Pacemaker Therapy in Pregnancy for the Management of Sinus Bradycardia-Junctional Tachycardia Syndrome* 

John W. Schatz, Maj, MC; James A. Fischer, Maj, MC; Roger F. Lee, Maj, MC; and Richard M. Lampe, Maj, MC

A patient with Ebstein's malformation and tachyarrhythmias is described. The management of her complicated rhythm disturbances during pregnancy and successful labor and delivery was made possible by a temporary transvenous pacemaker. Pacemaker therapy during pregnancy is reviewed. The role of drugs and their potential complications to the fetus make electrical therapy more desirable in these situations.

Fetal morbidity and mortality may be adversely affected by placental transfer of drugs. This has been well-documented in cases of heroin abuse.1 We recently participated in the management of a patient during parturition with alternating sinus bradycardia-junctional tachycardia rhythm disturbance (probably drug-induced) which required parenteral drug therapy for control. A pacemaker was required for successful management. This demonstrated the effectiveness of pacemaker therapy during pregnancy and presented a form of therapy that was without risk to the fetus.

CASE REPORT

A 21-year-old, gravida one, para zero, Caucasian woman was admitted to Silas B. Hays Army Hospital on November 28, 1972 with a history of sudden onset of palpitations, dizziness and fatigue. Her cardiac history began in childhood when she was told that she had a heart murmur and was advised not to participate in physical education classes. She never felt limited in her activities, but followed medical advice. At age 14 cardiac catheterization was performed. Hemodynamics and oxygen saturations were normal. Right ventricular angiography demonstrated displacement of the tricuspid valve leaflets into the ventricle and minimal tricuspid regurgitation, findings compatible with Ebstein's anomaly. She continued to do well until age 16, at which time she experienced the sudden onset of palpitations. Her physician placed her on digoxin, 0.25 mg twice daily, with cessation of her arrhythmia. Between ages 16 and 20, she experienced no recurrence. In late March, she became pregnant. On June 25, 1972 she was admitted to the hospital for paroxysmal supraventricular tachycardia. Oral quinidine sulfate, 200 mg every six hours, controlled the arrhythmia and the patient was sent home on quinidine sulfate and digoxin. While at home she experienced four to six episodes of palpitations during the months of July, August and September. Quinidine was increased to 300 mg every six hours with good control for October and most of November. On the morning of the twenty-eighth, she was treated successfully for paroxysmal supraventricular tachycardia with phenyl-

*From the Departments of Cardiology, Obstetrics and Pediatrics, Silas B. Hays Army Hospital, Fort Ord, California. The views of the authors do not purport to reflect the position of the Department of the Army or the Department of Defense.

Reprint requests: Dr. Schats, USAH, Fort Ord, California 93941

CHEST, 65: 4, APRIL, 1974

Figure 1. Twelve lead electrocardiogram during supraventricular tachycardia at a rate of 160 per minute and demonstrating retrograde P waves (retouched).

ephrine hydrochloride, after other vagal maneuvers had failed (Fig 1 and 2). However, she presented again that afternoon with the same rhythm disturbance. Physical examination revealed a ruddy-complexioned, young pregnant woman. Her systolic blood pressure was 90 mm Hg and pulse rate was 160 per minute and regular. Prominent jugular venous pulsations were present. Carotid pulsations were normal. There was no thyroid enlargement. Her chest was clear to auscultation and percussion. The first and second heart sounds were clearly discernible. A systolic ejection murmur, grade 2/6 was present along the left sternal border. An early diastolic sound was heard in the fourth intercostal space at the left sternal border. Abdominal examination revealed an enlarged uterus of approximately 35 weeks gestation. Her extremities were normal.

Hospital course consisted initially of vagal maneuvers (not including pressor agents) and an additional dose of digoxin, 0.25 mg, but to no avail. Intravenous propranolol, 3 mg in six

Figure 2. Twelve lead electrocardiogram after conversion to sinus rhythm demonstrating first degree heart block and prominent P waves of right atrial enlargement (retouched).
minutes, converted the arrhythmia to a sinus mechanism. Oral propranolol was started. This failed to maintain sinus rhythm. An additional 1 mg dose of propranolol, six hours after the oral dose, was given intravenously. There was no change in rhythm. Phenylephrine was given one hour later which caused sudden cessation of tachycardia and a 4.7 second period of ventricular asystole (Fig 3). The rhythms that followed demonstrated an alternating sinus bradycardia-junctional tachycardia syndrome (Fig 4). Procaine amide, intravenously, was also successful in temporarily halting the rapid supraventricular arrhythmia. At no time did fetal heart tones slow following intravenous procaine amide or propranolol. The drug regimen at this time consisted of digoxin 0.25 mg twice daily, quinidine 300 mg every six hours and procaine amide, 250 mg every six hours. The tachyarrhythmia was poorly controlled. On November 30, 1972 her membranes spontaneously ruptured and labor rapidly ensued. Parenteral penicillin and streptomycin were given. A temporary transvenous bipolar catheter was passed to the apex of the right ventricle and pacing performed with a demand pacemaker (Medtronic, Model 5880). Pacing at 98 beats per minute was sufficient to override her tachyarrhythmia (Fig 5). Caudal anesthesia was performed at 5 cm dilatation. The second stage of labor was shortened by cephalic suction and a mid-forceps delivery. Fetal heart tones remained regular throughout labor. A five pound, Apgar 3 (1 minute) girl was delivered who responded to vigorous naso-tracheal suctioning (Apgar 8 at 5 minutes). Delivery was well tolerated by the mother. Two days later the pacemaker was removed. Two short episodes of paroxysmal supraventricular tachycardia occurred when the procaine amide was stopped. Reinstitution of this medication easily controlled the arrhythmia.

