

THE USE OF TEMPORARY EXTERNAL PACEMAKER TO CONTROL SURGICALLY PRODUCED COMPLETE ATRIO-VENTRICULAR BLOCK DURING OPEN HEART SURGERY

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Medical treatment of total heart block has been known for many years. In spite of very effective new drugs, however, such as Isuprel^R (isoproterenol), it is often not possible to prevent death by medical treatment alone.

The introduction of the implantable pacemaker by Chardack in 1960¹ brought new scope to the management of this problem, and, after favorable results were reported, the use of this pacemaker became widespread. The pacemaker was first used in Turkey in December 1962 at Hacettepe Medical Center in the treatment of a 66 year old man who had had multiple Stokes-Adams attacks. The results were successful.²

Total heart block may be caused by congenital or acquired heart disease, as well as by open heart surgery. The purpose of this paper is to report a case in which a large ventricular septal defect was closed with the aid of a heart-lung machine and the resulting heart block was controlled by a temporary external pacemaker.

Case Report

S. B. (64-193), a 13 year old girl, was first seen in January 1964 with the chief complaint of palpitations. Her history revealed that palpitations had been present since infancy and that she had developed cyanosis on exertion during the last five years. She was the product of an uneventful pregnancy and normal spontaneous delivery. There was nothing else of relevance in her history.

Physical Examination : The patient's weight was 21 kg; height, 140 cm; blood pressure, 90/70 mmHg. Femoral pulsations were of good quality. There was no thrill over the precordium. A grade 3 (grading VI) systolic murmur was heard in the left fifth intercostal space along the sternal border. The second heart sound was accentuated over the pulmonary area.

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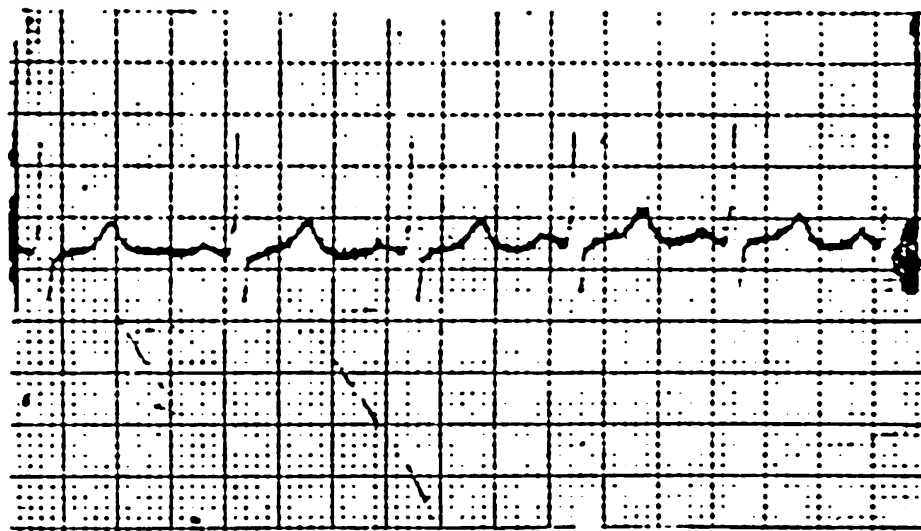
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Laboratory Examination : Hemoglobin, 19.5 gm/100 ml; WBC, 8000/cmm; NPN, 30.5 mg %; PPD (10 U), negative.

X-ray studies of the heart with barium swallow showed both ventricles to be enlarged. The pulmonary conus was prominent and pulmonary vascularity was increased in both lungs.

Electrocardiography showed right axis deviation and biventricular hypertrophy (Figure 1). Cardiac catheterization was performed on May 29, 1964,



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Figure 1. Preoperative electrocardiogram with normal sinus rhythm.

through the left antecubital vein under local anesthesia. The right atrium, right ventricle and pulmonary artery were reached. There was no pressure gradient between the pulmonary artery and right ventricle. Right ventricular and pulmonary artery pressures were equal to the systemic pressure. There was a left-to-right shunt at the ventricular level. Arterial oxygen saturation was 97.8 per cent and no right-to-left shunt was detected at rest. The final diagnosis was ventricular septal defect with severe pulmonary hypertension.

On April 8, 1965, open heart surgery was performed. The chest was opened by a vertical sternal split incision. After the precordium was opened, a markedly enlarged right ventricle with a fibrotic plaque (soldier's heart) was observed. The pulmonary artery was three times the size of the aorta. A systolic thrill was palpable on the pulmonary artery. Both venae cavae and the right femoral artery were cannulated in the usual manner. After instituting total by-pass, the right ventricle was opened by a transverse incision. A defect, three cm in diameter, was found in the muscular part of the interventricular septum and was closed with a teflon patch. Complete atrio-ventricular (A-V) block occurred while the patch was being sutured. The stitch which was thought to be the cause was removed, but the A-V block persisted. Therefore, platinum electrodes were placed in the myocardium and connected to the external pacemaker (Figure 2). The heart rate was regulated at 88 beats per minute. The

