A case of complete heart-block in diphtheria with an account of post-mortem findings / by G.B. Flemming and Alex Mills Kennedy.

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Reprinted from "Heart."
Vol. II, No. 2, November, 1910.
Pages 77 to 83.

London:
Shaw & Sons, Fetter Lane, Fleet Street, E.C.

[*Reprinted from "Heart."*
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(*Glasgow.*)

Heart-block is a condition which has been fairly fully investigated in chronic heart disease, but only a few cases have been reported in acute affections of the heart. Possibly this is not due to the great rarity of the condition, but to the fact that it has been recognised only occasionally. Cases have been described in influenza,[5] rheumatic fever,[1 & 2] ulcerative endocarditis,[3 & 4] typhoid,[6] pneumonia,[6] and in one case of diphtheria,[6] but in only four cases have post-mortem examinations been made. In one, a case of acute rheumatism reported by Gerhardt, the block lasted for at least two months; eventually the patient contracted enteric fever, and died 3½ months after the commencement of the attack of rheumatic fever. Inflammatory lesions were found in the auriculo-ventricular bundle. In the second case (James) ulcerative endocarditis was the cause of death, and an ulcer was found on the left surface of the intraventricular septum immediately beneath and posterior to the undefended space; no miscroscopical examination was made. In the third case (Jellinek and Cooper) death was due to acute gonorrheal endocarditis; there was an acute necrosis of the intraventricular septum with thrombosis of blood-vessels, and there can be no doubt that the A-V bundle was involved in the lesion. Lastly, Magnus-Alsleben[6] describes a case of complete heart-block in diphtheria in a child aged 8 years. An examination of the heart including the A-V. bundle showed the presence of a very severe parenchymatous degeneration.

In the course of an investigation into the heart's action in acute fevers the following case came under observation.

The patient, a female, 10 years of age, was admitted to Ruchill Hospital, Glasgow, on the fifth day of illness, suffering from typical diphtheria of a severe type. She was a poorly nourished and neglected child. On admission the temperature was 99·2°, the pulse 96, and the respiration 24. The tonsils and pharynx were congested; the right tonsil was slightly ulcerated; there was a considerable quantity of loose dirty membrane on the back of the throat. The toxæmia was severe.

Nothing abnormal was found in either the heart, lungs, abdomen, or urine; 8000 units of antitoxic serum were given on the day of admission; the illness was of 10 days' duration. On the sixth day of illness, the day after admission, the patient's pulse rate fell to 62; it rose again on the seventh day as high as 88, but fell on the evening of that day to 40. On

the eighth day it varied between 80 and 52; on the ninth day its maximum speed was 72, its minimum 48; on the tenth day its lowest rate was 40, its highest 62. From the sixth day until death the temperature varied between 99° and 97·4°. Two days before death occurred the heart was considerably dilated, the left border of cardiac dullness was 3½ inches from the middle line, the right border in the middle line, the upper border in the third interspace; the apex beat was in the fifth interspace 3¼ inches from the middle line. Pulsation was visible in the third, fourth, and fifth left interspaces. There was very rapid and obvious pulsation in the neck, immediately above the clavicles, which tracings showed to be due to auricular contractions. The first sounds were somewhat soft and muffled, the second was occasionally duplicated. No sounds corresponding to the auricular contractions could be heard. On the day before death there were signs of palatal paralysis, the voice was nasal and fluids were regurgitated through the nose. The patient died on the tenth day of illness. Permission was obtained for a post-mortem examination of the heart alone. It was found to be distinctly dilated and rather pale in colour. Both the aortic and pulmonary valves were competent; the mitral valve admitted two fingers with ease, and the tricuspid three. A block of tissue was removed from the cardiac septum which contained the *pars membranacea* septi, the adjacent portions of the auricular and ventricular muscular septa, the central fibrous body and the attachments of the aortic cusp of the mitral valve, and the septal cusp of the tricuspid valve. The block extended sufficiently far back to include the mouth of the coronary sinus. It was cut out in such a manner that its long antero-posterior diameter was at right angles to the long axis of the heart.

