Disseminated intravascular coagulation in miliary tuberculosis is rare. Goldfine et al. described an elderly patient who developed rectal hemorrhage in the wake of respiratory arrest and died in spite of heparin treatment. Unfortunately, no information is provided regarding the patient's metabolic status at the time of arrest or subsequently, and both shock and profound acidosis are among multiple known etiologies of DIC. Mavligit et al. reported a patient with miliary tuberculosis who developed gastrointestinal hemorrhage and survived after treatment with heparin, isoniazid, ethambutol, and streptomycin. Bleeding disorders have been associated with miliary tuberculosis, and DIC has been suspected retrospectively. Huseby and Hudson have recently described three cases of miliary tuberculosis and concomitant adult respiratory distress syndrome. All three of their cases and a previously reported case by Homan et al. had evidence of coagulation abnormalities consistent with DIC. Drug-induced coagulation abnormalities must also remain a consideration and the importance of this has recently been emphasized by a report of DIC occurring secondary to isoniazid-induced hepatitis. Sahn and Neff also noted drug-induced marrow changes and stressed the difficulty of differentiating the effect of the underlying disease from that of drug-induced abnormalities. In addition, there are numerous reports of antituberculosis drug-induced leukopenia and thrombocytopenia.

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Symptomatic Unilateral Cannon "a" Waves in a Patient with a Ventricular Pacemaker

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A 64-year-old woman was referred because of intermittent pulsations of the left side of the neck, face, and scalp that were first noticed after the insertion of a ventricular pacemaker. The pacemaker had been inserted because of symptomatic 2:1 atrioventricular block. Right cardiac catheterization showed cannon "a" waves, and phlebographic studies revealed stenosis of the right innominate and internal jugular veins. The symptoms were abolished by conversion to an atrial synchronous pacing system. Comments are offered on the hemodynamic findings, the "pacemaking syndrome," and the use of atrial synchronous pacing.

Giant or cannon "a" waves of a jugular pulse can be observed when the atria contract against the closed atrioventricular valves and the atrial volume is discharged retrogradely into the peripheral veins. The occurrence of such cannon "a" waves is therefore to be expected in various forms of atrioventricular dissociation, particularly in complete heart block and during ventricular pacing in the presence of a preserved sinus mechanism. Rarely are significant subjective symptoms produced. The following case report is of note because as a result of coexistence of ventricular pacing (VVI) and unilateral stenosis of the right innominate and internal jugular vein, the pulsations in the neck were unilateral and of such a degree that conversion of the pacing system to an atrial synchronous mode (VAT) was necessary ("VVI" is a code designation for a ventricular inhibitory pacemaker).

CASE REPORT

A 60-year-old white woman was admitted for intermittent pulsations of the left side of her neck, face, and scalp, which

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were particularly prominent when she was in the supine position. She had had intermittent second-degree atrioventricular block since 1964 but had been asymptomatic until October 1975. At that time a ventricular inhibitory pacemaker was inserted at another hospital, when the patient had become symptomatic from persistent 2:1 atrioventricular block. She had complained of fatigue, dyspnea, and near syncope.

Physical examination showed an alert, oriented, cooperative, middle-aged white woman in no distress. The findings from examination of the head, ears, nose, and throat were within normal limits. The thyroid gland was normal. The veins of the neck were flat at an elevation of 45°. Intermittent pulse-synchronous waves of varying and cyclic intensity were visible and palpable in the left side of the neck. No similar pulsations were observed on the right. Carotid pulses were equal, and there were no murmurs. The pulse generator was in a well-healed left infraclavicular pocket. The lungs were clear. Examination of the heart showed a regular rhythm at 72 beats per minute; the point of maximal impulse was not palpable, and there were no heaves or thrills. The first heart sound was of varying intensity; the second heart sound was normal. A soft ejection murmur was heard at the apex. Third or fourth heart sounds were not audible. The blood pressure was 130/80 mm Hg in both arms. The abdomen was soft, and the liver and spleen were not palpable. There were no abdominal masses, nor were there pulsations similar to those in the neck. The liver did not pulsate. Femoral pulses were present and equal bilaterally. There was no peripheral edema. Varicose veins were noted in both calves. Findings from the neurological examination were within normal limits. A complete blood cell count and differential count, levels of electrolytes, automated analysis of blood chemistry (SMA-12), clotting factors, and urinalysis revealed no abnormalities.

