SELECTED REPORTS

Bradycardia-Dependent Bundle Branch Block in Acute Myocardial Infarction*

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A 78-year-old woman with an acute myocardial infarction exhibited left bundle branch block during episodes of bradycardia but had normal intraventricular conduction with normal or rapid heart rates. The details of the patient's course are presented together with possible mechanisms for the uncommon entity of bradycardia-dependent bundle branch block that she exhibited.

Unlike tachycardia-dependent bundle branch block, bradycardia-dependent bundle branch block is a little known entity. This report documents the occurrence of bradycardia-dependent bundle branch block in a patient with an acute myocardial infarction.

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CASE REPORT

A 78-year-old white woman was admitted to the coronary care unit of the Albany Medical Center Hospital with an acute inferior wall myocardial infarction. She was subsequently readmitted four weeks later with an extension of the same process. On the third hospital day a change in the monitored electrocardiogram was noted. This tracing retrieved from a tape memory loop and those obtained at intervals over the next 70 minutes using modified chest leads (MCL) are shown in Figures 1 and 2. Strips A, B and C were recorded with MCL 1 and strips D, E and F using lead MCL 6.

Complexes 1, 2 and 3 show the pattern that the patient had exhibited during the preceding three days; the QRS was of normal duration and the P-R interval prolonged at 0.32 second. After the third complex, second degree A-V block is evident and is followed by alternating junctional escape and sinus capture beats, all with left bundle branch configuration. Junctional escape beats (4, 6, 8 and 10) occur after an interval of 1.45 to 1.46 second while capture beats (5, 7 and 9) occur with a R-R interval of 1.28 to 1.34 second (Fig 1A and 1B). These irregular R-R intervals deny the initial impression of an idioventricular rhythm and the interpretation of bradycardia-dependent bundle branch block is confirmed in subsequent tracings. Monitoring from MCL 6 reveals that the counterpart of the broad negative complexes in MCL 1 are upright, broad, notched complexes characteristic of left bundle branch block. Three minutes after the intravenous administration of 1 mg of atropine, the sinus rate accelerates with transient return of first degree block and reappearance of normal intraventricular conduction occurs (Fig 1C). With return of first degree A-V block, the first junctional escape beat (16) with a R-R interval of 1.44

![ECG rhythm strip before and after atropine administration.](image-url)
second and the first sinus capture beat (17) with a R-R interval of 1.12 second again reveal a left bundle branch block pattern. The subsequent beat (18) with a R-R interval of 0.85 second exhibits normal intraventricular conduction. Strip D (Fig 2) shows persistence of sinus acceleration following atropine administration but with varying degrees of A-V block. Twenty minutes after the initial dose of atropine, an additional 0.5 mg was administered intravenously. Further slight acceleration of the sinus rate results in the recurrence of 2:1 A-V block with alternate P waves buried in the T waves (Fig 2E). Again lengthening of the R-R interval induces left bundle branch block. With the passage of time, a reduction in the rate of sinus discharge occurs and is followed by an increasing number of consecutively conducted beats (Fig 2F). Normal intraventricular conduction occurs with short R-R intervals of 0.63 second and prolonged conduction with R-R intervals of twice this duration.

During the next two days the patient had an anterior wall myocardial infarction, developed persistent left bundle branch block and died in cardiogenic shock. Postmortem heart examination confirmed the patient's clinical course.

**DISCUSSION**

Conductivity in myocardial tissue is a function of the rate of rise of phase zero of the action potential and is directly related to the magnitude of the resting membrane potential at the onset of the action potential. The rate of rise of the action potential and the rate of conduction are maximal when a cell is completely repolarized. Singer and co-workers have shown that all pacemaker tissues of the heart, including both bundles, exhibit spontaneous depolarization during diastole (phase IV of depolarization). In such cells, therefore, the period of time during which they are completely repolarized is quite transient. At slow rates, the impulse originating in the atria or the A-V node will arrive when this period has elapsed and the membrane potential is progressively decreasing. Such an impulse will result in diminished excitability, while one delayed by impaired A-V conduction may find the bundle completely refractory. This mechanism is considered the most likely explanation of bradycardia-dependent bundle branch block (Fig 3—point A). Similarly, at rapid rates, tachycardia-dependent bundle branch block can be explained utilizing the dependence of conductivity on resting membrane potential before phase III repolarization is completed (Fig 3—point B).