This block was cut in serial sections from above downwards, in the horizontal plane. Nearly 400 sections were cut, and a series at intervals of five were mounted. The sections were of an uniform thickness of 20 μ and were stained with hæmalum and Van Gieson's stain.

Examination of the sections (Fig. 4, 5, and 6) shows that the A-V node and the first part of the A-V bundle are involved in a well-marked acute inflammatory condition. This is evidenced by the presence of a number of focal collections of round cells and markedly congested capillaries in the node and upper half of the bundle. These cells are for the most part lymphocytes, although occasionally a few larger mononuclear cells, probably of fixed tissue origin, are seen among them. The lowest lesion to be found in the bundle is a large sized inflammatory focus situated a little to one side of the middle line of the bundle; it consists of lymphocytes, a few of the larger cells already mentioned, and also some polymorphonuclear leucocytes. It is evident that this focus is causing considerable damage to the fibres passing through it. The lower half of the bundle and its branches show nothing abnormal.

There are also a number of similar inflammatory foci with congested capillaries in the auricle, bordering on the nodal tissues.

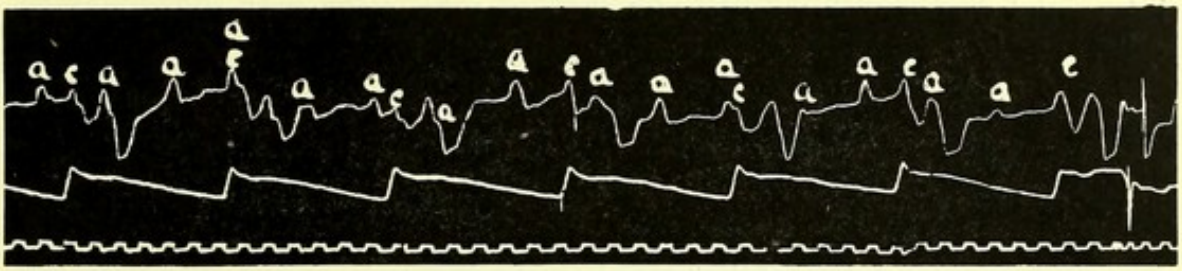

Fig. 1. A tracing from the radial artery and right jugular, showing complete heart-block. Ventricular rate, 46; auricular, about 110. The tracing was taken on the ninth day of illness. The time marker is in 1-10th sec..

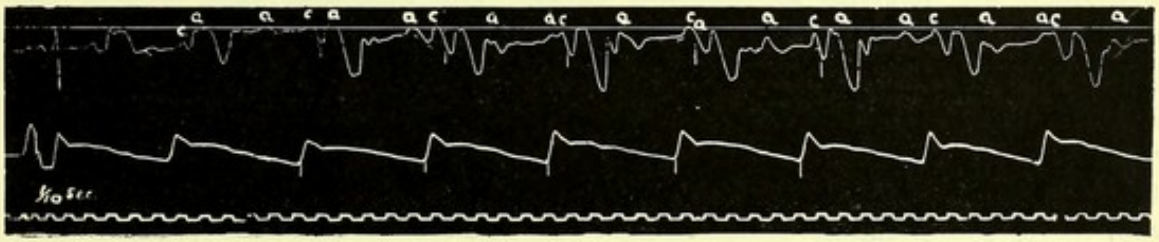

Fig. 2. A tracing showing complete heart-block. Ventricular rate, 54; auricular, about 100. The tracing was taken about nine hours before death occurred.

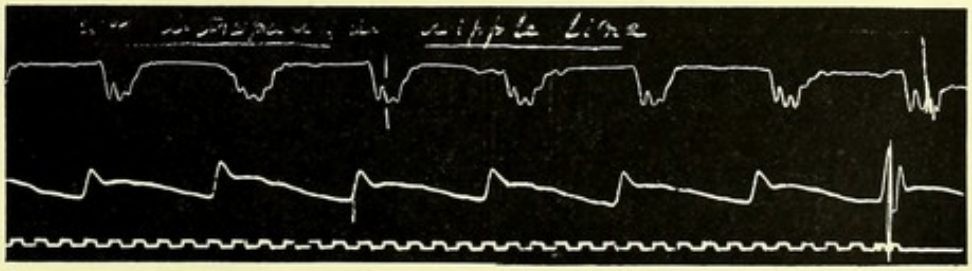

Fig. 3. A tracing from the radial artery and the fifth left intercostal space in the nipple line. The upper tracing is inverted. It shows that with each ventricular contraction there is a corresponding wave in the radial artery. The tracing was taken at the same time as the tracing in Fig. 3; that is, on the tenth day of illness.

