Two cases of acute endocarditis / by John Cowan [and others].

TWO CASES OF ACUTE ENDOCARDITIS

By JOHN COWAN, A. M. KENNEDY, A. R. PATERSON, and JOHN H. TEACHER.

With Plates 2 and 3

In a recent number of this Journal,[1] two of us reported a case of partial heart-block, occurring during an attack of acute rheumatism and persisting for a fortnight; and we suggested that the condition might be due to the involvement of the *a.–v.* bundle in one of those little inflammatory lesions which are so frequently present in the myocardium in the acute infections. The favourable issue of our case precluded confirmation of our theory, but we have again met with cases where it seemed probable that the *a.–v.* bundle or node was implicated in the way which we had suggested, and in two of them we were able to secure a microscopic examination of the parts.

Our first case was a man aged 26 years, who was admitted into hospital on June 5, 1909, complaining of weakness and of breathlessness on exertion of some months' duration. He stated that he had not been in good health since an attack of enteric fever in 1903 for which he was in hospital in Japan for about three months. He never regained his former health, and his feet were often swollen at night, but he was able to work as a clerk until July, 1908, when he was thrown out of employment by the failure of his firm. He stayed at home in fairly comfortable circumstances until he procured a new situation on February 19, 1909. In the early part of February he had caught cold and he was feeling 'run down' when he commenced his new work. This entailed a daily walk of four miles, and he felt exhausted at night, and also during the day when walking uphill, but though he steadily became weaker he continued at his post until six weeks before admission, when he was compelled to go to bed. His cough, accompanied by a slight mucoid spit, persisted from February until May. He had frequent attacks of shivering at night after getting home; they were fairly severe, and lasted for fifteen to twenty minutes, and he felt hot after he got into bed, and often sweated profusely in the early morning. About a fortnight before admission his right forearm and elbow became sore to the touch and on movement, and this continued for a few days. Four days before admission the left hip and ankle became painful, and this still persisted.

His family history was good. He had always been healthy prior to 1903, but he admitted on cross-examination that he had suffered from growing pains and an attack of tonsillitis in boyhood. He had also had an attack of gonorrhoea early in 1903. On admission he was found to be a thin, pale, badly nourished man, with flabby muscles and little subcutaneous fat. The left hip and ankle were painful and tender, and there was a small

[1] 1910, iii. 115.

tender area of induration on the back of the left forearm. The right calf was, he said, painful, but it seemed normal on examination. There were several minute ecchymoses on the conjunctival surface of the right lower eyelid, and a fairly large one on the conjunctiva of the left bulb, while ophthalmoscopic examination revealed a medium-sized flame-shaped haemorrhage on the course of the left superior temporal vein (A. J. Ballantyne). The lungs were normal, the tongue was clean and digestion good; the liver and spleen were slightly enlarged; the urine contained a small amount of albumen. The heart was but little enlarged, though there was a well-marked mitral systolic murmur. The pulse was frequent, regular, small, and soft; and he was slightly fevered at night.

Progress after admission was slowly but continuously in the wrong direction. Many embolisms occurred, chiefly in the limbs, but once at any rate in the spleen, and twice in the kidneys. He had several 'faint turns' of a few minutes' duration without obvious cause. The weakness increased, and after the middle of July oedema was more or less continually present in the legs. There were occasional rigors, and generally profuse sweats at night. On August 25 a left hemiplegia occurred, and he died on August 29.

Post-mortem examination revealed an acute endocarditis of the mitral valve. Two emboli were present in the right middle cerebral arteries, and septic infarctions were present in the left kidney and the spleen; from the latter the pneumococcus and B. coli communis were isolated on culture. Blood cultures during life had proved sterile.

The mitral valve was dilated. Numerous vegetations were present on the cusps, some small and wart-like, others long and luxuriant. The endocardium of the left auricle was thick and opaque, and a few minute granulations were present on the contra-fossal wall, immediately above the valve: the endocardium at this point was notably thickened. There was no evidence of mural implication elsewhere.