The electrocardiogram showed a 1:1 response to the pacemaker at 70 beats per minute and sinus P-waves at 75 beats per minute. An x-ray film of the chest showed the cardiac contour to be normal; a unipolar pacing electrode was found to be positioned properly in the apex of the right ventricle. The pulmonary fields were clear, without evidence of congestive changes.

**Course of Hospitalization**

The patient underwent right cardiac catheterization and selective phlebographic studies of both internal jugular veins by manual injection of meglumine diatrizoate (Renografin) (study conducted by I.R.Z.). Simultaneous tracings of the left internal jugular and right carotid pulses were obtained (Fig 1). Stenosis of the right internal jugular vein at the junction of the right innominate vein was demonstrated (Fig 2); there was massive dilatation of the left internal jugular vein throughout its course. Pressure recordings obtained in the inferior vena cava failed to reveal pulses of similar magnitude. The mean pressures in the inferior vena cava, right atrium, and superior vena cava were normal.

It was believed that in some way the atrial contraction had to be used, either in atrial synchronous pacing (VAT) or in bifocal pacing (DVT) to relieve the patient’s symptoms. In either event an electrode would have to be placed in the atrium, as well as a second electrode in the ventricle. This could be done by removing the system on the left and replacing it with an entirely new transvenous system on the right, or by a less satisfactory method of removing the present system and, via thoracotomy, attaching the appropriate leads directly to the atrium and ventricle. A third alternative was to utilize the existing ventricular electrode, and insert an atrial electrode into the atrial appendage through an ipsilateral vein.

The last approach was selected as being the most appropriate. A bipolar wire (American Optical J Wire) was inserted through the subclavian vein and was positioned in the atrial appendix according to a previously described technique. Thresholds were 1.0 v and 1.2 ma in the atrium and 1.6 v and 1.3 ma in the ventricle. The amplitude of the P wave was adequate to trigger the atrial input circuit of a programmable atrial synchronous pacemaker (Cordis Omniloc System).

Thereafter, cannon “a” waves could no longer be observed, and the patient experienced complete relief from the left-sided pulsations. Intermittent conversion of the system to the fixed-rate mode of pacing with an externally applied magnet resulted in immediate reappearance of the cannon waves and
patient’s symptoms suggest two possibilities. The thrust of a normal right atrial contraction was diverted solely into the innominate and left internal jugular veins; and had the stenosis on the right not been present, the patient would have had much milder symptoms because of the buffering effect of a larger venous reservoir. Or is it possible that this patient has usually vigorous atrial contractions and would have experienced bilateral pulsations of a similar magnitude even if the right internal jugular vein were not stenosed? The absence of symptoms in the left arm was attributed to the fact that the veins of the proximal portion of the arm are protected by valves, while the internal jugular vein is not similarly protected. It is possible that a competent eustachian valve at the mouth of the inferior vena cava prevented the propagation of the cannon waves into the inferior vena cava and further accentuated the jet into the innominate and jugular veins.

Upon superficial examination the symptoms experienced by this patient may resemble the so-called “pacemaking syndrome.” This syndrome, first described by Mitsui et al. in 1969, consists of systemic symptoms (such as dyspnea, weakness, dizziness, pain in the chest, cold sweats, flushing of the face, and palpitations) and is believed to be caused by an inappropriate rate of pacing, indicated by a fast atrial rate. The symptoms of this syndrome are promptly relieved by a change in the rate of pacing. We believe that our patient does not fit these previously mentioned criteria. Her symptoms were unrelated to heart rate and were clearly related to the recorded “a” waves in the jugular pulses. Furthermore, these symptoms were unilateral, were limited to the neck and head, and were not associated with the other systemic symptoms of the “pacemaking syndrome.”

