cessful management. Cineangiography was invaluable in establishing that aortic regurgitation was not secondary to progression of the previous dissecting aneurysm, and that the present lesion could be dealt with independently. This patient demonstrates that early cardiac catheterization and cineangiography should be performed in order to establish the definitive diagnosis in any patient with blunt chest injury who develops a cardiac murmur, a widened pulse pressure, evidence of congestive heart failure, or who gives historic data suggestive of any change in cardiac status. Because a normal heart poorly tolerates acute left sided valvular lesions, early operative repair based on accurate demonstration of the pathologic anatomy will increase the chance of survival of such patients.

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REFERENCES

Antegrade Concealed Conduction to the His Bundle; Documentation with His Bundle Recording*

Ruben Chuquimia, M.D.; ** William Toune, M.D.; † Hamnent Deshmukh, M.D.; ‡ and Kenneth M. Rosen, M.D. || F.C.C.P.

*From the Department of Cardiology, Cook County Hospital, and Section of Cardiology, Abraham Lincoln School of Medicine, University of Illinois College of Medicine, Chicago, Ill. Supported in part by NIH contract 71-5478 under the Myocardial Infarction Program, National Heart and Lung Institutes, National Institutes of Health, Department of Health, Education and Welfare. **Attending Physician, Cook County Hospital; Associate in Medicine, Abraham Lincoln School of Medicine, University of Illinois College of Medicine, Chicago, Ill. †Director, Adult Cardiology, Cook County Hospital; Assistant Professor, Loyola University, Chicago, Ill. ‡Fellow in Cardiology, Cook County Hospital, Chicago, Ill. ||Chief of Medicine, Cardiology Section, Abraham Lincoln School of Medicine, University of Illinois College of Medicine, Chicago, Ill. Reprint requests: Dr. Rosen, Cardiology Section, PO Box 6999, Chicago 60680

A patient had blocked interpolated premature atrial contractions, followed by P-R prolongation in the subsequent cycle, suggesting antegrade concealed conduction to the A-V node. His bundle recordings revealed this interpretation to be erroneous. The atrial premature beats were blocked distal to the H recording site. The P-R prolongation in the subsequent beat reflected delay in the His bundle ("splitting" of H potentials). This was thus an example of antegrade concealed conduction to the His bundle, a new manifestation of concealed conduction.

Concealed conduction is defined as the effect of a partially penetrating impulse on the conduction of a subsequent impulse. Examples of concealed conduction include the P-R prolongation following an interpolated premature ventricular contraction (PVC) and the P-R prolongation following a blocked interpolated atrial premature contraction (PAC). These are generally considered examples of retrograde and antegrade concealed conduction to the A-V node respectively.

In this report, we present recordings from a patient who had blocked interpolated premature atrial contractions (PAC) which were following by P-R prolongation. To our surprise, His bundle electrograms revealed the PAC's to be blocked distal to the His bundle recording site, with subsequent P-R prolongation reflecting splitting of the His potential (delay in the His bundle). This is a new manifestation of concealed conduction, reflecting partial penetration of an antegrade impulse to the His bundle.

CASE REPORT

The patient was a 15-year-old girl admitted to Cook County Hospital for evaluation of palpitation and chest pain. Electrocardiograms were within normal limits except for the presence of arrhythmia. There were multiple premature atrial contractions, both conducted (Fig 1A) and blocked (Fig 1B). Occasional blocked premature atrial contractions were interpolated and followed by P-R prolongation (Fig 1C). Prior to electrophysiologic study, the P-R prolongation following blocked interpolated PACs was felt to reflect antegrade concealed conduction to the A-V node.

Electrophysiologic Studies

His bundle electrograms were recorded utilizing previously described catheter techniques. High right atrial electrograms were recorded with a bipolar catheter placed in the high right atrium.

The patient was in sinus rhythm with an atrial rate of 80 to 85/min. A-H interval was 75 msec (normal 54-130 msec) and H-V interval was 35 msec (normal 31-55 msec). There were multiple premature atrial contractions with coupling intervals (measured from the preceding sinus P wave) of 520 msec to 370 msec (Fig 2A and 2C). Atrial premature beats with coupling intervals of 400 msec or less (H-H intervals of 460 msec or less) were blocked distal to the H recording site (Fig 2B). Atrial premature beats with coupling intervals of 370 msec were blocked distal to the H recording site and interpolated. Following the blocked interpolated contraction, there was P-R prolongation reflecting "splitting"
Most examples of concealed conduction reflect partial penetration to the A-V node. Typical examples of this phenomenon would include the P-R prolongation following either a blocked atrial or interpolated ventricular premature contraction, the compensatory pause following a premature ventricular beat in atrial fibrillation, and the resetting of a junctional pacemaker following retrograde penetration of a ventricular premature beat in a patient with A-V dissociation.

Experimental studies in animal hearts, utilizing recording of both tissue electrograms as well as cellular action potentials, have demonstrated that most premature impulses are concealed within the A-V node. However, premature atrial impulses may also be concealed within the His-Purkinje system. Long cycle lengths predispose to concealment within the latter system, since His-Purkinje system refractory periods are markedly cycle length dependent. At long cycle lengths, a premature impulse may be able to traverse the A-V node, and encounter portions of the His-Purkinje system which are refractory.