A digoxin level which was later available was 3.4 nanograms per milliliter (toxicity commonly occurs at levels over 2.0 nanograms per milliliter.*

At present, two months after discharge, mother and child are doing well.

**DISCUSSION**

The complicated rhythm disturbance in our patient may in part have been drug-induced. Digoxin level was elevated in the mother and may well have been so in the fetal blood. Drugs used to control maternal arrhythmias may cross the placenta and be injurious to the fetus.*

*Performed by Bio-Science Laboratories, Van Nuys, California

---

**FIGURE 3.** Monitor lead demonstrating 4.7 seconds of ventricular asystole after injection of phenylephrine intravenously (continuous recording).

**FIGURE 4.** Monitor lead demonstrating recurrent supraventricular tachycardia, sudden cessation, and depressed sinus node function (tachy-brady syndrome).

**FIGURE 5.** Monitor lead demonstrating ventricular pacing at 98 beats per minute and 1 to 1 retrograde conduction (pacemaker spike not well seen in this lead).
Thus, the role of electrical management may be more desirable in these situations. Pacemaker usage offers a mode of therapy which does not depress fetal heart rate or contractility.

Pacemaker therapy during pregnancy was first reported by Shouse and Acker in 1964.\textsuperscript{5} Since that time, approximately nine additional cases have been reported, all significantly, without maternal or fetal mortality.\textsuperscript{5,6} The indication for pacing has been complete heart block, congenital or acquired, occasionally associated with ventricular tachyarrhythmia. Prior to pacemaker availability, heart block in pregnancy was accompanied by a 6 percent incidence of heart failure, 9 percent incidence of toxemia, a 13 percent maternal death rate and 15 percent fetal loss.\textsuperscript{7} These statistics are not necessarily the result of Stokes-Adams episodes, but may represent the potential role of brady-arrhythmias for accentuating heart failure and anoxic injury to the fetus.\textsuperscript{8}

The only morbidity associated with pacemaker use has been with permanent units installed prior to pregnancy. These have included local irritation with ulceration from a unit located in the breast region,\textsuperscript{9} transient pacemaker competition,\textsuperscript{9} and temporary loss of capture which required an increase in milliamperage.\textsuperscript{9} No episodes of thromboembolism occurred.

In summary, pacemaker therapy offered a favorable alternative to drug therapy in the treatment of a refractory arrhythmia during pregnancy. There appears to be no associated fetal morbidity or mortality.

ACKNOWLEDGMENTS: We are indebted to Mrs. Arleen Burston, Mrs. Frauke Moore and Mrs. Vicki Taylor for their assistance in preparation of this manuscript.

REFERENCES


Esophageal Ulceration and Oral Potassium Chloride Ingestion*

T. Rosenthal, M.D.;** R. Adar, M.D.;† J. Militianu, M.D.;‡ and V. Deutsch, M.D.¶

In a patient with rheumatic heart disease and a large left atrium compressing the esophagus, potassium chloride produced severe esophageal ulceration, possibly contributing to the patient's demise. This is the second report of esophageal ulceration caused by oral potassium therapy.

Ulceration of the bowel due to oral potassium therapy has been previously reported.\textsuperscript{1-3} Most lesions described were of the ulcerating-stenosing type.\textsuperscript{1-3} Experimentally, similar lesions were produced both in small and large bowel,\textsuperscript{4} however, there is only one report of an esophageal ulceration due to oral potassium therapy.\textsuperscript{5} The potassium ulcers described in the small bowel were mostly treated surgically,\textsuperscript{5} and the esophageal ulcer described by Pemberton\textsuperscript{6} apparently healed spontaneously on withdrawal of the drug.

The present case is the second reported in the literature of an esophageal ulcer. It is somewhat unique because the potassium ulcer may have contributed to the death of the patient.

CASE REPORT

A 65-year-old woman was admitted with pain in the chest growing progressively worse over the preceding two months. At the age of ten, she had rheumatic fever and at 44, following an episode of hemoptysis and palpitations, a diagnosis of rheumatic heart disease with mitral stenosis and aortic regurgitation was made. A mitral commissurotomy was performed.

Four years after the operation, she again developed signs of cardiac failure. Chest x-ray examination showed cardiomegaly with marked enlargement of the left atrium compressing the middle third of the esophagus against the vertebral column and causing a slight delay in the passage of barium through the mid-esophagus. The patient received diuretic treatment with oral potassium supplement in the form of 0.5 gm tablets of noncoated potassium chloride, taken with water at the time of meals.

Careful history taken during the present admission revealed that for two months prior to admission, swallowing dry food resulted in a dull pain which began beneath the xiphoid and spread upwards along the sternum, radiating towards the neck and the left shoulder. A sensation of strangulation accompanied the dysphagia.

During the two weeks prior to her admission, these symptoms became more prominent culminating in severe acute precordial pain on the day of admission. The physical examination revealed the known cardiac signs, as well as evidence of left heart failure. The abdomen was normal except for a palpable liver. Laboratory tests were noncontributory.

Barium examination of the esophagus and stomach revealed dilatation of the mid-esophagus caused by extrinsic compression. In the middle of this segment a large ulceration

*From the Departments of Internal Medicine**, General and Vascular Surgery† and Diagnostic Radiology‡, the Chaim Sheba Medical Center, Tel-Hashomer, and the Tel-Aviv University Medical School, Tel-Aviv, Israel. Reprint requests: Dr. Deutsch, Chaim Sheba Medical Center, Tel-Hashomer, Israel.

ESOPHAGEAL ULCERATION AND ORAL POTASSIUM INGESTION 463