Idioventricular Rhythm in Acute Myocardial Infarction

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SUMMARY
An ectopic ventricular rhythm (IVR) with a rate of 60 to 100/min was detected in 36 of 100 consecutive patients with acute myocardial infarction by constant monitoring of the electrocardiogram. This mechanism was not apparent clinically and was usually transient, lasting 4 to 30 beats. It was frequently associated with inferior myocardial infarction and usually occurred during sinus bradycardia or the slow phase of sinus arrhythmia. Unlike true paroxysmal ventricular tachycardia (PVT), IVR did not progress to ventricular fibrillation and did not influence the prognosis adversely. Recognition of IVR is important in order to avoid unnecessary and perhaps dangerous treatment with cardiosuppressive drugs and electrical cardioversion.

Additional Indexing Words: Arrhythmia, Ventricular tachycardia, Intensive coronary care

Before the advent of constant monitoring of the electrocardiogram and intensive coronary care, ventricular tachycardia was thought to occur rarely in acute myocardial infarction and was invariably associated with a grave prognosis.1-3 In a review of arrhythmias in acute myocardial infarction, Master and associates3 mentioned "the rarity of ventricular tachycardia in coronary artery thrombosis." But recent investigations have shown that ventricular tachycardia occurs rather commonly and is perhaps the most frequently observed disturbance in rhythm in the immediate postinfarction period.4-7 Most of these episodes were not detected previously because they were transient and usually not obvious clinically. The high incidence of ventricular tachycardia in these patients is in keeping with the frequency of this finding after experimental coronary artery ligation.8

In general, two types of ectopic ventricular rhythm have been reported in the course of acute myocardial infarction. The first has a gradual onset usually during sinus bradycardia or the slow phase of sinus arrhythmia; its rate is relatively slow, 60 to 100/min, and its duration is brief, 6 to 30 beats. The second ectopic ventricular rhythm has a more rapid rate and is usually initiated abruptly by a premature beat that interrupts the preceding T wave. The latter form, called "paroxysmal ventricular tachycardia (PVT)," is often sustained and may progress to fatal ventricular fibrillation.

The slower form of ectopic ventricular rhythm has been observed in 36 of 100 consecutive patients with acute myocardial infarction monitored by a relay telemetric system.9,10 At first this mechanism was called "nonparoxysmal ventricular tachycardia,"11 because it was similar to the nonparoxysmal A-V nodal tachycardia originally described by Pick and Dominguez12 with the exception that the pacemaker was idioventricular. We have now abandoned this term in favor of the more descriptive designation, "idioventricular rhythm (IVR)" in acute myocardial infarction. The purpose of this report is to define

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this interesting ectopic rhythm and to describe its clinical setting, cause, prognosis, and management.

Methods

Patients with acute myocardial infarction were admitted to the study if they had an episode of characteristic chest pain in addition to electrocardiographic evidence of transmural infarction and a typical rise in serum glutamic oxaloacetic transaminase (SGOT) concentration within 12 hours of entering the hospital. Each patient was examined at least four times daily, and routine electrocardiograms were obtained twice daily for the first 72 hours. The electrocardiogram was monitored continuously by the telemetry system for the first 72 hours in the hospital.

The telemetry unit has been described in detail in previous publications.\(^9,10\) Essentially it consists of a RKG-100 transistorized, battery-powered, frequency-modulated transmitter and a receiving apparatus. The latter comprises a record and playback console and an arrhythmia detector that provides for continuous recording on magnetic tape of electrocardiograms from one or two subjects, the automatic detection of arrhythmias from such subjects, the encoding of arrhythmias on tape, and the rapid playback of recorded signals with automatic search for arrhythmias.