The His bundle recording technique has been useful in delineating concealed conduction in man. Damato and Lau presented examples of A-H prolongation following blocked atrial premature beats and interpolated ventricular premature beats, thus documenting antegrade and retrograde concealed conduction to the A-V node. Other examples of concealed conduction documented with the His bundle recording technique include pseudo A-V block (at the A-V node) secondary to the conduction or formation of subsequent impulses.

**DISCUSSION**

The term concealed conduction describes the effects of a partially penetrating cardiac impulse disturbing of H potentials (Fig 2C). This reflected antegrade concealed conduction of the blocked interpolated PAC to the His bundle resulting in His bundle delay in the subsequent cycle.

**FIGURE 1:** Rhythm strips (lead 2). Panel A: Conducted PAC with slight aberrant conduction. Note deformation of the second T wave due to PAC. Panel B: Blocked PAC superimposed on second T wave. Panel C: Blocked interpolated PAC (superimposed on second T wave) followed by P-R prolongation suggesting antegrade concealed conduction to A-V node.

**FIGURE 2:** His bundle electrograms demonstrating concealed conduction to His bundle. Lead V1, His bundle electrogram (HBE) and high right atrial electrograms (AE) are shown in each panel. High right atrial electrogram of sinus beat is labeled A; atrial premature contraction labeled Ax, H potentials are labeled H. Coupling interval between A and Ax are listed under AE. Coupling intervals (H-H) between conducted H potential of sinus beat and H potential of APC are listed under the HBE. Panel A: Conducted APC with aberrant conduction. Panel B: APC blocked distal to H recording site. Panel C: APC blocked distal to H recording site. APC is interpolated. Note: P-R prolongation in subsequent beat reflects splitting of H potentials. Second H potential is labeled H'.
concealed His bundle depolarizations, and repetitive block in both the A-V node and His-Purkinje system during Wenckebach periods.5,10

The present case is a new manifestation of concealed conduction. Atrial premature beats were blocked distal to the His bundle recording site. The actual anatomic site of block could not be delineated but was within the His bundle (in or distal to the H recording site) or within both bundle branches. When these blocked atrial premature beats were interpolated, the next conducted beat was conducted with “splitting” of H potentials producing P-R prolongation.

Splitting of H potentials, the recording of two high frequency His bundle electrograms separated by a variable interval, was reported by Narula11 as suggestive of disease within the His bundle. Subsequent pathologic studies by Bharati and co-workers12 have confirmed this, by demonstrating pathologic lesions in the His bundle in patients with split H potentials. However, split H potentials do not have to reflect pathologic changes in the His bundle. Schuilenburg and Durrer13 and also Wu et al14 have reported that functional block may be produced within the His bundle with coupled extrastimuli, resulting in H potential splitting. This type of functional block would be most likely in a patient with a short A-V nodal functional refractory period, allowing consecutive impulses with short coupling intervals to enter the His bundle. The splitting of H potentials in the present case, does not necessarily reflect a diseased conduction system.

The surface electrocardiogram in the present case was misleading, since it suggested antegrade concealed conduction to the A-V node. The demonstration of concealed conduction to the His bundle, could only be documented with recording of His bundle electrograms. We predict that many other examples of both antegrade and retrograde concealed conduction to the His bundle will be found.

REFERENCES


Myocardial Sarcoidosis Presenting as Acute Mitral Insufficiency*

S. Zoneraich, M.D.;** M. P. Gupta, M.D.;† J. Mehta, M.D.;‡ O. Zoneraich, M.D.;§ and Z. Wessely, M.D.||

A young patient with acute mitral insufficiency with a rapid and fatal course is presented. On autopsy, diffuse sarcoid granulomatous infiltration was found in the anterior and posterior walls of the left ventricle, in the interventricular septum and in both papillary muscles. Sarcoidosis as a cause of valvular involvement should be considered in patients with the sudden appearance of mitral incompetence, conduction defects and arrhythmias.

Sarcoidosis is a disease of unknown etiology involving practically every organ of the body. The incidence of myocardial sarcoidosis is about 20 percent of patients with this disease1 and 0.025 percent of total autopsies as reported by Bashour et al.2 The diagnosis is very often missed antemortem because of sudden death and other fatal cardiac arrhythmias and conduction disturbances.3,4 There are several case reports of sarcoidosis involving the heart in the English literature. However, to the best of our knowledge, only one case with acute mitral insufficiency has been attributed to papillary muscle dysfunction secondary to infiltration by sarcoid granulomas seen on autopsy.4

Here we present the case of a young woman who had a brief cardiac history, severe mitral incompetence and suddenly died. The autopsy revealed several foci of noncaseating granuloma in the papillary muscles and in the left ventricular wall.

*From the Departments of Medicine and Pathology, Division of Cardiology, Queens Hospital Center, Long Island Jewish-Hillside Medical Center Affiliation, Clinical Campus of the School of Medicine, State University of New York, Medical School at Stony Brook, Stony Brook, NY.
**Associate Professor of Medicine; Head, Division of Cardiology.
†Assistant Professor of Medicine and Physician-in-Charge, Cardiac Catheterization Unit.
§Fellow in Cardiology.
||Associate Professor of Medicine.
|Associate Professor of Pathology, Associate Director, Department of Pathology.

Reprint requests: Dr. Zoneraich, Long Island Jewish-Hillside Medical Center, Jamaica, New York 11432.