Perhaps worth emphasizing is the potential use of the atrium in more patients than has been the custom in the United States. According to a recent survey, not more than 2 or 3 percent of the pacemakers inserted transvenously have been atrial synchronous or bifocal pacing units, because there is a lack of confidence in the stability of the permanent atrial electrode inserted in this fashion. A dislodgment occurs with about the same frequency as for transvenously placed ventricular electrodes, which is about 4 percent in the first two weeks. In addition to preventing cannon “a” waves and the “pacemaking syndrome,” the use of the atrium in pacing enhances cardiac output from 5 to 35 percent in our experience, and it may be important in patients with borderline left ventricular function.

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Ebstein's Disease Associated with Complete Atrioventricular Block*

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The first documented case of Ebstein's anomaly associated with complete heart block is reported in a 49-year-old man who presented with symptoms of low cardiac output and complete heart block. His bundle electrography performed during recovery of intermittent atrioventricular conduction demonstrated infra-His block with prolonged H-Q interval and normal A-H interval. Treatment consisted of permanent cardiac pacemaker implantation.

Ebstein's disease, a congenital cardiac anomaly in which the tricuspid valve is deformed and displaced downward into the right ventricle, is characterized clinically by a constellation of electrophysiographic abnormalities and dysrhythmias.1-4 Most prominent among these derangements are supraventricular tachycardias,4 bizarre forms of right bundle branch block,2,4 intra-atrial conduction disturbances4 and type B WolfParkinson-White syndrome.4-6 To our knowledge, bradyarrhythmia and high grade atrioventricular conduction block have not been previously documented in Ebstein's anomaly. This report describes the first case of Ebstein's anomaly associated with complete heart block.

CASE REPORT

A 49-year-old white man presented to the University of California, Davis-Sacramento Medical Center emergency room in October, 1975, complaining of fatigue, severe dyspnea and dizziness of six weeks' duration. His cardiac history dated to childhood when a heart murmur was noted and decrease in exercise tolerance began. There was no history of rheumatic fever or scarlet fever and family history was negative for cardiac disease.

The patient was first hospitalized for cardiac symptoms in 1972 at another institution because of exertional dyspnea. Physical examination and chest roentgenogram at that time revealed cardiomegaly with partial enlargement of the right atrium. Electrocardiogram (Fig 1) demonstrated normal sinus rhythm at 78/min, complete right bundle branch block and right axis deviation of +165°. Cardiac catheterization was performed on that admission and demonstrated normal right and left heart hemodynamics. However, right ventricular cineangiography revealed downward displacement of the tricuspid valve, a right ventricle of reduced volume and moderate tricuspid regurgitation into a grossly enlarged right atrium with a large saccular portion which had a trabeculated endocardium. An atrial septal defect was demonstrated with a small right-to-left shunt by venae caval dye curves. Left ventricular cineangiography revealed a slightly enlarged chamber with normal contractile pattern and competent mitral valve. The saccular, trabeculated portion of the right atrium was interpreted as representing "atrialization of the ventricle" due to an anomalous attachment of the tricuspid valve, which also accounted for its incompetence. Diagnosis was Ebstein's anomaly of the tricuspid valve. After discharge, the patient was lost to follow-up until he presented to the Sacramento Medical Center in October, 1975.

Physical examination revealed blood pressure 130/80 mm Hg, pulse 40/min and regular, respirations 14/min. Cardiac examination revealed the point of maximum impulse in the fifth intercostal space at the midclavicular line. S1 varied in intensity, S2 was physiologically split and there was a grade 2/6, high-pitched, early systolic ejection murmur along the left sternal border which increased slightly in intensity with inspiration. There were no gallops or diastolic murmurs. Examination of the chest, abdomen and extremities revealed no abnormalities.

The electrocardiogram showed advanced atrioventricular block with idioventricular rhythm at a rate of 38/min and intermittent conduction of sinus beats. Chest roentgenogram disclosed prominent right heart border and normal pulmonary vascularity. Routine hematologic data, serum chemistry and urine studies were normal.

The patient was admitted to the cardiac care unit where a temporary right ventricular pacemaker was inserted. Right heart catheterization, performed with an end hole electrode catheter, revealed the following data: right atrial mean pressure = 2 mm Hg, right ventricular pressure = 20/2 mm Hg, pulmonary artery pressure = 20/7 mm Hg. Simultaneous unipolar intracardiac electrogram and pressure, obtained as the catheter was withdrawn from the right ventricle to right

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