Results

One hundred consecutive patients with acute transmural infarction were admitted to the study between April 15 and December 30, 1966. There were 64 males and 36 females with an age range of 38 to 84 years. The electrocardiograms demonstrated anterior myocardial infarction in 52, inferior myocardial infarction in 41, and combined anterior and inferior infarction in seven patients. Known previous myocardial infarction was documented in 14 patients. Twenty-one patients died in the hospital, 13 within the first 72 hours. Death was a result of cardiogenic shock or intractable failure in 12 instances, ventricular tachycardia and fibrillation in four, and presumed thromboembolic complications in the remaining five.

True PVT with a rate of 118 to 224/min occurred in eight patients, four of whom had anterior, three inferior, and one combined anterior and inferior myocardial infarction. Four of these were terminated by quinidine or procainamide, while the remaining four progressed to fatal ventricular fibrillation. Seven episodes occurred in the first 24 hours, and there was no diurnal variation. In only one case was the onset of the ventricular tachycardia clinically apparent. This arrhythmia was initiated by ventricular premature beats with an R on T phenomenon observed in all.\(^13\) The mean peak serum glutamic oxaloacetic transaminase (SGOT) concentration was 224 units/ml in patients with PVT as opposed to 96 units/ml in those without ventricular arrhythmias.

One to 14 runs of IVR with a rate of 60 to 100/min were detected in 36 patients by the monitor but were recorded only twice by routine electrocardiography. Most episodes lasted 4 to 30 beats and were usually recorded during sleeping hours (figs. 1 and 2). In one instance, the arrhythmia persisted for more than 8 hours and was possibly related to digitalis overdosage. It occurred in the presence of inferior myocardial infarction in 22 cases and of anterior infarction in 14. Its onset was invariably associated with sinus bradycardia or the slow phase of sinus arrhythmia. IVR usually terminated after slowing of its rate, allowing the sinus mechanism to regain dominance. The rate at the onset was slightly in excess of that of the sinus node frequently giving rise to varying degrees of fusion, a manifestation of the interplay between the two pacemakers (fig. 1). The mean peak SGOT concentration in this group was 84 units/ml, not significantly different from that of those patients without ventricular arrhythmias. This mechanism was not seen after the first 48 hours and did not result in rapid ventricular tachycardia or fibrillation in any of the patients. All of these disappeared spontaneously with no specific drug or electrical therapy. Only three patients died in this group, two of cardiogenic shock and one of a cerebral embolus.

Discussion

Ectopic ventricular rhythms occur commonly in acute myocardial infarction and may be divided into two groups, PVT and IVR. True PVT is defined as a succession of
These strips demonstrate three runs of IVR in a 54-year-old man with acute inferior myocardial infarction. They were recorded with a bipolar chest lead during the early morning hours of the first hospital day with the patient asleep. Note the marked sinus arrhythmia in strips A and B with IVR initiated by a ventricular fusion beat in A and a slightly premature ventricular beat in B. In strip C the rates of the sinus mechanism and the IVR are nearly identical giving rise to 11 successive fusion beats, QRS complexes five to 15.

The strips represent a continuous recording in a 48-year-old man with acute inferior infarction. Note the alternating dominance of the sinus arrhythmia and the IVR and the similarity of the rates. The first run of IVR is introduced by a fusion beat and the second by a slightly premature ventricular beat. Alternating dominance of the heart by sinus arrhythmia and IVR is commonly seen in the immediate postinfarction period.
three or more ventricular premature beats with a rate in excess of 100/min. Although usually related to important heart disease and drug intoxication, PVT has been reported in at least 35 subjects with no known heart disease. IVR is a slower ventricular mechanism with a rate of 60 to 100/min. The distinction is important because of differences regarding clinical background, mode of production, prognosis, and management (table 1). IVR occurred more commonly than PVT in the present series and was more frequently associated with inferior myocardial infarction. Mean peak SGOT levels were much higher in patients with PVT than in those with IVR suggesting more extensive myocardial necrosis in the former. IVR was less apt to be detected clinically because of its brief duration, its relatively slow rate simulating normal sinus rhythm, and its failure to induce changes in vital signs. IVR lacked the abrupt onset and termination of PVT; it was usually initiated during sinus bradycardia or the slow phase of sinus arrhythmia by a ventricular premature beat that occurred late in diastole giving rise to a fusion beat. Similarly, the sinus mechanism regained control of the heart when its rate exceeded that of the IVR. Ventricular fusion beats were seen much more frequently in IVR than in PVT because of the close approximation of the sinus and idioventricular rates in the former. In our experience IVR has not given rise to ventricular fibrillation, and the prognosis would seem to be more favorable than for PVT.

