SUMMARY Five patients with acute posterior myocardial infarction developed varying degrees of atrioventricular (A-V) block prior to rupture of their interventricular septums (IVS). In three of these five the time of septal rupture coincided with the resumption of conducted sinus rhythm. During the period of A-V block, the only stable escape rhythm appeared to originate in the A-V junctional region, and in two patients early in their course there was evidence of enhanced A-V junctional automaticity. Although the A-V node contained extensive infarction in all five hearts, its distal part, and the entire His bundle and proximal branches appeared to be spared. The anatomical pattern of dissection within the ruptured IVS was similar in three hearts, extending upward to the crest of the IVS and penetrating into the interial septum in two of these. Multiple major coronary narrowings were present in all five hearts, especially of a dominating right coronary artery; recent thrombosis was present in the right coronary artery in four of the five cases. The A-V node artery was markedly narrowed by focal fibromuscular dysplasia in three hearts, compounding the ischemia for the A-V node and eliminating one important source of collateral circulation to the interventricular septum.

CORONARY THROMBOSIS AND MYOCARDIAL INFARCTION can cause sudden death in a number of different ways, but rupture of the interventricular septum is seldom one of them. Although a ruptured septum can lead to sudden death, it more often causes the onset or abrupt worsening of congestive failure which the patient usually survives for several days or longer.1 2 Furthermore, some of the possible causes of sudden death during rupture of the interventricular septum such as heart block or arrhythmias are just as often seen during infarction of the septum without its perforation. In fact, when one considers the blood supply of the human interventricular septum,3 it is surprising that heart block is not an even more frequent complication of septal infarction (with or without rupture) than it is. In the most recent clinicopathologic correlation for this series in CIRCULATION, Vlodaver and Edwards4 reviewed their experience with 98 examples of rupture of some portion of the left ventricle complicating acute myocardial infarction. Atrioventricular (A-V) conduction disturbances were observed in 36% of their 17 cases with isolated rupture of the interventricular septum who had clinical observations (one did not). They found antero septal infarction associated with septal rupture in exactly half of the cases. While most previous investigators have emphasized a preponderance of anterior infarcts with ruptured septum,5 6 7 other observers have found more posterior infarcts.6 8 There is likewise some difference in the reported incidence of recent coronary thrombosis in association with septal rupture, an association which Wessler, Zoll, and Schlesinger9 considered to be ubiquitous and an essential feature of the pathogenesis. Some of these differences in findings are simply attributable to differences in investigative interest. For example, exact determination of the type and location of histopathological lesions in the coronary arteries requires special attention and examination. Similarly, transient disturbances in cardiac rhythm or conduction are more apt to be documented when careful continuous electrocardiographic monitoring is done than when it is not.

Whatever the incidence of posterior infarction or heart block may be in all subjects who suffer rupture of the interventricular septum, there is much to be learned from a careful examination of the pathogenesis of each of these events when they may coexist. The present report describes the clinical course and pathological findings of five patients who died after rupture of the interventricular septum due to myocardial infarction. In every one of these there was heart block and the myocardial infarction was posterior in location by electrocardiographic definition. Special attention was directed to the nature and location of the coronary obstructive lesions, and to histological abnormalities in the cardiac conduction system.

Case Reports

Case 1

A 69-year-old retired candle salesman developed chest pain about three months after the onset of gradually increasing dyspnea. On his admission to the hospital the substernal pain lasted several hours at a time. Although known to be normal in the past, his blood pressure shortly after admission was found to be 180/120 mm Hg. He was sweating profusely and complained of being sleepy with excessive yawning. An electrocardiogram made several hours after admission showed an acute posterior myocardial infarction with normal sinus rhythm. A white blood cell count was 18,000/cu mm with 81% neutrophils. Several observers found the cardiac rhythm to be "regular" after the time of admission, but an ECG on the third hospital day demonstrated dissociation of sinus rhythm and a slightly slower A-V junctional rhythm (fig. 1). On the fourth hospital day a new loud systolic murmur appeared just to the left of the sternum, the blood pressure fell to 98/80 mm Hg and the patient's general condition deteriorated. He died the day after the onset of the new murmur.
The ECG of case 1 shows an acute posterior myocardial infarction and A-V dissociation, with the A-V junctional rhythm being slightly slower than the sinus rhythm. Short periods may represent transient sinus capture, as near the end of leads I and III, where ventricular cycle lengths become shorter.

