Early and Late Atrioventricular Block in Acute Inferior Myocardial Infarction

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In a group of 288 patients with acute inferior (diaphragmatic) myocardial infarction, second and third degree atrioventricular (AV) block was diagnosed in 37 (14%). Three of the 37 died. The AV block in the 34 survivors could be differentiated into two distinct types, namely, early and late AV block.

In 15 patients, second and third degree AV block developed within 6 hours of the first signs of infarction. In these 15 patients, all signs of AV block disappeared within 24 hours after infarction. Second and third degree AV block appeared suddenly in the vast majority, and first degree AV block could be detected in only a few patients and for a very short time before normalization of conduction. Atropine either abolished AV block completely or caused a marked acceleration of ventricular escape rhythm.

In 14 patients, second and third degree AV block developed later than 6 hours (in 12 later than 24 hours) after infarction. It was heralded and followed by relatively long periods of first degree AV block in all cases (except in two patients who were admitted 72 hours after infarction). The total duration of AV block was longer than 40 hours in all of these patients, and the ventricular rate was relatively high. In no patient was abolishment of AV block achieved by atropine, and ventricular acceleration was relatively slight in all. In five patients, early and late AV block could be recognized consecutively.

The two types of AV block seem to have different causes. Increased vagal tone is probably operative in the first type, and metabolic changes due to ischemia in the second. Response to atropine and sympathomimetic drugs is much better, and cardiac pacing only rarely indicated, in patients with early than in those with late AV block.

Atroventricular (AV) block is a common complication of acute inferior myocardial infarction, occurring in 9 to 33% of patients (1–6). Its time of onset after the first signs of acute infarction is reported to range from 30 minutes to 7 days (3,4,6–9). Block of very early onset is usually of short duration and its response to the administration of atropine is much more dramatic than that of late onset block (8,10,11).

We examined the course of AV block complicating acute inferior myocardial infarction. Our purpose was to determine whether different clinical cases exist and, accordingly, if AV block complicating acute inferior myocardial infarction can be separated into distinct types.

Methods

Patients. Two hundred eighty-eight patients with acute inferior myocardial infarction were admitted to the coronary care unit of this hospital from January 1, 1978 to April 30, 1982. Atrioventricular (AV) block developed in 40 patients (14%). Six of these were excluded from the study because three died within 72 hours of admission so that no late hospital course could be studied, and in the other three only first degree AV block developed (of late appearance and long duration) so that no "dynamic process" could be ascertained. Thirty-four patients, 28 men and 6 women ranging in age from 45 to 79 years (mean 60.5), were studied prospectively once AV block was identified. Acute inferior myocardial infarction was defined according to the criteria of the New York Heart Association (12). The time of onset of the first signs of infarction was recorded on admission according to the patient’s or accompanying person’s statement.

Clinical management. Medical treatment consisted of an initial injection of atropine (0.5 to 1.0 mg) intravenously, which was changed to isoproterenol infusion if a slow ventricular rate persisted. Treatment was given whenever the heart rate was below 50 beats/min or if bradycardia of lesser degree caused hypotension. In patients in whom isoproterenol infusion had to be sustained (usually longer than 1 hour), a temporary pacemaker was introduced. Patients were monitored continuously for at least 4 days. If AV block persisted for more than 4 days monitoring was continued...
until 48 hours after normal AV conduction was established. Electrocardiographic rhythm strips were recorded hourly. In patients with a pacemaker, the activity of the pacemaker was diminished gradually until a stable spontaneous rhythm could be recorded.

Results

The 34 patients were classified into groups on the basis of the course of the AV block during hospitalization. Second or third degree AV block appearing within 6 hours of infarction defined 15 patients in Group 1. Group 2 included 14 patients with high grade AV block appearing after the first 6 hours. In five patients, high degree AV block appeared within 6 hours and disappeared only to reappear in a late phase of the hospitalization period; they made up Group 3.

Group 1 (early block). Fifteen patients (male:female ratio = 12:3, age 62.4 ± 10.2 years) had initial evidence of high degree AV block within 6 hours of infarction. In 11 second degree (4 patients) or third degree (7 patients) AV block was present on admission, usually within 1 hour but not later than 3 hours after the first symptoms of infarction. In 8 of these 11 patients, a history of one or more syncopal attacks before admission was elicited. The lowest ventricular rate encountered in this group ranged from 28 to 60 beats/min (mean 37.9 ± 8.9). The total duration of the AV block in the group was 6.1 ± 5.8 hours.

Atropine injection led to complete restoration of normal conduction in three patients and an acceleration of 21 to 45 beats/min (mean 28) in the others. In five other patients, normalization of conduction occurred within 1 hour of isoproterenol infusion. In the remaining seven patients, a temporary pacemaker was introduced. Normal conduction was established in all patients in Group 1 within 20 hours of the first signs of infarction. Short periods of first degree AV block were identified in two patients before and in four patients after the occurrence of higher degrees of AV block (Fig. 1).

Group 2 (late block). In 14 patients (male:female ratio = 12:2, age 61.1 ± 9.0 years), high degree AV block developed later than 6 hours after the first symptoms of infarction. In 12 patients, a period of stable first degree AV block anteceded, followed by high grade block. The two patients in whom first degree AV block was not observed before a higher degree of block appeared were admitted approximately 72 hours after infarction because of syncope. First degree AV block was noted on admission (in three patients) or within 24 to 75 hours (mean 61.2) in the remaining patients. The lowest encountered ventricular rate in this group was in the range of 27 to 70 beats/min (mean 50.9 ± 2.1). The total duration of the AV block in this group was 114.9 ± 45.4 hours.