Unfortunately, the literature regarding this mechanism is confusing because of an array of descriptive terms; it has been referred to as slower ectopic ventricular rhythm,4,7 ventricular tachycardia with Wenckebach-type exit block,5 accelerated idioventricular rhythm (personal communication from H. J. L. Marriott), type 1 idioventricular rhythm,13 ventricular tachycardia with slow rate,16 slow ventricular tachycardia,17 and atrioventricular dissociation with idioventricular rhythm.18

In his description of ventricular arrhythmias following experimental coronary occlusion, Harris8 observed that a “ventricular focus with a frequency of impulse formation almost equal to that of the S-A node was alternately gaining and losing dominance of the cardiac rhythm.” Subsequently the ectopic rhythm became sustained, followed by reappearance of alternating dominance as the

Table 1

<table>
<thead>
<tr>
<th></th>
<th>IVR</th>
<th>PVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevalence in this series</td>
<td>36%</td>
<td>8%</td>
</tr>
<tr>
<td>Site of infarction in myocardium</td>
<td>Inferior in 22 of 36</td>
<td>None specific</td>
</tr>
<tr>
<td>Rate per minute Onset</td>
<td>60 to 100</td>
<td>&gt; 100</td>
</tr>
<tr>
<td>Termination</td>
<td>Idioventricular escape or ventricular fusion beat</td>
<td>Abrupt with R on T</td>
</tr>
<tr>
<td>Fusion beats</td>
<td>Gradual slowing</td>
<td>Abrupt with compensatory pause</td>
</tr>
<tr>
<td>Duration</td>
<td>Very common</td>
<td>Rare</td>
</tr>
<tr>
<td>Vital signs</td>
<td>Brief; 4 to 30 beats</td>
<td>Often sustained</td>
</tr>
<tr>
<td>SCOT</td>
<td>Unchanged</td>
<td>Tachycardia; hypotension</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Same as in patients without ventricular arrhythmias</td>
<td>Significantly higher than in patients without ventricular arrhythmias</td>
</tr>
<tr>
<td></td>
<td>Favorable; spontaneous disappearance</td>
<td>Grave; 50% fatal</td>
</tr>
</tbody>
</table>

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ectopic activity subsided. His figure 1 is strikingly similar to our examples of IVR in patients with acute myocardial infarction.

Bashour and associates have recently reviewed ventricular arrhythmias in 42 patients with acute myocardial infarction. Ectopic ventricular rhythm occurred in 70% of their cases with true ventricular tachycardia recorded in 15 patients and a “slow ectopic ventricular rhythm” seen in six. Their figure 3 shows a slow ectopic ventricular rhythm which is similar to that of our tracings. Unlike our series, the slow ectopic ventricular rhythm occurred more commonly in anterior myocardial infarction and in patients with higher mean SGOT levels. Their figure 6 purports to demonstrate suppression of slow ventricular rhythm by intravenous diphenylhydantoin therapy. We are somewhat dubious about this result because of the frequency of spontaneous termination of this mechanism.

Spann and co-workers noted ventricular tachycardia in 15 of 30 patients with acute myocardial infarction whom they monitored for an average of 52 hours. Eleven had ventricular tachycardia with a rate in excess of 100, while four had a “slower ectopic ventricular rhythm” with a rate of 60 to 100/min. Most were transient and clinically unapparent and were absent on restudy 3 weeks to 12 months after the infarction. Ventricular arrhythmias were the most frequently seen disturbances in rhythm in this series. Unfortunately, no electrocardiograms were published.