At postmortem examination the major abnormalities were those found in the heart and the changes caused by recent congestive failure. An acute posterior myocardial infarction was obvious on gross examination, and a 2 cm perforation was present within infarcted posterior interventricular septal myocardium, opening into the right ventricle about 3 cm below the level of the coronary sinus. The right coronary artery provided both the sinus node branch and that supplying the A-V node, each originating at the usual site. Only small atheromata were present in the right coronary artery except at the margo acutus, where a larger atheroma plus a recent occluding thrombus were present (fig. 2). Within the first centimeter of its course inward from the crux of the heart, the A-V node artery was markedly narrowed by fibromuscular dysplasia (fig. 3). The left circumflex artery crossed the margo obtusus to supply most of the posterior surface of the left ventricle, but just proximal to the margo obtusus there was another atheroma plus recent coronary thrombosis (fig. 2). Directly at its origin the left anterior descending coronary artery was narrowed about 90% by an old atheroma.

On gross examination of the septal perforation its left ventricular opening was near the midportion of the septum, thence extending with a jagged shearing tear posteriorly to empty into the right ventricle at a higher level. Both the sinus node and the A-V node with His bundle were examined with subserial sections prepared in a fashion previously described in these clinicopathologic correlations. A portion of the interventricular septal rupture had tunneled up toward the central fibrous body, ending just below the A-V node (fig. 4). There was extensive hemorrhagic degeneration within the posterior half of A-V node, but the His bundle and its proximal branches appeared well preserved. There were no important abnormalities in the sinus node.

Case 2

A 71-year-old secretary had recently retired from her job and was generally in good health except for mild hyperten-

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Recent coronary thrombosis was present in both the left circumflex (LCX) and right (RCA) coronary arteries of case 1. Unless otherwise indicated, the stain in all photomicrographs is with the Goldner trichrome method. Magnifications are indicated with reference bars.

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Focal fibromuscular dysplasia markedly narrowed the lumen of the A-V node artery of case 1. Goldner trichrome stain in A, with Verhoef-Van Gieson elastic stain of an adjacent section in B.
sion for the previous 10 years. She presented to the emergency room with substernal pain of two hours duration, associated with nausea and apprehension. An ECG demonstrated an acute posterior myocardial infarction with coupled premature atrial beats. The leucocyte count was 16,750/cu mm with 87% neutrophils. Her blood pressure on admission was 130/90 mm Hg but gradually decreased to about 80/60 without clinical evidence of shock. Her heart tones became weaker in this same period of time but cardiac findings did not significantly change until several hours later when an ECG demonstrated complete A-V block with an escape A-V junctional rhythm and occasional sinus captures (fig. 5). The night of admission she seemed to be resting comfortably although occasionally confused. When a nurse attempted to obtain her blood pressure later that night, she found the patient dead.

At necropsy examination most of the posterior and lateral portions of the left ventricle and the posterior half of the interventricular septum were grossly infarcted. This region corresponded to the distribution of a long right coronary artery. Beneath the posterior margin of the septal attachment of the mitral valve there was a ragged excavation into the septum. From this opening a dissecting rupture extended upward into the posterior margin of the interatrial septum behind the A-V node (fig. 6). From that location it communicated via a rupture into the right atrium. The crest of the dissecting septal infarct also was present anteriorly beneath the His bundle but the His bundle was well preserved (fig. 7). The same dissecting infarction of the septum also extended into the posterior free wall of the left ventricle and into the counterpart free wall of the left atrium. There was a recent opening from the ventricular free wall dissection at the margo obatusus into the pericardial space which was filled with enough blood to produce tamponade. The opening from the septal rupture into the right atrium occurred in a small mound of granulation tissue and appeared older than the epicardial rupture.