Sarachek invoked the concept of supernormal conduction to explain the occurrence of normal conduction at faster rates in bradycardia-dependent bundle branch
block; excitation occurs at point C (Fig 3) for normally conducted beats. Massumi\(^1\) takes issue with this concept in his fifth criterion for the diagnosis of bradycardia-dependent bundle branch block, "the concept of supernormal conduction must not apply to normally conducted beats."\(^4\) These two conflicting viewpoints center on the definition of supernormal conduction in the diseased heart.\(^5,6\) Both authors use the dependence of conductivity on resting membrane potential in the same manner to explain bradycardia-dependent bundle branch block. Massumi\(^1\) raises the question of digitalis inducing this type of block, but the patient described here did not receive digitalis.

**REFERENCES**


**Cervical Chemodectoma with Extensive Pulmonary Metastases**

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An unusual case of malignant cervical chemodectoma with pulmonary metastasis is presented. The rarity of diffuse visceral spread is emphasized and the distinction from benign pulmonary chemodectomatosi is made by both clinical and pathologic features.

Carotid body chemodectomas (nonchromaffin paragangliomas) were first reported in 1743 by Van Haller. Since then, approximately 500 cases have been described in the literature.\(^1\) The benign or malignant potential of this tumor has been the subject of much controversy and the advisability of resection has been widely debated.\(^2\) It has been estimated that about 5 percent of chemodectomas are clinically malignant and exhibit local invasion. The number of cases of histologically proved distant metastases, however, is extremely small.\(^3\) Because of the rarity of distant visceral metastases, we would like to present our experience with the following case, in which extensive pulmonary metastases were the most prominent clinical and radiologic feature. We believe that this is the first case reported in the literature in which pulmonary metastases caused the presenting symptoms.

**Case Report**

A 20-year-old white woman, previously in excellent health, was admitted to the pulmonary Service of Montefiore Hospital and Medical Center in October, 1971. She had been employed in a local department store, where she demonstrated the use of hair sprays. After approximately six months of such activity, she noticed the onset of a "flu-like" syndrome of general aches and pains and temperature elevations of 101\(^*\) to 102\(^*\) F. An x-ray film of the chest taken at that time was interpreted as abnormal, and she was admitted to another hospital for study. After inconclusive studies that included a scalene node biopsy with negative results and treatment with antibiotics, the patient was transferred to Montefiore Hospital and Medical Center.

Physical examination revealed a young, well-developed, well nourished, white woman in no distress. Her temperature was 101.6\(^*\)F, with a pulse rate of 90 per minute and regular, respiratory rate of 26 per minute and blood pressure of 110/60 mm Hg. The examination of the head, eyes, ears, nose and throat was within normal limits. A 2 x 2 cm mass was felt on the left side of the neck, in the region of the bifurcation of the common carotid artery. A freshly healed scar was present in the right supraclavicular region. The heart sounds were normal, no murmurs were heard; the lungs were clear to percussion and auscultation. The abdomen was soft, not tender and no masses were palpable. There was no organomegaly. The extremities were normal. Pelvic examination was not permitted.

Laboratory studies revealed a hematocrit of 28 percent. The white blood cell count was 9,200, with 67 percent lymphocytes, and 5 percent monocytes. The erythrocyte sedimentation rate was 90 mm in one hour. A bone marrow aspirate showed minimal red cell hypoplasia. The following results of laboratory studies were either negative or within normal limits: urinalysis, fasting blood sugar, blood urea nitrogen, uric acid, serum sodium, potassium, chloride and CO\(_2\) content, calcium, phosphate, protein, albumin, globulin, glutamic oxaloacetic transaminase, lactic acid dehydrogenase, alkaline phosphatase, cholesterol, bilirubin, protein electrophoresis, lupus erythematosus cell preparation, antinuclear antibody titer, direct and indirect Coombs test. Intradermal skin tests to intermediate and second strength purified protein derivative, histoplasmin, coccidioidin and blastomycin were negative. The mumps antigen test was positive. The ECG was within normal limits.

The admitting chest x-ray film revealed multiple nodular densities, varying in size from several millimeters to about 1 cm scattered throughout both lung fields and more marked at the bases (Fig 1).

Pulmonary function tests revealed a vital capacity of 2,100 ml (63 percent); forced expiratory volume in one second (FEV\(_1\)) of 90 percent; FEV\(_2\) of 100 percent; and a single breath diffusion capacity of 11.6 ml/min/mm Hg (48 per-