A block of tissue containing the auriculo-ventricular bundle and node was embedded in paraffin and cut in serial sections on a plane at right angles to the long axis of the heart (A. M. K.). Microscopic examination revealed well-marked congestion of both node and bundle. The node was otherwise normal, though two small foci of round cells were found in the auricular muscle in its immediate vicinity. The bundle was involved in three separate places by foci of the same kind. The first was of small size, and was situated immediately below the node; the second, which was considerably larger, was found a little lower down, between the auricular muscle and the bundle, and implicated both; the third, of fair size, occupied the central part of the bundle, and occurred immediately above the point where it passed through the central fibrous body. The cells in the foci were all mononuclear, and the majority were evidently lymphocytes. A few larger cells with a fair amount of protoplasm were also present, and were probably of connective tissue origin. The fibres of the *a.–v.* bundle seemed normal, save in the immediate vicinity of the two larger foci, where they were evidently degenerate. The fibrous tissue of the bundle was not excessive in amount. The cardiac muscle generally showed evidence of interstitial myocarditis in an early stage, and the muscle fibres in some of the patches were degenerate.

The pulse during the patient's residence in hospital was always regular, until immediately before death; and the venous curves were normal, save that

the *a–c* interval was invariably prolonged, measuring about 0·25″ and never touching 0·2″ or 0·3″.

The little lesions which are present in the bundle seem sufficient to account for the trifling delay in the conduction of the stimulus from auricle to ventricle.

The second patient was a van-driver aged 24, who was admitted into hospital on July 15, 1909. On July 1 he had had one of his right upper molars extracted on account of a gumboil from which he had suffered for a day or two. In the evening he felt sick and shivery, and probably was fevered. These symptoms continued, and on the afternoon of July 4 he had a definite rigor. On the 7th he felt better, and on the 8th he went to Ardrossan, walking from the station to the house at which he stayed (2 miles). After this he improved until the 12th, though he had a slight cough after the 6th, with discomfort in the left chest. But on the 12th he had to go to bed as the pain in the side became worse, and was much intensified on coughing. The left arm, too, was stiff, and he was very breathless and had to sit up in bed at night. On the evening of the 13th he was better, and able to sleep quietly lying down.

On July 14 he felt so well that he dressed and went downstairs to breakfast, which he did without any difficulty or assistance; after breakfast he sat down in a chair, and on attempting to rise an hour later found that his legs were powerless, and 'had no feeling' in them. He had to be carried back to bed. In the evening he felt 'pins and needles' in the right leg.

His previous health had been good, save for attacks of measles and enteric fever in childhood; but he was often troubled with gumboils, and was frequently wetted at his work. His family history was unimportant.

On admission he was found to be notably emaciated, with small muscles and scanty subcutaneous fat. His complexion was somewhat sallow, and his expression anxious, while he had to be propped up in bed on account of breathlessness. There was a large fixed antero-posterior curvature of the spine with the maximum curve about the ninth or tenth dorsal vertebra, which he stated had been present for about eight years. The respirations were both thoracic and abdominal, the expansion of the chest being poor (1″). The skin was hot and dry, and there was a little oedema of the feet, and slight pitting with the stethoscope on auscultation over the cardiac area. He was slightly fevered at night. The patient, though weak, was quite clear mentally, spoke correctly, and answered questions accurately and sharply. He had little power in his legs, but was able to pull up both knees, though he was unable to move the toes of the right foot. The movements were more free on the left side. There was considerable anaesthesia in both legs, the right leg being insensitive to touch as high up as the groin, the left half-way up the thigh: painful stimuli were appreciated correctly in the left leg above the ankle; in the right they were only felt as contacts and that after an appreciable delay. Thermal sensations were appreciated correctly in the left leg. Power was perhaps deficient to some slight extent in the left arm, but in the right was good; and sensation seemed normal. The plantar, cremasteric, and abdominal reflexes were absent, and the knee-jerks could not be elicited. The epigastric reflexes were present and equal. The flexor jerks in the arms were present and equal, and the jaw-jerk was present. The right pupil was slightly smaller than the left; both reacted to light and on accommodation.

The patient had control over his bladder, and testicular sensation was normal. The tongue was dry and coated, many of the teeth were absent, and most of the remainder were carious, and the mouth generally was very foul. The pulse was soft, regular and infrequent. Palpation of the apex beat, which was full and widespread, showed that every other beat was missed at the radial, the cardiac action being regularly coupled. A systolic murmur was audible all over the cardiac area. It was heard best at the apex, where it was long and

soft, replacing and running out of the first sound; the second sound was not audible here. The respirations were difficult and cyclic, but without any full apnoea. The upper part of the right chest was dull on percussion, and the respiratory murmur here was deficient, with prolongation of expiration. In the left lower axilla friction sounds were audible; the stools contained a little mucus. The urine was concentrated and dark in colour, and contained a trace of albumin.