The main left coronary artery and its anterior descending branch had only moderate degrees of atherosclerosis with no thrombi, but near its origin the left circumflex branch (which was small and short, terminating proximal to the margo obtusus) contained an old atheroma which narrowed its lumen more than two thirds. The right coronary artery gave rise to the sinus node branch and was patent up to that point; just beyond an old atheroma was found that left less than 5% of the original lumen patent. As indicated earlier, the right coronary crossed the crux of the heart to supply the A-V node.
RUPTURED SEPTUM AND HEART BLOCK/James

and most of the posterior surface of the left ventricle. The hemorrhagic infarction which dissected from the interventricular septum up into the atrial septum also involved the posterior margin of the A-V node, and the nearby A-V node artery was narrowed by fibromuscular dysplasia (fig. 8). By contrast, the His bundle and its proximal branches appeared relatively normal, as did the sinus node.

Case 3

A 56-year-old dentist presented to the emergency room with epigastric discomfort which he thought was indigestion. He had not been relieved after demerol administered two hours previously by a physician neighbor. His blood pressure was 85/60 mm Hg and a leucocyte count of 18,550/cu mm contained 85% neutrophils. An ECG showed an acute posterior myocardial infarction, and complete heart block with a slow escape A-V junctional rhythm made intermittently irregular by premature supraventricular beats of uncertain origin (fig. 9). Immediately after admission to the hospital his course became complicated by intermittent bouts of ventricular fibrillation requiring cardioversion. The blood pressure was difficult to maintain even at the initially low admitting level. Digitalis was administered because of congestive heart failure. Heart block with complicating arrhythmias persisted until the fourth hospital day when conducted sinus rhythm returned for the first time. About the same time a new loud systolic murmur was first heard along the left sternal border. Following this, hypotension became an intractable problem and congestive failure progressively increased until death on the fifth hospital day.

At postmortem examination the heart was covered by fibrinous pericarditis. Most of the posterior wall of the left ventricle and posterior half of the interventricular septum were acutely infarcted. An irregular hole was found near the...
posterior margin of the left side of the interventricular septum just beneath the mitral valve; from this a necrotic dissection excavated upward toward the A-V node (figs. 10, 11) and thence across to the right ventricular endocardium where it opened by two separate tracts, one appearing slightly older (more granulation tissue) than the other. The anatomical course of this septal rupture resembled portions of that in case 2. In addition to the hemorrhagic infarction which extended from the interventricular septum directly into the posterior margin of the A-V node (fig. 11), there was also extensive focal necrosis of the midportion of the A-V node and significant narrowing of the A-V node artery by fibromuscular dysplasia (fig. 12, 13). The His bundle and the sinus node were not remarkably abnormal, except for the effect of the pericarditis on the sinus node.16

The main left coronary artery was patent, as was the small left circumflex branch for a distance of about 1 cm; the sinus node branch originated within the patent part of the left circumflex. Just beyond, the left circumflex was completely occluded by an old atheroma, beyond which the artery extended only a short distance to terminate proximal to the margo obtusus. The left anterior descending coronary artery was about 95% occluded by an old atheroma 1 cm from its origin. The long right coronary artery crossed the crux of the heart to supply most of the posterior wall of the left ventricle as well as the A-V node. About 2 cm from its origin from the aorta, the right coronary contained a recent thrombotic occlusion superimposed on an old atheroma.