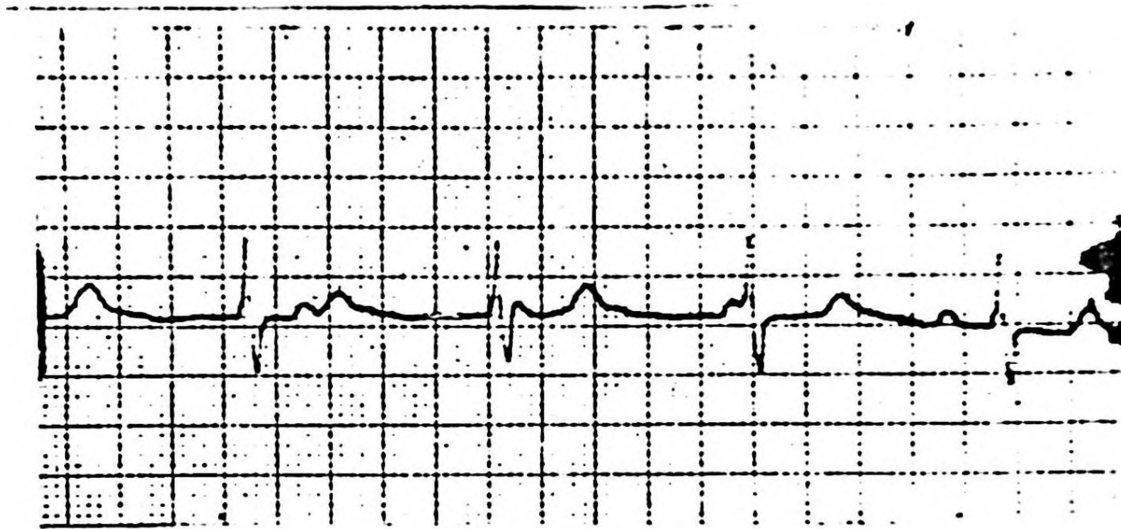


Figure 2. Patient after surgery, showing portable temporary external pacemaker. The electrode, seen at top, leads to the heart. Desired electrical activity is controlled by the upper dial, pulse rate by the lower dial.

patient was taken off extracorporeal circulation in the usual manner. Blood pressure and other vital signs were satisfactory. The patient was given steroids with the thought that the block might have been caused by edema and/or a hematoma at the site of the removed stitch. On the eleventh post operative day the pacemaker was stopped to test the patient's reaction, but was started again because her pulse rate dropped to 60 beats per minute. In order to obtain a satisfactory myocardial response, it was necessary to increase the electrical activity of the pacemaker from 12 to 17 milliamperes.

The electrodes were removed on the seventeenth post operative day, but, at midnight on the nineteenth day, the pulse rate slowed suddenly to 43 beats per minute. Isuprel[®] was administered and the rate increased to 80 per minute. In spite of persistent total heart block, the clinical condition continued to be satisfactory and the patient was discharged on the fortieth post operative day under treatment with Alupent (Figure 3). Follow-up examinations over a period of eight months indicated no change in the heart block.

On November 9, 1965, at the time of her regular examination, her mother stated that she herself had discontinued the medicine because the pulse rate was noted to be fast. Electrocardiography revealed normal sinus rhythm (Figure 4). The patient has been seen every three months since that time. There has been no change in the sinus rhythm and she has been able to lead a normal, active life.



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Figure 3. Electrocardiogram of patient at time of discharge. Complete A-V block is present,



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Figure 4. Patient's electrocardiogram eight months after surgery, showing normal sinus rhythm.

Discussion

There are two points of interest in this case. First, the operation itself was performed in spite of pulmonary hypertension. The patient's pulmonary and systemic pressures were equal on cardiac

catheterization and cyanosis was present on exertion. In other words, this patient was in the early stage of Eisenmenger complex, and would have been considered inoperable by many authorities.³ We believe that if the patient has no cyanosis at rest and the predominant shunt is from left to right, an operative chance should be given to these unfortunate children, since death is the only alternative to surgery in these cases. Of course, the surgical mortality is high among those who have severe pulmonary hypertension.⁴ Hallmar and Cooley reported 50 per cent mortality in patients whose pulmonary pressure is 80 per cent or more of systemic pressure.⁵ In our patient, the pulmonary pressure was 100 per cent of systemic pressure. In spite of this, the child benefitted from surgery.

The second interesting point in our case was the rôle of the temporary external pacemaker in the control of operatively produced total heart block. There is a special danger of total heart block during open heart operations for tetralogy of Fallot, endocardial cushion defects and some ventricular septal defects. Better knowledge of the location of the conduction system and improved suturing techniques have helped in preventing this often fatal complication. In spite of these advances, total heart block still remains a serious problem, especially in these operations. Patients with surgically produced heart block may die suddenly, even though their clinical condition may be good, if sinus rhythm does not return or if the heart block is not controlled. Because of this, in every center where open heart surgery is performed, pacemaker control should be available. In our case, an external temporary pacemaker was used to control a surgically produced heart block, because we considered it the best method to employ on a patient whose heart block was thought not to be permanent. The pacemaker may be turned off temporarily to test the patient's reaction and heart rate, and the electrical voltage can be regulated to obtain a satisfactory myocardial response. Furthermore, the electrodes can be easily removed when the pacemaker is no longer needed. Its use is a great advantage to the surgeon and the danger of often fatal complications is greatly decreased.

In the case reported here, total heart block returned to normal sinus rhythm eight months after surgery. Since the stitch was removed immediately after the occurrence of the block, temporary edema and/or hematoma is the most likely explanation. The temporary nature of the block further supports this supposition.

Summary

We have presented a case of ventricular septal defect with severe pulmonary hypertension in which total heart block developed during surgical repair. The rôle of pulmonary hypertension is discussed in the prognosis and surgery. Details of the management of surgically produced heart block occurring during the repair of a large ventricular septal defect are given and the rôle of the pacemaker in reducing the mortality from heart block is emphasized.

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