In a series of 130 patients reported by Lown and associates, 29% had ventricular tachycardia, and it was the most frequent of the serious arrhythmias. He described two types: a potentially malignant form with abrupt onset, rapid rate, and serious dynamic consequences, and a more benign variety with brief runs of 4 to 20 beats. The rate of the latter ranged from 70 to 250/min, and variations in rate at the onset were attributed to “exit block from the ectopic site with Wenckebach’s structure.” In a recent symposium Gazes also commented on ventricular tachycardia with slow rates presumably due to varying degrees of exit block. Although most of our examples of IVR demonstrated slight variations in rate, no abrupt changes were observed in any of them, and we have no evidence to substantiate the possibility of constant or varying exit block.

Kurland and Pressman documented ventricular tachycardia in 25% of their patients monitored for 1 to 3 weeks. Their figure 2 shows a ventricular mechanism in which the onset occurred after slowing of the supraventricular rhythm and in which the rate was only slightly in excess of the latter.

Ventricular tachycardia was documented in 22 of 150 patients studied by Goble and co-workers and was the most common arrhythmia in this series. Others have concurred in this high incidence, but details regarding rate and mechanism are not available.

The mechanism of production of IVR and PVT can be postulated on the basis of recent investigations by Hoffman and Cranefield. Cardiac cells are classified as automatic or self-excitatory, and nonautomatic or excitable only by a propagated impulse. Automatic cells are characterized by spontaneous diastolic depolarization which lowers the transmembrane potential to the level of the threshold potential. This phenomenon is referred to as phase 4 depolarization. Normally automatic cells are found in the sinoatrial node, other areas of the atria, and in the His-Purkinje system, while the muscle fibers of the atria and ventricles are not automatic. Since many automatic cells coexist, these authors have subdivided them into actual pacemakers, those which initiate a propagated impulse, and latent or potential pacemakers which have the capacity for spontaneous impulse formation. Arrhythmias are produced by changes in the activity of normally automatic cells with or without changes in conductivity. In IVR there is an increase in the firing rate of a latent pacemaker, intramyocardial Purkinje fibers, related to enhanced phase 4 depolarization in these cells. This is associated with a decrease in the automaticity of the normal pacemaker in the sinus node allowing the idioventricular mechanism.
to escape and gain control of ventricular activation. In PVT there is an abrupt and marked increase in the rate of automatic ventricular cells causing a rapid ventricular rhythm, without appreciable change in the rate of impulse formation in sinus nodal fibers.

Rational management of the patient with IVR is related to knowledge of its mode of production and its consequences. If it represents a potentially rapid ventricular tachycardia with varying degrees of exit block, then cardiosuppressive drugs or electrical cardioversion are clearly indicated. But we have seen no evidence of rapid acceleration of the rate, varying exit block, or progression to ventricular fibrillation. With the exception of one case, IVR was not sustained and usually lasted less than 30 beats.

Based on the present experience, we think that patients with uncomplicated IVR should not be treated with cardiosuppressive drugs for several reasons: First, IVR has not been associated with rapid ventricular rates and has not progressed to ventricular fibrillation, at least in this group of patients. Second, IVR may actually be a protective mechanism to prevent sustained bradycardia as suggested by Pick and Dominguez for nonparoxysmal A-V nodal rhythm. Finally, these drugs should be withheld because their effects are largely unpredictable in the face of increased phase 4 depolarization in Purkinje cells. As Hoffman and Cranefield have demonstrated changes in conduction induced by drugs like quinidine or procainamide vary greatly depending on alterations in the slope of phase 4 depolarization and the level of the membrane potential.

References

1812—Trotting Horse Method of Resuscitation from Near-Drowning

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