Case 4

A 65-year-old retired fire captain had suffered from poorly controlled hypertension for about 10 years. Six years before his terminal admission he had had a small anterior myocardial infarction, and thereafter had occasional spells of unconsciousness which were unexplained. The terminal admission was for substernal chest pain which began intermittently but then became sustained for many hours and was associated with weakness, sweating, and nausea. His blood pressure which had previously averaged about 200/100 mm Hg was now 130/80, and a leucocyte count of 14,580/cu mm contained 80% neutrophils. An ECG on admission showed conducted sinus rhythm with an acute posterior myocardial infarction. The night after admission he had more chest pain and the ECG then demonstrated complete heart block with a slow escape A-V junctional rhythm (fig. 14). Complete A-V block persisted on numerous ECG examinations until the sixth hospital day, when Wenckebach cycles first appeared to conduct a sinus rhythm. On the seventh hospital day all sinus beats were conducted although first degree A-V block was present; at that time a new loud systolic murmur was first heard at the left sternal border. The next few hours there was persistent hypotension with increasing congestive failure and the patient died.

At necropsy the major abnormalities were in the heart except for some nephrosclerosis and changes due to congestive failure. There was an old healed anterior infarction near the apex of the heart. An acute myocardial infarction was present in the posterior wall of the left ventricle and posterior half of the interventricular septum. A large irregular cavity

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**Figure 8.** Associated with the dissecting rupture into the interatrial septum of case 2 there was also hemorrhagic necrosis of the posterior portion of the A-V node, marked by two open arrows in A. The margin of the dissection (open arrow in B) is very near the A-V node artery (AVNA), which is slightly narrowed at this point by its thickened wall.

**Figure 9.** This ECG of case 3 shows an acute posterior myocardial infarction and predominantly A-V dissociation with a slow A-V junctional rhythm. Some of the early beats (circles) may represent sinus captures but most are either re-entrant or automatic A-V junctional premature beats.
RUPTURED SEPTUM AND HEART BLOCK / James

The hemorrhagic necrosis of interventricular septum seen beneath the A-V node (two open arrows) in Figure A gave way to dissecting rupture at a point only 2 mm further posteriorly shown in Figure B. Communication of the rupture with left ventricle is marked with an asterisk in B, and the open arrow marks the A-V node artery which is narrowed.

Case 5

A 64-year-old steel worker developed severe retrosternal chest pain at his job and was brought to the hospital. He was ashen in color, sweating profusely and very weak. There had

The main left coronary artery was patent and so were the proximal portions of its two main branches. However, about half way down the anterior interventricular sulcus the left anterior descending branch was completely occluded by an old atheroma and the left circumflex artery was similarly occluded just before it terminated near the margo obtusus. The sinus node branch originated from the left circumflex proximal to the occlusion; this branch was easily dissectible and was patent for its entire course. The right coronary crossed the crux of the heart to supply the A-V node and most of the posterior wall of the left ventricle; about 2 cm after its origin from the aorta it was completely occluded by a recent thrombosis.

The sections from which these two photomicrographs from case 3 were made are 2 and 4 mm more posterior within the region of septal junction than the one in figure 10B; thus, the four sections shown in figures 10 and 11 are about 2 mm from each other in series. Here near the posterior margin of the A-V node the dissecting rupture (large open arrows in A and B) extends from the interventricular septum directly into the area of the A-V node. Long thin black arrows mark the A-V node artery.
been no previous symptoms of heart disease and he had worked regularly. Blood pressure on admission was 130/80 mm Hg and leucocyte count was 14,000/cu mm with 83% neutrophils. An ECG at that time showed an acute posterior myocardial infarction and a regular A-V junctional rhythm (fig. 17). On the second hospital day incomplete heart block appeared with Wenckebach cycles, and on the fourth hospital day the rhythm stabilized as 2:1 A-V block (fig. 18). Except for the effects of posterior infarction, the QRS complexes did not change at all. On the sixth hospital day there was not only normally conducted sinus rhythm but a P-R interval of only 160 msec. A few hours later a loud systolic murmur was first detected along the left sternal border. Thereafter his blood pressure became difficult to maintain and congestive failure progressively increased until his death on the eighth hospital day.

