Concealed Conduction: An Indication for Temporary Demand Pacing*

L. P. McLaurin, Capt, USAF, (MC) and John F. Moran, Major, USAF, (MC)

A patient with inferior wall myocardial infarction and type I second degree atrioventricular block is presented. This patient developed long symptomatic ventricular cycles caused by concealed conduction of some atrial impulses. He was successfully treated with temporary demand pacing and digitalis.

Although cardiac pacemakers have a proved place in the treatment of chronic heart block, there have been some differences of opinion regarding the efficacy of artificial pacing in heart block complicating acute myocardial infarction. Second degree atrioventricular (A-V) block can be divided into type I (Wenckebach) and type II (Mobitz). The former is the type usually associated with inferior myocardial infarction and has a better prognosis than type II which is most often associated with anterior infarction. Since type II A-V block usually results from extensive damage to the bundle branches, complete heart block with a slow, ineffective pacemaker focus may result, and artificial pacing must be immediately available. This is contrast to type I A-V block which is usually due to ischemia of the A-V node and is generally transient. Although complete heart block may occur in this situation, the pacemaker focus is usually located high in the junctional tissue and the rhythm is better regarded clinically as a form of atrioventricular dissociation rather than complete heart block. Since this latter rhythm disturbance is almost always reversible, artificial pacing is not needed as long as the ventricular rate remains above 50 beats per minute. The site of the infarction, the rate of the subsidiary pacing focus, and the clinical condition of the patient must be weighed against the increased risk of pacemaker insertion in the patient seriously ill with an acute myocardial infarction.

Herein is reported a patient with acute myocardial infarction who developed second degree A-V block of type I (Wenckebach). He tolerated this well until concealed A-V conduction of some atrial impulses resulted in clinically symptomatic pauses in ventricular activity. Temporary artificial demand pacing prevented these pauses and led to symptomatic improvement. The unusual characteristics of this patient's electrocardiograms and the successful intervention with an artificial pacemaker form the basis for this report.

**Case Report**

A 46-year-old man with two previous myocardial infarctions and chronic congestive heart failure was hospitalized with chest pain. An admission electrocardiogram showed characteristic changes of an acute inferior myocardial infarction with first degree A-V block. Three days later he developed second degree A-V block with Wenckebach cycles that varied from 9.8 to 21.1. The following day, nonconducted atrial premature beats causing long pauses were noted. The patient became diaphoretic, moderately hypotensive and complained of chest pain. A transvenous pacing catheter was positioned in the apex of the right ventricle. The patient was paced on a demand basis for the next 24 hours at which time he reverted to first degree heart block followed by sinus rhythm. The pacing catheter was removed and the patient continued to improve.

**Electrocardiograms**

The first rhythm strip (Fig 1) shows A-V dissociation with occasional sinus capture beats. The junctional escape interval is 0.90 sec, the same as the R-R interval during continuous junctional rhythm. The second strip (Fig 2) recorded soon after Figure 1 shows second degree A-V block with 3:2 Wenckebach cycles. There are two long pauses caused by ectopic premature atrial impulses which do not result in ventricular activation. A junctional escape beat does not come after the expected interval suggesting that the junctional pacemaker was affected by the atrial premature beat.

If no atrial premature beat occurred, there was no long cycle, and instead the 3:2 Wenckebach pattern resumed. The ventricular pauses associated with the dropped beat of the 3:2 Wenckebach cycles were slightly longer than the junctional escape interval of 0.90 sec (Fig 2 and 3). Since no junctional escape beat was seen, it is likely that the nonconducted sinus beat of the 3:2 cycle also affected the junctional pacemaker in a concealed fashion. In Figure 2 the beat that terminates the first long R-R cycle has a PR interval of 0.23

---

*From the Department of Medicine, David Grant USAF Medical Center, Travis AFB, California.

The opinions expressed are those of the authors and do reflect official Air Force policy.

Reprint requests: Dr. John F. Moran, Section of Cardiology, Loyola University Medical Center, 2160 South First Avenue, Maywood, Illinois 60153.

![Figure 1](https://example.com/figure1.png)

**Figure 1.** This rhythm strip shows atrioventricular dissociation with sinus captures having a PR interval of 0.27 sec. Most of the beats are junctional in origin with a cycle length of 0.90 sec.
sec. Later, however, the beats that terminated the long R-R cycles had a shorter PR interval of 0.20 sec. In those beats with the shorter but consistent PR intervals, there is aberration of the QRS complex and slight shortening of the long R-R cycles down to 1.40 sec. As shown in Figure 3 recorded two minutes later, this pattern of group beating became repetitive, slowed the ventricular rate significantly, and resulted in the symptoms described above.

