lessening of inferior wall ischemia. This can be a useful criterion for the interpretation of T-wave changes in the presence of hemiblock associated with ischemic heart disease.

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Intra-His Bundle Block Complicating Acute Inferior Myocardial Infarction*

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The unexpected findings of atrioventricular block or delay in the His bundle and proximal branches are described in a 51-year-old man with acute inferior myocardial infarction with narrow QRS complexes. With the information from the His bundle electrogram, the site of atrioventricular block is precisely localized.

It has been suggested that heart block in inferior myocardial infarction may be due to the effect of increased vagal tone at atrioventricular conduction, to ischemia of the atrioventricular node and His bundle, or to both. In first, second, or complete atrioventricular block with normal-width (<0.10 second) QRS complexes, the conduction delay is generally above the His bundle. Unfortunately the standard electrocardiogram sometimes fails to localize the site of delay or block precisely.

The purpose of this report is to describe the physiologic site of atrioventricular block in a 51-year-old man with acute inferior myocardial infarction with narrow QRS complexes as delineated by recording the His bundle electrogram.

METHOD

His bundle electrograms were obtained in this patient as part of a prospective study of patients with conduction disturbances. The His bundle electrograms (USCI No. 5) were performed under local anesthesia using a bipolar catheter introduced percutaneously via the right femoral vein using conventional techniques. The catheter was positioned across the tricuspid valve under fluoroscopic control and manipulated to achieve optimal recording of the His deflection. All records were obtained on a multichannel-oscilloscope photographic recorder (Electronics for Medicine VR-6) at a paper speed of 100 mm/sec with filter frequencies of 0.1 to 250 Hz for ECG leads and 30 to 250 Hz for electrogram recording. The low right atrium-to-His bundle (LRA-H) potential interval was measured from the onset of the low right atrial depolarization to the first rapid deflection of the His bundle potential. The His-to-ventricle (H-V) interval was measured from the first rapid deflection of the bundle of His potential to the earliest ventricular activity either on surface or intracardiac recording. The right bundle-ventricle (Rb-V) interval was measured from the initial rapid deflection of the first potential after the His deflection to the earliest ventricular activity. When split His bundle potentials were recorded with intact atrioventricular conduction, measurements were made of the LRA-H, H1-H2, and H2-V intervals. Normal range for the LRA-H interval during sinus rhythm in our laboratory is 80 to 150 msec; for the H-V interval, 35 to 55 msec; and for the Rb-V interval, 15 to 29 msec.

CASE REPORT

A 51-year-old man was admitted to the coronary care unit of St. Luke's Episcopal Hospital for acute sudden onset of chest pain of 24 hours' duration. His ECG on admission showed acute inferior myocardial infarction. Three hours after admission, the patient had Wenckebach or Mobitz type 1 atrioventricular block which progressed to transient complete atrioventricular block with a ventricular rate of 40 beats per minute. Atropine was given intravenously with good response. On the second hospital day, the patient's standard ECG demonstrated a sinus rhythm at a rate of 80 beats per minute with a prolonged P-R interval of 0.32 seconds. The QRS complexes were narrow and measured 0.08 seconds. His bundle electrograms on the second hospital day showed two high-frequency potentials recorded between the atrial and ventricular electrograms representing "split" His potentials (Fig 1) as defined by Narula et al. Each atrial depolarization was followed by a biphasic proximal His bundle potential (H1) with LRA-H1 interval of 100 msec, H1-H2 interval of 95 msec, and H2-V interval of 127 msec. In addition, a separate biphasic spike was recorded between the H2 and V, probably representing a right bundle potential of Rb-V in-
The patient had a smooth and uneventful recovery from his myocardial infarct and was discharged with sequelae.

The increase in delay of the bundle branches and the Purkinje system. In keeping with this hypothesis, one would expect a bundle-branch block or wide QRS configuration. Normalization of the QRS configuration (Fig 1) can be explained by a relatively equal degree of conduction delay in both bundles giving rise to a narrow QRS complex with prolonged H-V interval, as shown in this case and in experimental animals by El Sherif et al. In order to explain the long H-V interval, an additional delay has to be postulated in the proximal part of the bundle branches.