At postmortem examination there were the extensive changes due to congestive failure plus renal nephrosclerosis and a small fusiform aneurysm of the distal abdominal aorta. The heart was covered by fibrinous pericarditis. The posterior wall of the left ventricle and posterior half of the interventricular septum exhibited acute myocardial infarction. The posterior papillary muscle was ruptured through an area of recent necrosis and lay free in the left ventricular cavity. In the posterior portion of the interventricular septum there was an irregular excavated cavity measuring 3 by 3 cm and eroding more than two thirds of the way through toward the right ventricle; loose necrotic tissue, mixed with fibrin and thrombi, covered what appeared to be a previous communication with the right ventricle. Hemorrhagic necrosis involved the A-V node, including one region which may have represented Mahaim fibers but was too far within...
the infarction to be certain (fig. 19). The His bundle exhibited a moderate degree of excessive partitioning by collagen but no recent infarction. The sinus node was extensively involved in the inflammation of overlying pericarditis (fig. 20).

The main left coronary artery was 85% narrowed by an old atheroma. Its left anterior descending branch was sclerotic but patent, while the left circumflex branch was 95% narrowed by an atheroma within its first centimeter. The sinus node branch originated proximal to the narrowing in the left circumflex artery, but was of course distal to the narrowing lesion in the main left coronary artery. The left circumflex artery terminated just proximal to the margo obtusus. The right coronary artery crossed the crux of the heart to supply the A-V node and most of the posterior wall of the left ventricle. The right coronary artery was extremely sclerotic throughout its course, but was completely occluded by an old atheroma about 2 cm proximal to the margo acutus; directly at the margo acutus a recent thrombus also occluded the right coronary.

**Discussion**

All five of these patients presented with an acute posterior myocardial infarction which was subsequently confirmed at necropsy examination. All five had varying degrees of heart block at some time in their course, with an escape A-V junctional rhythm but no other form of stable escape rhythm. While there was infarction and necrosis in every A-V node, the His bundle and its proximal branches were spared in each case. In every case there was either complete or near complete occlusion of a dominating right coronary artery, and in four of the five there was recent right coronary thrombosis. Either extensive infarction (all 5 cases) or hemorrhagic dissection (3 cases) was present all the way to the crest of the interventricular septum. In two of the five hearts this dissection extended directly up into the posterior portion of the interatrial septum and in one also tunneled via the left ventricular free wall to rupture into the pericardial sac and cause tamponade. Despite several differences there are a number of recurring features in these five cases of ruptured interventricular septum with heart block.

Because the blood supply to the human A-V node and proximal His bundle comes from the same main artery that is the principal primary source of blood supply to the posterior margin of the interventricular septum, other observers have indicated that heart block and ruptures of this portion of the septum are readily explained by interruption of this single common source of blood. However, most of the blood supply to the human interventricular septum is from the left anterior descending coronary artery, and the potential collateral circulation either to the A-V node or any portion of the septum is abundant. It is therefore unsurprising that multiple narrowings in the coronary circulation are required for most examples of either septal or any other cardiac rupture, just as was true in all five of the present cases. Wessler and his colleagues emphasized that the pathogenesis of rupture of the inter-

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**Figure 14.** ECG of case 4 shows an acute posterior myocardial infarction with complete A-V block and a slow stable A-V junctional escape rhythm.

**Figure 15.** Focal infarction of the A-V node of case 4 is illustrated here at two magnifications. The area boxed in A is seen in more detail in B. CFB is central fibrous body. The A-V node artery is narrowed.
ventricular septum in this respect was no different from the pathogenesis of rupture of any other portion of the heart.

In addition to necessary combined narrowings of either anterior descending and right coronary artery, or right coronary artery and left circumflex artery, or all three branches, another lesion found in three of these five hearts was the marked narrowing of the A-V node artery. This would not only compound ischemia and necrosis within the A-V node but also effectively interdict that important route of potential collateral circulation. Communication between the A-V node artery and Kugel's artery is a major source for collateral circulation to the A-V node, the His bundle, and much of the interventricular septum.17

It is less surprising that A-V conduction failed in any of these five hearts than that A-V conduction recovered at all. One must conclude that A-V conduction of sinus rhythm can eventually recover even with extensive focal necrosis within the A-V node (figs. 12 and 13), but at the same time one must remember that the earlier presence of complete A-V block probably represented temporary ischemic failure of the entire A-V node. The fact that the His bundle in each case seemed spared from the infarction fits well with the consistency with which A-V junctional escape rhythm emerged.