For a few days his general condition improved slightly. The coupled rhythm ceased on the 16th, and he rested fairly well at night. The sphincter ani was flaccid, and he had no control over the bowels, though micturition was normal. On the 17th movement was more free in the left leg, but the palsy below the right knee persisted. Anaesthesia was still present, but did not extend so far up on the left leg, being apparently normal above the knee. The left plantar reflex and knee-jerk had reappeared, but the right were still absent.

About midnight on the 18th he was noticed to be restless, and was found to be semi-conscious and unable to say more than 'yes', and there was a well-marked full right hemiplegia. He gradually became weaker and more comatose, and the respirations and pulse became more frequent. Râles appeared all over the lungs, and he died with cardiac failure on July 27, 1909. The pulse remained 'single' until the day of death, when it was noticed to be 'coupled' again for a short time.

Post-mortem examination revealed an acute ulcerative endocarditis of the mitral valve, engrafted on a lesion of old standing. The upper and middle lobes of the right lung were solid from a somewhat chronic pneumonia, and there were numerous infarcts in the spleen and kidneys. The left middle cerebral artery was occluded by a large pale embolus which resembled the vegetations on the mitral valve, and was associated with a very extensive softening which involved the lenticular and caudate nuclei, most of the internal capsule, the island of Reil, and a large portion of the central convolutions; the rest of the brain and the spinal cord seemed normal. Microscopic examination of the cord showed no evidences of local damage (J. H. T.). Streptococci were isolated on culture from the spleen. No blood cultures were made during life.

The mitral valve was stenosed, the cusps being notably shortened, the chordae tendineae short and thick, and the tips of the papillary muscles fibrous. There was well-marked evidence of fibrosis spreading upwards from the bases of the cusps in the direction of the central fibrous body of the heart. Enormous soft recent vegetations almost occluded the valve, and there was in addition extensive ulceration and perforation of the anterior mitral cusp. There was no evidence of any acute mural endocarditis; but the endocardium of the left auricle was thick and opaque, with a special area of thickening about as large as a shilling on the contra-fossal wall just above the valve.

A block of tissue containing the auriculo-ventricular bundle and the node was embedded in paraffin, and cut in serial sections on a plane at right angles to the longitudinal axis of the heart (A. M. K.). Microscopic examination revealed considerable congestion of the tissues generally. The node was the site of a profound inflammatory disturbance, many focal collections of round cells being present, as well as, in places, a diffuse infiltration. The bulk of the cells were lymphocytes, but a few large mononuclear cells, both round and spindle-shaped, were also present. The fibres of the *a.-v.* node were probably normal, and there was no evidence of old-standing fibrosis. The walls of a microscopic artery (Plate 3, Fig. 2) were infiltrated with round cells, though the lumen was patent; the other vessels seemed normal. The intensity of the lesions varied at different levels, but the whole of the node was more or less affected. The bundle, on the

other hand, was not implicated, save at one place immediately below the node, where one or two small foci in the auricular muscle immediately adjoining it passed in for a short distance between a few of its fibres.

There was a well-marked myocarditis, involving both ventricle and auricle, but it was only extreme at the base of the mitral valve, and in the auricular sections immediately above the node itself.

The tracings which were obtained in our second case differ from those in our first. The early tracings are somewhat difficult to interpret and are discussed in detail elsewhere.[1] In the last tracing which we obtained, with a 'single' rhythm, the *a–c* interval is apparently very short, measuring little more than 0·05″.