The development of unequal delay in either bundle gave rise to a QRS pattern of right bundle-branch block and prolonged H-V interval (Fig 3). Although the H-V interval of 85 msec was shorter than the H-V interval in Figure 1, the H-V interval is prolonged. Critical analysis of Figure 3 suggests that alteration in the His bundle conduction time (H-V interval) was associated with the pattern of right bundle-branch block. We feel that the conduction disorder can, thus, be ascribed to the presence of a longitudinal pathway with asynchronous refactoriness and conduction velocities in the distal bundle. The fact that the H-V interval is prolonged suggests a high degree of block within the His bundle. The increase in delay of H-V conduction provided more asynchronous refactoriness to both bundles.

It is worthwhile to consider, but less likely, that the recording of two different His bundle potentials in the same patient with a lesion within the His bundle potentials is, in fact, the result of a coincidental situation in which such factors as location and extension of the lesion, site of the focus of activating the ventricles, electrode distance, and angle between catheter and His bundle play a role.

From the conventional ECG, it could not be predicted that this conduction defect was localized within the His bundle, as was revealed by the recording of the His bundle electrograms. This case is of interest because it
shows conduction delay within the bundle of His and proximal bundle branches with normal configuration of QRS complexes. Localization of the block, within the atrioventricular node would have been anticipated had only the standard ECG been available. The impulse propagation through the atrioventricular node was normal, as was reflected by the normal LRA-H conduction.

Involvement of the distal conduction system has usually been considered to be uncommon in patients with inferior infarction. The blood supply to most of the human His bundle and its proximal branches is dual in origin. Anatomically, this can best be explained on the basis of transient, but unequal, ischemia of the His bundle selectively inhibiting conduction to the distal His bundle and its proximal branches. The atrioventricular node may be ischemic but survives because of its relatively low oxygen requirement compared to those of the contractile systems. It has also been suggested that release of potassium from the surrounding infarct tissue or of lysosomal enzymes from the polymorphonuclear leukocytic infiltrate may also play a part.

ACKNOWLEDGMENT: We wish to thank Miss Joyce Childress for her help in manuscript preparation.

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Unusual Roentgenographic Manifestation of Pneumocystis carinii Pneumonia

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An immunologically deficient patient had two large nodular densities on the chest x-ray film. On autopsy, large solid accumulations and diffuse pneumonitis with Pneumocystis carinii were found. This represents an unusual manifestation of Pneumocystis carinii.

Pneumocystis carinii pneumonia is an infection that becomes apparent clinically in patients with impaired host defenses. Roentgenographic abnormalities may precede symptoms by months and usually consist of soft infiltrates spreading from the hilum to the base and later to the entire lung. Rarely, small nodular densities have been reported. It is the purpose of this presentation to report a case of Pneumocystis carinii infection manifested as diffuse pneumonitis and solid accumulation of the organism. This is an unusual radiographic manifestation of the organism.

CASE REPORT

This 28-year-old woman had systemic lupus erythematosus diagnosed at the University District Hospital in 1974. Since the patient had severe renal impairment, she began to receive hemodialysis three times weekly. The patient was being observed while receiving 100 mg of cyclophosphamide (Cytoxan) and 50 mg of prednisone daily.

Twelve hours prior to admission, she developed severe pleuritic chest pain accompanied by shortness of breath, fever, and palpitations. There was no history of cough or hemoptysis. Upon admission the patient had blood pressure of 150/70 mm Hg, pulse 120 beats per minute, temperature 37.7°C (99.9°F) and a respiration rate of 26/min. Findings from physical examination were unremarkable, except for a prominent butterfly rash and an arteriovenous fistula in the left thigh, which was surgically induced to be used for hemodialysis. Cyclophosphamide therapy was discontinued. The patient had marked leukopenia and thrombocytopenia, and a bone-marrow aspiration showed a reactive marrow. The findings from a lung scan and chest x-ray film were normal.

A reactivation of the systemic lupus erythematosus was considered, and the prednisone dosage was increased to 100 mg daily. The high fever, pleuritic chest pain, and palpitations persisted. Therapy with 100 mg of gentamicin sulfate (Garamycin) injected intramuscularly every 72 hours was started. Blood, sputum, and urine cultures for fungi and aerobic and anaerobic organisms were reported as negative.

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CHEST, 69: 3, MARCH, 1976