The ventricular muscle as seen in the sections shows evidence of an interstitial myocarditis; there is a marked infiltration with inflammatory cells, mostly lymphocytes, in addition to focal collections of similar cells.

The auricular muscle also shows similar well-marked evidence of myocarditis. Here, as before, lymphocytes predominate. There is marked capillary congestion, and in places it is evident that some of the muscle fibres of the auricle have undergone fragmentation.

Clearly, the heart is the seat of a myocarditis, and the A-V node and first part of the A-V bundle have become involved in the inflammatory process. The cellular reaction throughout is chiefly lymphocytic. The lower half and the branches of the A-V bundle, as shown in the sections, are not involved in the condition.

Both vagi were examined by Donaggio's second method for demonstrating early degenerative changes, but nothing abnormal was found.

Tracings were taken on the three last days of the illness, and they all show the same condition. Unfortunately, no tracings were taken during the early stages of the illness, when the pulse was fairly frequent. It will be seen from the tracing (Fig. 1) that the auricles and ventricles are beating at different rates. The auricular rate is 110, the ventricular about 46. Now the pulse rate in complete heart-block is usually between 30 and 40, while in this case it is slightly over 46, and in Fig. 2 it is 54. From this it might be suggested that some of the auricular beats were being conducted to the ventricles, but from an examination of the tracings there is no doubt that the block is complete, the rhythm of the one being independent of the other. It is possible that the rapid action of the heart in this case is dependent upon the general toxæmia. The tracing taken from the apex beat (Fig. 3), at the same time as the tracing in Fig. 2, shows that each ventricular contraction is followed by a pulsation in the radial artery; the tracing is inverted, for it is the dilated right ventricle which is producing the so-called "apex beat." The tracings were taken with Mackenzie's clinical polygraph, with Biggs's time-marker added.

It has been shown that the heart-block may be produced experimentally by vagus stimulations, and as the patient showed signs of paralysis of the palate, it was considered advisable to examine the vagi; however, under the miscroscope, these showed no signs of degeneration. It must be remembered that palatal paralysis set in the day before death, whereas heart-block was established at least three days before death.

It seems to us that the block is dependent upon the numerous inflammatory foci in the auricular-ventricular node and bundle, interfering with the proper conduction of the contraction-stimulus from auricle to ventricle.

The case is of special interest in that it demonstrates that death in diphtheria, where the heart rate is slow and where signs of palatal paralysis are present, is not necessarily the result of inhibitory cardiac impulses.

Fig. 4. Section 141. × 90. Shows the A-V node with a round-celled inflammatory lesion in it.

Fig. 5. Section 166. × 90. Shows the A-V bundle to the right, separated by a fibrous band from the proper cardiac muscle on the left. In the bundle a large inflammatory focus is seen. It consists for the most part of lymphocytes with a few larger fixed tissue cells; there are also some polymorphonuclear leucocytes present. This lesion is apparently damaging the fibres passing through it.

Fig. 6. The same section as Fig. 5. × 180.

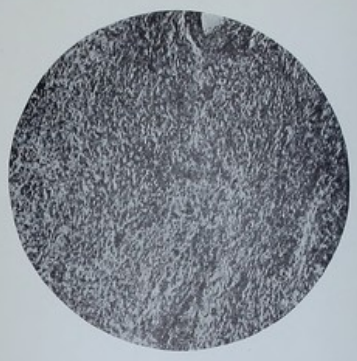

FIG. 4

FIG. 5.

FIG. 6.

SUMMARY.

A case of diphtheria in a child of 10 years is recorded, in which there was an acute inflammatory condition of the heart muscle and primitive cardiac tissue, producing complete heart-block, cardiac failure, and death.

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CONTENTS.