STOKES-ADAMS ATTACKS IN PREGNANCY

BY

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Stokes-Adams seizures are a dramatic feature of some cases of complete heart block. Fortunately they are uncommon in the congenital form of heart block (Campbell and Suzman, 1934; Brown, 1950; Wood, 1956), and they must be a rare complication of pregnancy. The following case is therefore of particular interest.

Case Report

A 22-years-old woman was admitted to hospital on April 9, 1959 in the 18th week of her first pregnancy. She had been fit until six days before, when she developed lightheadedness, followed by flushing, and a throbbing occipital headache. At 7 p.m. on the day of admission, in bed, she began to have frequent sensations of lightheadedness, accompanied by throbbing occipital headache, and she lost consciousness repeatedly. In hospital, at 9.45 p.m., she was having two or three episodes of ventricular arrest a minute, each lasting 10 seconds. Her face blanched and she lay virtually unconscious; each attack was followed by a flush and a distressing headache. She was aware only of the headache. Between attacks the heart rate was about 100 a minute, the rhythm was regular and the blood pressure was 120/70. The first heart sound varied in intensity and there was a soft systolic murmur, maximal at the left sternal border; the second sounds were soft. There were no other clinical abnormalities. The uterus was the expected size for an 18-week pregnancy, and foetal movements were felt.

A cardiogram (Fig. 1) showed that during the Stokes-Adams attacks there was ventricular asystole and the P waves occurred regularly 107 times a minute. Between the periods of asystole there was complete atrio-ventricular dissociation, with a ventricular rate of 115 a minute.

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At 10.05 p.m. ephedrine hydrochloride 1 grain (64 mg.) was given I.V.; within 15 minutes the Stokes-Adams attacks had ceased. The ephedrine was repeated I.M. at 10.30 p.m. and for 2 hours there was no change. After midnight brief Stokes-Adams attacks recurred, and despite I.V. ephedrine hydrochloride 1 grain (64 mg.) at 12.40 and a similar I.M. dose at 2 a.m. she continued to have several bouts of ventricular arrest a minute, each lasting one to three seconds. Over the next three hours the attacks became more prolonged and were again lasting 10 seconds.

An intravenous infusion of M/2 sodium lactate was begun at 5.55 a.m. and 200 ml. were given in 70 minutes. Twenty minutes after the beginning of the infusion the length of the periods of cardiac arrest was reduced to 1–2 seconds, and by the end of the infusion there was only an occasional dropped beat at the cardiac apex. Fifteen minutes later the pulse was regular, but a tracing showed that there was still atrioventricular dissociation (Fig. 2).

During the next two days occasional dropped beats were detected; subsequently the rhythm has been regular. Treatment was continued with ephedrine hydrochloride orally, ½ grain (32 mg.) every four hours for 24 hours, then every six hours. The patient went home on the thirteenth day.

Previous History. At the age of 13 she had been discovered at a routine examination to have a cardiac murmur and was, referred for investigation. She was a symptom-free, active girl. There was no history of scarlet fever, rheumatic fever, chorea or diphtheria. Her pulse rate was 56 a minute, rising to 64 after exertion and the rhythm was regular. There was a soft mitral first sound and a systolic murmur at the apex and the pulmonary area. The second pulmonary sound was accentuated. The blood pressure was 130/65. The cardiogram (Fig. 3) showed atrio-ventricular dissociation, and fluoroscopy an enlarged left ventricle and vigorous pulsation of the aorta. A ventricular septal defect was suspected and no treatment or restriction of activity was advised. One year later, the only fresh development was some enlargement of the pulmonary artery, shown on fluoroscopy.

Present Investigations. The plasma bicarbonate before and after the administration of the sodium lactate was 20.7 and 24.0 m.-equ. per l. respectively. Twelve days later it was 21.5 m.-equ. per l. The blood urea measured 13 mg. per 100 ml. The urine contained no albumin.

The chest X-ray was normal. Fluoroscopy showed no abnormality apart from an impression of enlargement of the left ventricular outflow and inflow tracts.

Further Progress. The ephedrine was continued and pregnancy proceeded normally. A systolic thrill became palpable at the left sternal edge. At the twenty-sixth week the plasma bicarbonate level was 24.0 m.-equ. per l. At
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...term a healthy child was born; the labour and puerperium were normal and the child was breast-fed for three months.

