm. Hg; the pressure corresponding to the lower part of the greatest oscillation of the mercury column, as judged by the eye, was in each case about 50 mm. less. In these three cases the peripheral arterial walls were thickened. Notwithstanding this high pressure the second aortic sound in two of these cases was muffled, faint, and dull, though no diastolic murmur could at any time anywhere be detected. One of these patients had a distinct capillary pulse. It seemed to us that the arterial capillary area was fed so infrequently, whilst it was being constantly drained, that when the systole happened, the free passage of blood would diminish the recoil upon the semilunar valves, thus accounting for the faintness of the second sound, the same phenomenon being responsible for the capillary pulse.

In five of these patients the systems other than the cardio-vascular were but little affected, the blood in Hb. and cell-content was normal, and in all drugs, with the possible exception of potassium iodide in one case, were without apparent salutary influence. Rest in bed, however, seemed a great factor in their restoration to a useful life.

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[3] *Practitioner*, May, 1907.