During the *a–c* interval five events occur: (1) the auricular systole; (2) the passage of the stimulus from auricle to ventricle; (3) the latent period; (4) the period of ventricular systole prior to the opening of the aortic valves; (5) the transmission of the aortic wave to the carotid artery. The normal *a-c* interval measures about 0·2″, being somewhat shorter in a quickly beating heart, the auricular systole lasting for nearly 0·1″, and the period between the commencement of the systolic rise on the apical tracing and the carotid wave in the jugular pulse measuring about 0·05″. An *a–c* interval of 0·1″ is almost certainly, and an interval of 0·06″ is surely, too short to permit the normal sequence of events, and it seems probable that the shortness of the interval is due to the more or less synchronous contraction of auricle and ventricle. A single large wave not infrequently occurs in the jugular curve synchronous with the occurrence of the carotid wave (the 'nodal' extra-systole of Mackenzie), and is usually considered to be the result of auricular contraction at a time when the auriculo-ventricular valves are shut. From our previous argument it is evident that in Mackenzie's 'nodal' extra-systole the ventricle must have contracted before the auricle, for otherwise the delay in the appearance of the carotid wave would produce a carotid elevation subsequent to the auricular wave. If a period of between 0·05″ and 0·1″ elapses between the auricular and the carotid waves in the jugular curve it seems probable that the contractions commence simultaneously in both auricle and ventricle.

Conclusions of this kind have for some time been floating in the minds of those who are interested in cardiac work (Mackenzie, Hay, Ritchie), and it is extremely interesting to find that in our second case, where the clinical evidence is in favour of the theory that the auricular and the ventricular contractions commenced simultaneously, the pathological lesion is almost wholly confined to the *a.–v.* node, and the stimulus probably originated at this level. But we must not forget that our theory makes a lesion in the *a.–v.* bundle in one case responsible for a defect in function, and a lesion in the *a.–v.* node in our second case a cause of increased irritability.

We can find but few records in the literature of systematic examination of the 'primitive tissue' of the heart. Mackenzie, however, reports two cases

[1] Cowan and Ritchie: Coupled Rhythms of the Heart-case, below, p. 66, Case VII.

where it was involved by lesions of the kind which we have described: (1) a case of cardio-sclerosis with 'nodal' rhythm for two months before death; the *a.–v.* node and bundle were affected; (2) a mitral case, with attacks of paroxysmal tachycardia; here the sino-auricular node was involved as well as the *a.–v.* node and bundle. Schönberg reports five cases of persistent irregularity of the heart in which the sino-auricular node was affected: the *a.–v.* bundle and node were not examined. Vaquez reports a case of paroxysmal tachycardia in which the sino-auricular and *a.–v.* nodes and the *a.–v.* bundle were involved.

The sensory disturbance in the left leg raises a question of great interest, for the only lesion found in the central nervous system was confined to the left cerebral hemisphere. The sensory disturbance was noticed on July 15th, 16th, and 17th, but could not be followed after the 19th on account of the mental disturbance produced by the second embolus. The area involved was diminishing on the 17th.

It is, of course, well known that unilateral cerebral lesions produce relative weakness of the muscles on the same side as the lesion, as well as palsy on the opposite side, but there are few cases on record of bilateral sensory loss. A recent paper by G. Bergmark contains, however, four cases, two of which were observed by himself. His first case was a man aged 66, who developed a right hemiplegia on March 6. Two days later there was complete right hemianaesthesia and 'marked hypalgesia of the left lower extremity', which had disappeared three weeks later. His second case, a man aged 74, developed a left hemiplegia in September; when examined four months later, the right leg below the knee and the distal phalanges of the right finger were insensitive to cotton-wool touch. *Post-mortem* examination revealed an extensive lesion occupying almost the whole of the internal capsule, the greater part of the lenticular nucleus, and the external and lower portions of the optic thalamus. He quotes two cases, recorded by Freidrich Müller, with hypalgesia in the distal part of the lower extremity on the non-paralysed side.

The cause of the bilateral loss is somewhat obscure. Bergmark rejects the obvious theory of bilateral innervation proposed by Müller, as he found that in his first case defective sensation in the mesial parts of the trunk on the non-paralysed side disappeared rapidly, while that in the legs lasted for longer and disappeared simultaneously. He thinks that a more plausible explanation can be found by assuming a double cerebral lesion, that in the 'healthy' hemisphere being relatively slight and transient, and the result of the disturbance of the cerebral blood supply.

The rarity of bilateral sensory disturbances is probably more apparent than real, for it is but seldom that sensation can be accurately tested immediately after the occurrence of a cerebral lesion, as the disturbance of cerebral function commonly affects the intelligence of the patient and affords obvious sources of inaccuracy; and the sensory loss is often transient.