**DISCUSSION**

Concealed conduction, a term introduced by Langendorf,\(^7\) refers to the partial penetration of the specialized conducting system by an impulse that exerts an electrophysiologic effect but fails to emerge. It can be inferred from the clinical electrocardiogram by analysis of changes in subsequent events. Changes that may occur as a result of concealed A-V conduction include: (1) blocking of a subsequent atrial impulse which has occurred at a time when the conducting system should be excitable; (2) failure of a subsidiary pacemaker to discharge at the anticipated time; (3) delay in conduction of the succeeding impulse, and (4) enhancement of conduction of a successive impulse.\(^8\)

Impulses may enter the A-V junction and fail to emerge because of decrement of their action potential or because of elevation in threshold of the conducting tissue encountered by the impulse.\(^9\) Either of these phenomena may be the result of partially refractory conducting tissue. Conditions for such a situation may be found wherever there is a junction between two fibers that differ in action potential duration.\(^10\) Anatomically, this might occur at the atrio-nodal junction, within the atrioventricular node-His bundle-Purkinje system, or at the junction of the bundle branches and the ventricular myocardium.

Our patient tolerated his second degree block of type I (Wenckebach) until blocked premature atrial beats began to appear, and his ventricular rate decreased. Evidence for concealed conduction of the premature atrial impulses can be inferred from the absence of a junctional escape beat at the anticipated time. A junctional escape beat would have appeared if the atrial premature beat had not penetrated to the junctional pacemaker and discharged it, thus preventing the junctional escape beat from occurring.

The PR intervals of the beats that terminate the long R-R cycles in the first patient are somewhat shorter (0.04 to 0.05 sec) than the PR intervals of the first beats of the Wenckebach cycles. This could occur if A-V conduction took place during a supernormal phase induced by the preceding premature atrial beat. It is also possible that early excitation of the A-V nodal tissue by the premature atrial beat could have promoted early recovery of the A-V node, as recently described by Moe et al.\(^11\) This would also result in a decreased conduction time. That this beat was an idioventricular escape beat seems less likely in the presence of a consistent PR interval seen over many cycles.

If these beats are assumed to be conducted from the atrium, then this patient manifested aberrant ventricular conduction in many of the beats that terminated the long R-R cycles (Fig 3). Since the specialized ventricular conduction system should have been excitable at this time, the aberrant ventricular conduction could have resulted from concealed conduction of the atrial premature beats into the bundle branch system. Concealed
conduction into the His-Purkinje system has been demonstrated experimentally in animals and alluded to in humans. Cohen and colleagues, using the His bundle electrogram technique, have recently documented a case in which an artificially induced atrial impulse was conducted in a concealed fashion to a level below the His bundle. Bradycardia dependent bundle branch block cannot be ruled out. Since the blocked atrial premature beat reached the junctional pacemaker and discharged it, it seems likely that the impulse also reached the bundle branches causing aberrant ventricular conduction of the next impulse.

Demand pacing prevented the long ventricular pauses by artificially supplying an adequate "escape" rhythm. Digitalis which had been considered contraindicated was given after the initiation of pacing, and his symptoms of congestive heart failure improved. He subsequently returned to sinus rhythm, pacing was discontinued, and he survived.

REFERENCES


Editorial Expression

The vigilance of the coronary care unit personnel prevented a possible sudden death through the recognition of the concealed conduction defect of atrial impulses in the setting of an acute inferior myocardial infarction—a setting in which high nodal ischemia is usually temporary and serious bradycardia is not anticipated.

The authors are congratulated for renewing an awareness of the importance of concealed conduction defects and the need for temporary demand pacing.

George C. Griffith, M.D.
La Canada, California

Chronic Recurrent Tracheoesophageal Fistula

Joseph C. Kiser, M.D., Theodore A. Peterson, M.D., and Frank E. Johnson, M.D.

The management of two patients with reccomendation following congenital tracheoesophageal fistula repair is presented. Complete repair as a secondary operation can be accomplished successfully. The difficulties of diagnosis as well as the technical aspects of secondary repair are emphasized.

With increasing experience, the operative mortality rates for repair of congenital tracheoesophageal fistula have steadily fallen and the methods of treatment have markedly improved. In the literature there are many excellent reviews of the subject both in the form of monographs and reports of series. Although recurrent tracheoesophageal fistula is uncommon, its occurrence has continued to result in a high mortality. On occasion, the recurrent fistula has become chronic allowing for a carefully planned definitive surgical procedure as indicated in the following two case reports.

CASE REPORTS

Case 1

The patient is a 21-year-old white man. His initial surgery was on the third day of life (March, 1946). Gastrostomy followed by primary repair of a congenital tracheoesophageal fistula was performed. His early postoperative course was complicated, requiring multiple dilatations. He was discharged ten weeks following the initial procedure. In May,