STOKES-ADAMS ATTACKS IN A CHILD

BY

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It is recognized that heart block may occur in several acute infections, but apart from diphtheria and acute rheumatism it is rare and even in the latter it is uncommon. Often the outcome of this complication in children is fatal, but in the following case, a boy aged 12, with heart block and pericarditis, recovery took place.

For three months before admission to hospital he had felt tired and had to rest on returning from school. He had been restless for the past few nights and eventually had to remain in bed for three days. During this time he had throbbing of his heart and headache. For two days he had a troublesome unproductive cough, and on the day before admission he had two attacks of unconsciousness. On coming round he perspired and vomited. He had one attack of unconsciousness the day before coming to hospital and on admission he complained of giddiness, shortness of breath, and persistent coughing. Apart from measles there was no history of any previous illness.

On admission the patient was very ill. He presented grey cyanosis and was distressed by severe dyspnœa. The temperature was normal and the pulse was small and 160 a minute. The blood pressure was 80/40. The respiration rate was 44. There was prominent pulsation and distension of the cervical veins. The apex beat was diffuse and was just outside the mid-clavicular line. The heart sounds were distant and the second sound was split and accentuated in the pulmonary area. There were no murmurs. Numerous rales were heard over the chest. No other abnormal sign presented on further examination; there was no distension of

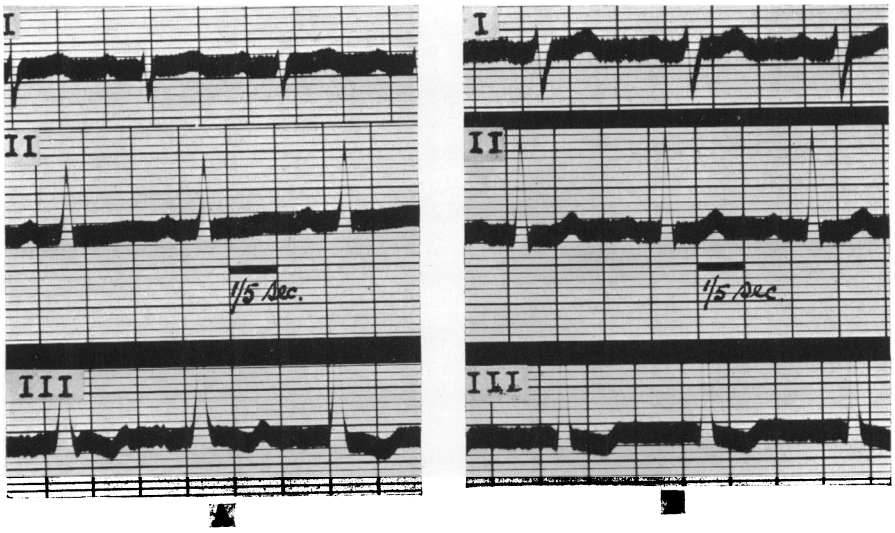

FIG. 1.—Electrocardiograms. (A), taken two weeks after admission, shows flat T waves in lead II and inverted T waves in lead III; (B), taken six weeks later, shows an upright T wave in lead II.

liver and no ascites or œdema. The diagnosis of acute rheumatic pericarditis was considered likely. The patient's general condition deteriorated and he was given oxygen by nasal catheter. Next day the pulse rate dropped to 48. The diastolic blood pressure rose to 70 but the systolic was difficult to record. On the morning after admission, I witnessed two Stokes-Adams attacks. They were preceded by restlessness, an anxious expression, complete irregularity of the pulse, and quick and deep breathing. The pulse, the heart sounds, and later the respiration, ceased. Pallor gave way to cyanosis. There was no twitching. No pulsation of the vessels of the neck was observed during the cardiac standstill. Unconsciousness lasted for ten seconds. After coming round the patient became very flushed and perspired profusely. He was given ephedrine and digitalis and the pulse remained at 80 to 90.