Conclusions.

Microscopic examination of the *a.-v.* node and bundle in two cases of acute endocarditis revealed well-marked inflammatory lesions in the parts. In one case, where the *a–c* interval in the jugular curve was prolonged, the lesions implicated the bundle; in the other, where the *a–c* interval was very short, the node alone was affected.

REFERENCES.

1. Bergmark, G., *Brain*, 1910, xxxii. 342.
2. Cowan, M'Leod, and Paterson, *Quart. Journ. Med.*, Oxford, 1910, iii. 115.
3. Mackenzie, James, *Diseases of the Heart*, Lond., 1908, 309, 315.
4. Schönberg, S., *Frankfurter Zeitschr. f. Pathol.*, 1908, ii. 153.
5. Vaquez, H., *Archives des Mal. du Cœur*, Paris, 1909, ii. 609.

DESCRIPTION OF FIGURES.

Case I.

PLATE 2, FIG. 1. Tracing showing a slight delay in conduction. a–$c = 0{\cdot}25''$.

FIG. 2. Diagram showing the situation of the microscopic lesions in the *a.–v.* bundle.

FIG. 3. Microphotograph of lesion no. 1. × 90. A small round-cell collection is shown in the substance of the *a.–v.* bundle (1). (2) is the central fibrous body of the heart.

FIG. 4. Microphotograph showing lesion no. 2. × 90. A round-cell collection is visible between the auricular muscle (2) and the *a.–v.* bundle (1), and infiltrates both. The *a.–v.* bundle here is congested, and its fibres are degenerate.

FIG. 5. Microphotograph showing lesion no. 3. × 90. A larger round-cell collection is shown in the centre of the *a.–v.* bundle (1). (2) = auricular muscle. The *a.–v.* bundle here is congested, and its fibres are degenerate.

Case II.

PLATE 3, FIG. 1. Tracing showing the close proximity of the *a.* and *v.* waves in the jugular pulse. a–$c = 0{\cdot}06''$.

FIGS. 2–4. Microphotographs of the *a.–v.* node at different levels, showing focal and diffuse round-cell infiltrations. The artery in Fig. 2 is *not* thrombosed: it is cut obliquely and its walls are infiltrated. All the capillaries are congested.