In fact, there was evidence of actual enhancement of A-V junctional automaticity in two cases (figs. 1 and 17) in an early phase of their clinical course. While it is impossible in these five cases to know the exact histological source for their supraventricular rhythms with narrow QRS complexes during the heart block, some experimental studies in the dog18-20 suggest that the origin would be at or near the histological junction between A-V node and His bundle. This would be close to the spared area in these five hearts. In experimental studies in the dog, such normal escape rhythms bore a remarkable mathematical ratio to the rate of the unchanged normal sinus node.18, 19 That such a ratio could not be established in the five present cases is attributable to both primary and secondary influences on the sinus node in each case (pharmacological therapy, autonomic reflex events, direct injury from ischemia or pericarditis). Furthermore, the rate of the escape A-V junctional rhythm was almost certainly similarly influenced to be faster or slower than might be anticipated when there is no associated infarction.

**Figure 16.** Old damage within the His bundle (arrows in A) is apparent from abnormal fat and collagen within it, but the interventricular septum directly below the His bundle shown in B has new infarction.
Accelerated or "active escape" A-V junctional rhythm (fig. 17) is not a rare finding\(^2,5,21-23\) early in the course of patients with acute posterior myocardial infarction who later develop heart block. What causes this positive chronotropic effect on the A-V junction deserves further study.

In one of these five patients (case 3) recurring arrhythmias and bouts of ventricular fibrillation were a major therapeutic problem. This is not so surprising, given the large amount of ventricular infarction and fibrosis in all five cases, as the fact that cardiac rhythm was relatively stable in four of the five. This stability included periods of active (accelerated) or passive (escape, during A-V block) A-V junctional rhythm. When complete A-V block is produced experimentally by selective suppression of the A-V junctional region of the normal dog,\(^9\) we have in several hundred experiments regularly found but one form of stable escape rhythm, and it originates in the A-V junction. Although it may be assumed that regular escape "ventricular" rhythms would be facilitated during the A-V block caused by myocardial infarction, that was not true in these five cases. On the contrary, there was more chaotic rhythm during intermittent or even sustained conduction of sinus rhythm than when the influence of sinus rhythm was blocked from reaching the ventricles. These observations fail to support the widespread belief that ventricular "automaticity" is enhanced during myocardial ischemia or infarction.

In at least three of the five cases the time of interventricular septal rupture closely coincided with the resumption of conducted sinus rhythm. These two events may not have been causally related, since the time of softening or weakening of the interventricular septum might simply be the same time when sufficient collateral circulation and recovery evolved around the A-V node for conduction to resume. However, it is also possible that improved or more coordinated left ventricular function as a consequence of normal A-V conduction may have sufficiently increased the stress on the weak interventricular septum from the left side to cause it to rupture. This would be somewhat of a paradox, a complication being due to improvement. While these relationships are, for the present, only conjectural hemodynamic events in the pathogenesis of septal rupture, they are clearly appropriate for further clinical investigation.

What happened to large volumes of infarcted and necrotic septal myocardium is something of a puzzle. In none of these five patients was any clinical evidence of arterial embolization detected. Portions of their illness were so complex that small emboli may not have been recognized, and insufficient search was made at necropsy examination to ex-
Hemorrhagic infarction involved both the interventricular septum (IVS) and A-V node (AVN) of case 5. The area boxed in A is seen at higher magnification in B and may have represented Mahaim fiber connections before the distortion produced by infarction which now obscures details in the area.