Ephedrine therapy was gradually discontinued in December, 1959, and the patient has remained well. When last seen in June 1960, she had no symptoms; the complete heart block persisted (Fig. 4). The child has developed normally. Clinical and electrocardiographic examination of the child and of his mother's parents has shown no evidence of heart block or other cardiac abnormality. There were no other near relatives.

![Cardiogram](image)

**Fig. 4.**—Cardiogram when last seen, June 1960. Atrio-ventricular dissociation persists. Atrial rate approximately 75 and ventricular rate 30 a minute.

**Discussion**

It is probable that this patient has the congenital form of complete heart block. The commonest anatomical abnormality associated with congenital heart block is ventricular septal defect (Yater et al., 1933), although this association has been challenged by Wood (1956). In the present patient the defect may be a small one in the membranous part of the interventricular septum, interfering with the A-V node but not giving rise to a large shunt of blood. The prognosis in congenital heart block depends upon the nature of any associated congenital cardiac lesion (Paul et al., 1958) but it is usually good (Wood, 1956). Stokes-Adams attacks are an uncommon complication.

_Causation of Cardiac Arrest._ Cardiac standstill in patients with complete heart block is usually ascribed to extreme depression of the idioventricular pacemaker. If this were the mechanism in this patient the depression was episodic, for between attacks the ventricular rate was 100–125 a minute. Excessive vagal stimulation depresses the idioventricular rate, and it is possible that stimuli were arising from the uterus, either from foetal movements or because of the rapid expansion and hypertrophy of the uterus.

The hypervolaemia of pregnancy may also have contributed. At 18 weeks the blood volume had increased by about 17 per cent (Dieckmann and Wegner, 1934).

The acidosis may have been another factor in the causation of ventricular asystole. It has been known for many years (Mines, 1912) that acidosis slows the processes of excitation and conduction and that a rise in pH has the opposite effect. It is not clear why this patient should have a mild acidosis. It is not a normal phenomenon in pregnancy, and there was no renal insufficiency.

_Treatment._ Many drugs have been employed in the treatment of Stokes-Adams attacks: sympathomimetic or vagolytic agents, and other drugs such as barium chloride. Bellet et al. (1955a) introduced a new concept. They studied the cardiac effects of intravenous hypertonic sodium lactate, and found that it accelerated cardiac rhythm in various states associated with slow ventricular action, and they were successful (Bellet et al., 1955b) in abolishing Stokes-Adams attacks in three out of four patients. They used molar, M/2 or M/3 solutions of sodium lactate and noted that the degree of cardiac acceleration depended upon the dosage and the speed of infusion. In the...
present case 200 ml. of M/2 sodium lactate given intravenously over a period of 70 minutes finally abolished the Stokes-Adams seizures.

Since Bellet's discovery several reports have been published (Swash and Wallace, 1956; Mouquin et al., 1957; Dragsted and Møller, 1958) on the use of sodium lactate, all of them favourable. (Unfavourable reports on a new form of treatment, however, tend not to be published unless some important side-effect arises.) Bellet et al. (1955a) advanced several theories to account for this action of sodium lactate. The lactate is known to be readily utilized as fuel and to increase the basal metabolic rate of heart muscle and other tissues; or the effect may be due to elevation of the pH of plasma and tissue fluids, or possibly to the supply of sodium ions per se. Some support for the pH theory is provided by the fact that the present patient had a mild acidosis when her Stokes-Adams attacks were occurring, and that this was largely corrected by the sodium lactate, but any of the other theories would also be applicable.

Complete heart block in pregnancy has been reported by Cazzola (1958) in one woman (probably rheumatic) and by Mowbray and Bowley (1948) in three patients (congenital). No previous reports of Stokes-Adams attacks in pregnancy have been discovered.

Familial heart block has been reported by Carp (1958), but there is no evidence of a familial cardiac defect in the present family.

Summary

A young woman with complete heart block, which was presumed to be congenital, developed multiple episodes of ventricular arrest in the eighteenth week of her first pregnancy. The attacks were abolished by an intravenous infusion of M/2 sodium lactate, and ephedrine was given as maintenance treatment. The pregnancy proceeded to a successful outcome and the patient remains well two years later. The heart block persists. The cause of the attacks, and their treatment are discussed.

The patient was under the care of Dr. A. G. Ogilvie and we are grateful for his assistance and for his permission to publish the case. Our thanks are also due to Dr. W. G. A. Swan for permission to publish his previous findings.

References

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