Atropine and isoproterenol were given to eight patients. Injection of atropine was followed by an increase in heart rate of 10 to 35 beats/min (mean 16). In no patient did the block disappear after drug administration. A temporary pacemaker was inserted in seven patients.

Group 3 (early and late block). In five patients both early and late types of AV block were observed. In four patients (male:female ratio = 4:1, age 65 ± 4 years), either second (one patient) or third degree (four patients) AV block was present on admission. In the fifth patient, the block appeared approximately 6 hours after the onset of symptoms. The lowest ventricular rate recorded when early block was present was 38.8 ± 7.3 beats/min. The early block lasted for only 2 to 4 hours in all but one patient in whom it lasted for 20 hours. Between 49 to 88 hours after admission, high grade (second degree in two and third degree block in three) AV block recurred, with the lowest en-
countered ventricular rate between 44 to 70 beats/min (mean 52.4 ± 1.4) lasting for about 3 to 7 days.

During the early block phase, spontaneous normalization of conduction occurred in one patient, and atropine was administered in the other four. Complete restoration of normal AV conduction occurred in two of these four patients, while first degree AV block persisted in the other two patients. The administration of either atropine or isoproterenol during the late block phase, though accelerating ventricular rate and sometimes lowering the degree of AV block, did not cause complete disappearance of block in any patient in this group (Fig. 1).

Discussion

Clinical types of postinfarction AV block. Two distinctive types of atrioventricular (AV) block could be differentiated in our patients with acute inferior myocardial infarction. A third type, combining the features of the first two types, was also identified. Thus, patients in whom AV block complicated acute inferior myocardial infarction could be separated into three groups.

In Group 1, second or third degree AV block was either present on admission or developed within 6 hours after the first symptoms of infarction. Ventricular rate was usually very low, and restoration to normal conduction was accomplished within a short time. This happened in three patients immediately after atropine administration. The appearance of high degree AV block was abrupt or followed a very brief period of first degree AV block.

In Group 2, high grade AV block developed later than 6 hours after infarction and the block developed gradually in all but two patients. Regression of the block was also slow and gradual, with relatively long periods of stable first degree AV block before normal AV conduction was reestablished. Ventricular rates during the high degree block were relatively high, and the block lasted for more than 3 days in most patients. The response to atropine was much less pronounced than in Group 1, and in no patient did it completely abolish the block.

In Group 3, the features of AV block were almost identical to those described in Group 1, but after regression either to completely normal conduction or to first degree block, second or third degree AV block reappeared and its course was identical to that recorded for patients in Group 2.

Previously reported studies. Julian et al. (8) noted that in 7 of 10 patients with second degree AV block complicating acute inferior myocardial infarction, the block developed on the first day, and in all of these it lasted only minutes to a few hours. In the remaining three patients, it occurred on the second, third and sixth day, respectively, and lasted a day or more. Third degree AV block occurred in seven of eight patients on the first day and lasted a few minutes to 48 hours. Webb et al. (10) reported on eight patients developing complete AV block within 30 minutes after infarction, but did not state the exact location. Atropine was given to these eight patients, and normal sinus rhythm was restored in five, in two additional patients, normal sinus rhythm was present after 2 hours. In another report by Adgey et al. (13), AV block developed in 17 patients (6% of their group with acute myocardial infarction) within the first hour and in 11 (4%) later. Sclarovsky and Agmon (11) recorded AV block in 31 patients with acute inferior myocardial infarction during the hyperacute and acute phases. In 14 of the 31, the block appeared within the first hours after infarction and disappeared within 1 hour.

Differentiation of AV block in acute inferior myocardial infarction into two distinct types was not recognized in many reports dealing with the subject (1–6). This may be explained by the high number of patients (possibly more than in our series) with the characteristics of our Group 3, in which the short period of near or complete normalization of conduction may pass unnoticed.

Mechanisms of early versus late AV block after infarction. Early block may be caused by increased parasympathetic tone. Webb et al. (10) observed that parasympathetic overactivity was present in 55% of their patients with acute myocardial infarction (exact location not stated). In 10% (eight patients), this increased parasympathetic tone was manifested by complete AV block. A high response rate to atropine supports this hypothesis (10,14). In our patients we frequently observed disappearance of AV block after the administration of either a cholinergic blocking agent (atropine) or a sympathetic stimulant (isoproterenol). Another possibility is that sudden ischemia due to coronary occlusion may cause AV block and the opening of the collateral circulation may abolish it. In light of the common blood supply (the right coronary artery) of the AV node and the inferior wall of the left ventricle in the majority of patients and the existence of collateral circulation to the AV node (15–17), this assumption provides a better explanation for the occurrence of this kind of block only in patients with inferior infarction.

Late AV block seems to be a consequence of ischemia causing metabolic alteration within the AV node. This may be due to injury of the cell membrane followed by electrolyte derangement, or possibly due to acidosis secondary to relative ischemia of the AV node.

The time of admission after infarction was significantly earlier in Group 1 than in Group 2, probably because patients in Group 1 had more severe symptoms as a result of the coexistence of infarction and block. The possibility that early bradyarrhythmias were undetected in Group 2 patients because of their relatively late admission cannot be definitely ruled out; however, there were no signs to support it.

Treatment. Management of the two groups should, in our opinion, follow different pathways. In the group with
early AV block, which has a high response rate to pharmacologic treatment and a short duration, atropine and isoproterenol are the treatment of choice. Cardiac pacing can be withheld and its use seems to be necessary only rarely. In patients in Group 2 (including, of course, patients in Group 3 in the second stage of AV block) whose bradycardia endangers the circulatory state, a pacemaker should be preferred because of these patients’ relatively poor response to heart rate-accelerating drugs and the longer duration of their block.

References