Fibrinous pericarditis involved the epicardial margin of the sinus node of case 5. The area boxed in A is shown at higher magnification in B. The sinus node is the pale region surrounding its central artery, and adjacent right atrial myocardium in A is marked RA. A small nerve is indicated with N in B.
The extensiveness of cavitation conspicuous patients had farction and thromboses of oxygen may be explained the importance of recent coronary thrombosis. In four of the five cases in this report there was recent coronary thrombosis in an appropriate artery. However, in case 5 the thrombus was distal to an old more proximal occlusion by an atheroma, and in case 1 there were two widely separate coronary thromboses, one in the right coronary and the other in the left circumflex. Since the time that the thromboses occurred in relationship to the pathogenesis of the septal infarction and rupture was not known, it is possible that the thromboses were a secondary rather than primary event, a complication of sustained hypotension or some other process.

Not only was the extent of septal liquefaction and tunneling excava.tion impressive, but there appeared to be a rather consistent anatomic route for this which deserves special comment. There was more necrosis in the midportion of the septum than near either subendocardial region. This may be explained on the right by the normal course of septal arteries being in the right septal endocardium, so that the maximal ischemia and eventual necrosis should be at the end of these vessels’ distribution which would be deep in the septum. It may be explained on the left by the usual sparing of left ventricular subendocardium in most infarctions, in part because of oxygen diffusion from the arterial blood contained in the left chambers. Rupture from the left ventricle into the necrotic midportion of septum would simply be due to the maximal stress on the area from the highest pressure chamber, and thence into the right ventricle because of the transmural pressure gradient.

After rupture occurred, the dissection in three of the five hearts extended directly up toward the central fibrous body where mechanical strength of the collagen probably blocked it (figs. 4, 6A, 7, and 10B). This could also explain why none of the dissecting rupture involved the His bundle, protected as it is by the surrounding collagen which is a part of the central fibrous body (fig. 7). On the other hand, the weak point in the region of junction between interatrial and interventricular septa is at their posterior margin, where the collagen of the central fibrous body disappears toward the mitral valve and is replaced by areolar tissue and fat. This is just the point where the dissecting infarct proceeded from the interventricular septum up into the interatrial septum (figs. 6B, 8B, and 11). From this point the dissecting rupture could proceed anteriorly within the interatrial septum to produce further damage in the A-V node (figs. 6A and 12B) or base of the tricuspid valve (fig. 7), or rupture into the right atrium (case 2), or dissect within the free wall of the infarcted left ventricle and rupture into the pericardial sac to cause tamponade (case 2). The latter process closely resembles the delayed rupture of false aneurysms of the left ventricle which are sometimes responsible for sudden unexpected death.

Given the dramatic and unmistakable hemodynamic consequences of ruptured interventricular septum, and the frequently attendant heart block, particularly when there is posterior myocardial infarction, it is surprising that abrupt or sudden death does not coincide with the time of rupture more frequently. This may be a special consequence of having patients already hospitalized for the painful and otherwise distressing symptoms of acute myocardial infarction of any location. With current public awareness of the symptoms of a heart attack, few patients would fail to seek medical help and subsequently be hospitalized considerably before the time of an anticipated rupture of the heart. One might predict that relatively few patients would survive the time of septal rupture were it not to occur under almost direct medical supervision.

There are at least three matters deserving further clinical investigation in the pathophysiology of ruptured interventricular septum. First, given the necessary substrate of multiple coronary lesions, one can predict that certain patients are “candidates” for septal rupture; examples include recent posterior infarction in a subject with known old occlusion of the left anterior descending artery, or recent anteroseptal infarction when the right coronary is known to be occluded or markedly narrowed. Second, based on events in the present cases, the time of recovery of conducted sinus rhythm following previous heart block may be a period of special hazard worth careful observation. Third, much more can be learned about the pathogenesis of heart block in ischemic heart disease and the pathophysiology of active (accelerated) or passive (escape, during sinus bradycardia or complete A-V block) A-V junctional rhythms by further utilization of modern methods for recording local electrical potentials from the region of A-V node and His bundle.

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