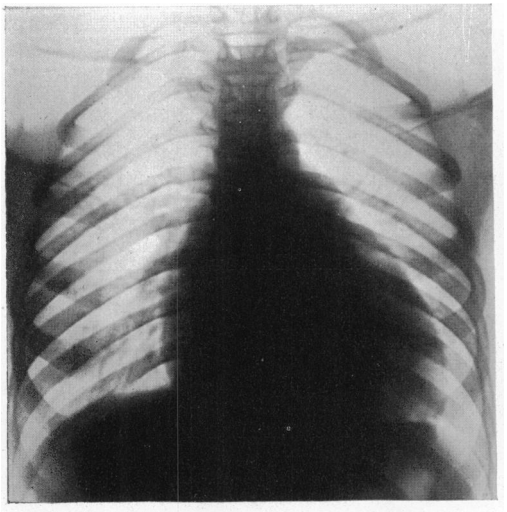

FIG. 2.—X-ray of chest one day after admission to hospital.

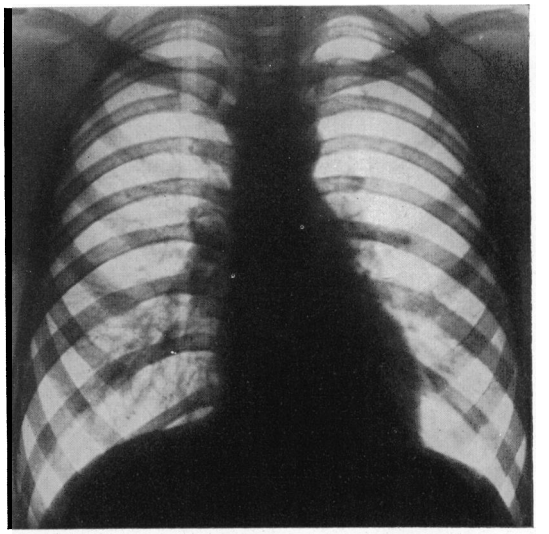

FIG. 3.—X-ray of chest eight weeks after admission to hospital.

During the next three days he had further attacks, but these became less frequent and the patient improved. A pericardial rub appeared on the third day and lasted for three days. At no time did he have any pyrexia or œdema and his later recovery was uneventful. His blood pressure rose to 110/70 and his pulse remained regular at 90. No electrocardiogram was taken during the period of irregularity and later it showed changes due to pericardial effusion with a normal P–R interval (Fig. 1).

Radiography also confirmed the pericardial effusion (Fig. 2 and 3).

DISCUSSION

It is known that primary carditis in childhood is often the first and only manifestation of rheumatic infection. Though Cheadle (1889) and later Moore (1909) stated that carditis was the central factor in rheumatic infection in childhood the diagnosis was often impossible owing to the absence of specific symptoms. In my case the pericarditis was probably rheumatic in origin and its presence was confirmed by the pericardial rub and later cardiographic and cardioscopic findings.

According to Lewis (1925) the cause of clinical heart block is firstly a lesion of the conducting tract, secondly vagal stimulation, or thirdly the result of poisoning. The first cause was operative in my case and the evidence points to heart block caused by rheumatic infection. It is obvious that this infection must have involved the myocardium and the pericardium but there was no evidence of endocardial involvement. The recovery of the patient precluded any pathological examination of his tissues, but certain conclusions can be drawn from the

clinical examination. In the cases that have come to necropsy there have usually been found inflammatory infiltration with rheumatic giant cells and fibrotic changes. In some cases there has only been exudation in the collagenous tissues of the membranaceous septum. Gross and Fried (1935) in 110 necropsies on rheumatic hearts frequently found inflammatory changes in the collagenous extension of the fibrous septum which abuts on the bundle tissue. In many of their cases a portion of the conducting system was entirely surrounded by relatively rigid collagenous tissue and they considered that this, when expanded by exudation, might compress the conducting fibres. It seems reasonable to suppose that in my case as there was no permanent lesion of the conducting system, the pathological change was in the nature of either a minor inflammatory condition of the bundle tissue itself or of such exudation which pressed on the bundle.

Summary

A case of insidious rheumatic pericardial effusion with Stokes-Adams attacks is described. A boy of 12 sought medical help after three months of ill health and only when disturbance of the conducting system had set in. Either minor inflammation of the bundle tissue itself or its compression by inflammatory vascularization of the collagenous mass in the septum membranaceum, would explain the symptoms.

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