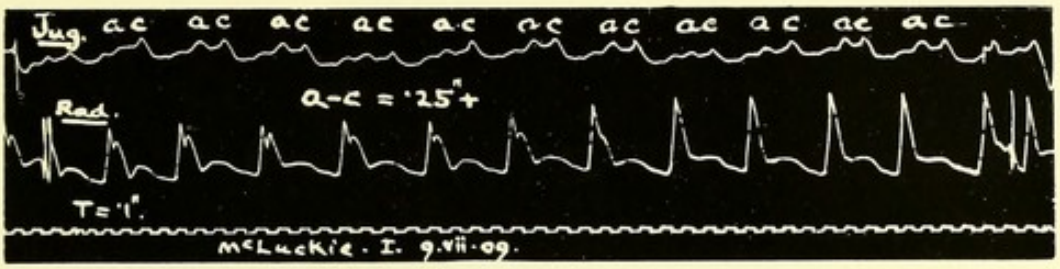

FIG. 1

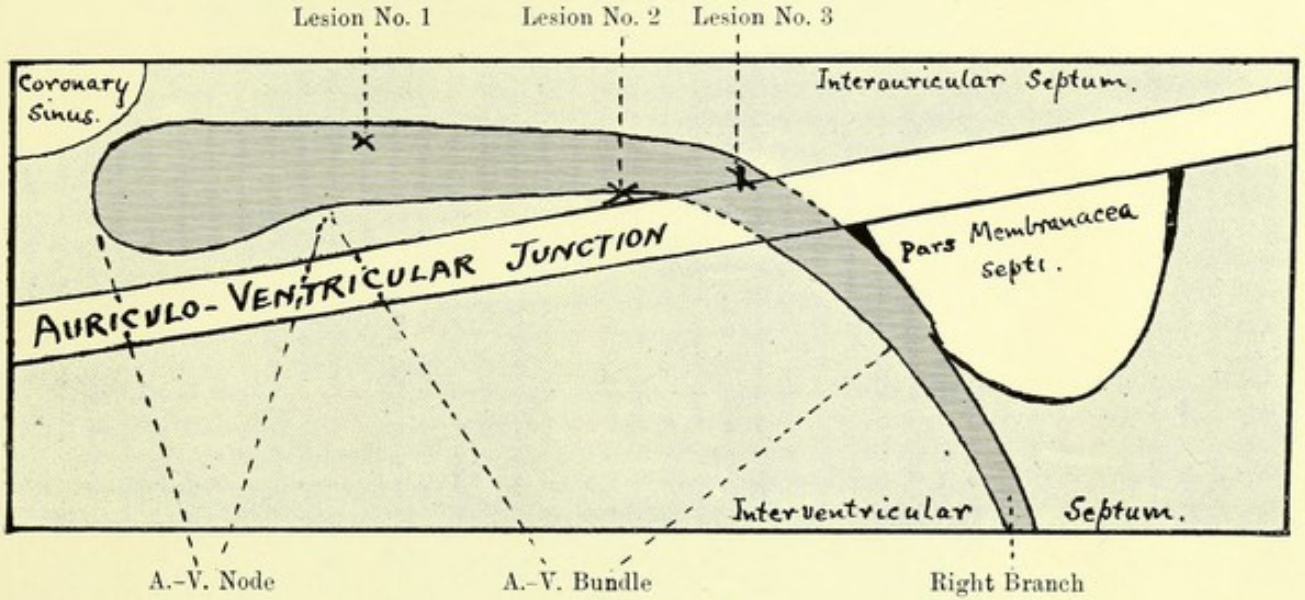

FIG.

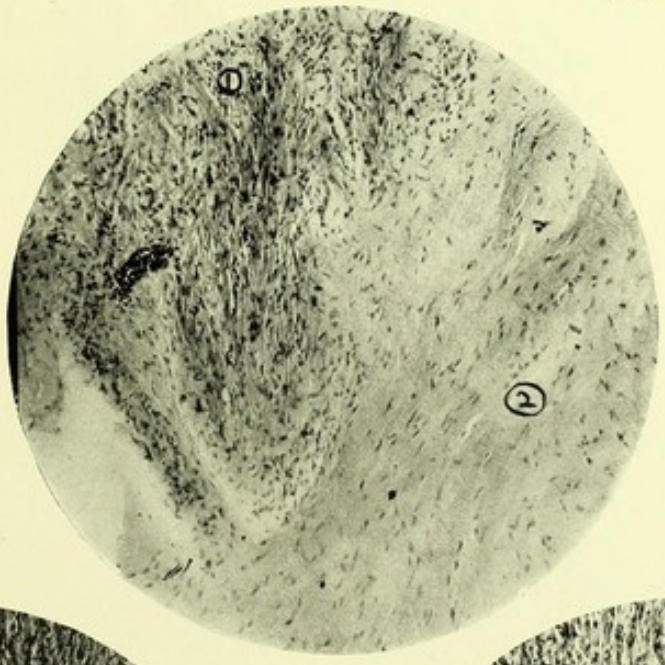

FIG. 3

FIG. 4

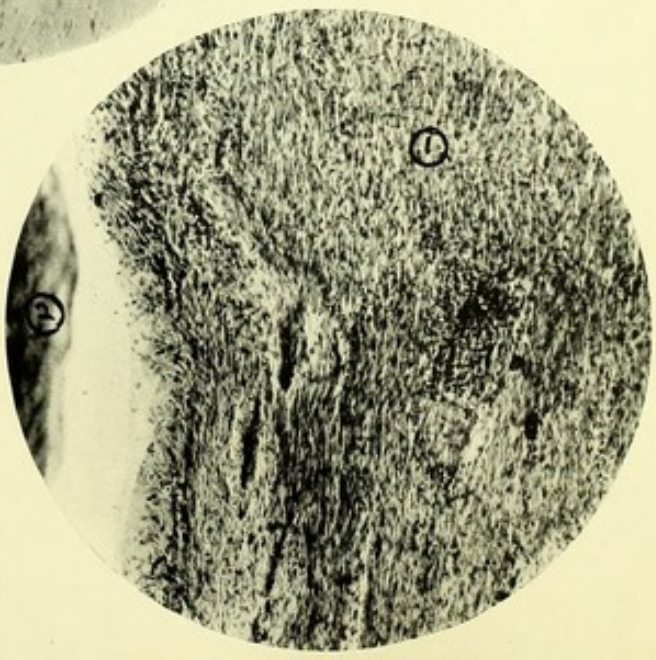

FIG. 5

CASE II

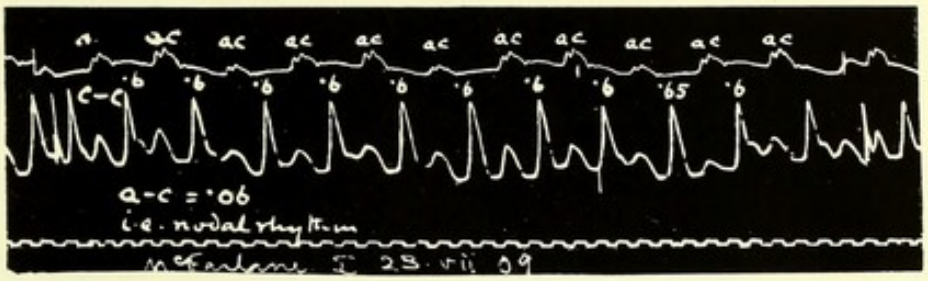

FIG. 1

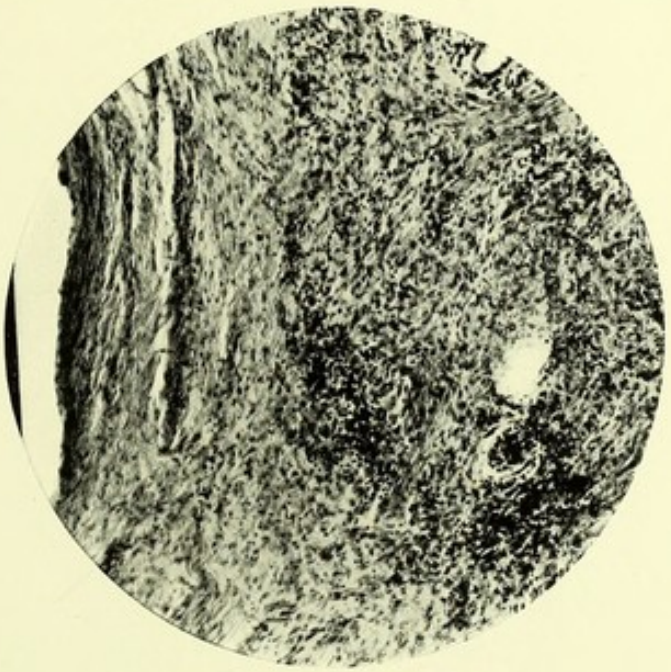

FIG. 2. Section 266

FIG. 3. Section 268

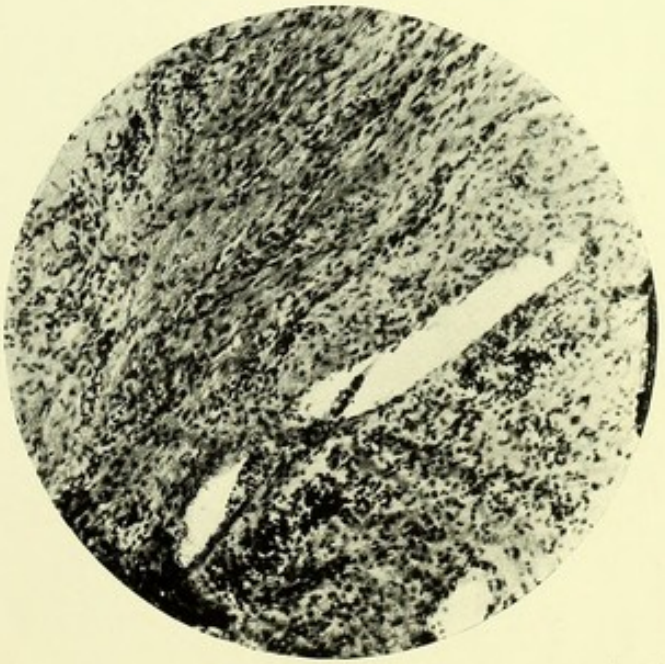

FIG. 4. Section 271