age distribution is similar in the two materials, but 
the predominance of women in Sievers' patients was 
not found in the present study. In accordance to 
previous reports, most of the ruptures in this series 
ocurred during the first three days after the 
infarction. A low incidence of rupture among 
patients with old infarction has been reported previ-
ously. A striking finding at autopsy in the present 
series was that the ruptures never occurred in large 
infarcts.

The clinical characteristics on admission of a 
patient prone to rupture with tamponade seem to 
be: 1) no history of previous myocardial infarction 
and 2) ECG signs of an acute myocardial infarction 
on admission involving the anterior or the lateral 
wall. In this series of 529 patients with acute 
myocardial infarction these characteristics would 
have given only one false negative suspicion. In this 
patient a persistent ventricular tachycardia was 
present on the admission ECG. On the other hand, 
these characteristics would have resulted in a false 
positive suspicion in as many as 90 out of the 529 
cases.

Even if a prediction of rupture would allow for 
prophylactic measures, this study could not reveal 
any pathophysiological principle to counteract like 
hypertension or an abnormal inflammatory response 
to the infarct. The only possibility therefore seems 
to be to attempt immediate surgical intervention 
when rupture and tamponade occurs.

Surgical intervention actualizes three questions of 
practical importance: 1) does the rupture reveal 
itself immediately in the only continuously moni-
tored parameter, the ECG? This problem will be 
dealt with in another paper, and 2) does a sudden 
circulatory arrest in association with a sustained 
QRS configuration for some minutes give any false 
positive diagnosis of rupture and tamponade? In 
this series of 529 patients with acute myocardial 
infarction there was one false positive diagnosis, 
which had two special aspects: the patient had a 
big aneurysm, and it was also the first patient in 
whom a clinical diagnosis of rupture was made in 
this CCU; 3) is it possible to intervene surgically on 
patients with rupture and tamponade with any 
success? This problem will be dealt with subse-
quently.

II. Electrocardiographic Changes*

L. Mogensen, M.D.; O. Nyquist, M.D.; E. Oriinius, M.D.; and 
A. Sjögren, M.D.

The possibility of surgical intervention in cardiac rupture with tamponade 
prompted a study of the value of the continuous electrocardiogram (ECG) for 
an immediate diagnosis. The ECG was recorded continuously in a coronary 
care unit (CCU) during a time period when 32 deaths occurred. At autopsy 
seven patients had myocardial rupture with tamponade and their ECGs all 
showed sudden bradycardia terminally. Another patient had tamponade from 
dissecting aortic aneurysm and also showed similar ECG changes. Two further 
patients showed nonpenetrating lacerations of the myocardium, and their ECG 
did not show sudden bradycardia. In all cases the bradycardia was of nodal 
origin within the first minutes. It is concluded that abrupt onset of brady-
arrhythmias in patients with acute myocardial infarction showing sudden uncon-
sciousness should give rise to a suspicion of cardiac tamponade.

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Table 1—Terminal Events and Findings at Autopsy in 32 Deaths from Acute Myocardial Infarction under Constant Electrocardiographic Recording in a Coronary Care Unit.

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>No.</th>
<th>Percent</th>
<th>Rupture of Free</th>
<th>Pericardial Tamponade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolonged shock and/or frank pulmonary edema</td>
<td>19</td>
<td>60</td>
<td>2*</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular fibrillation without shock or frank pulmonary edema</td>
<td>2</td>
<td>6</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Rapid hemodynamic deterioration with sustained QRS configuration</td>
<td>11</td>
<td>34</td>
<td>7</td>
<td>8**</td>
</tr>
</tbody>
</table>

*In both nonpenetrating. **In one case due to aortic dissection.

A n immediate and reliable diagnosis is essential in any attempt to intervene in patients with myocardial rupture and tamponade, and the diagnostic value of the electrocardiogram, the only constantly monitored sign of cardiac activity in most CCUs, has been studied previously.1,2 Dismans and coworkers3 made the correct diagnosis clinically in 10 out of 13 patients with myocardial rupture and tamponade. The diagnosis was established on the basis of 1) sudden development of unconsciousness, apnea and absence of arterial pulses in combination with 2) persistent cardiac rhythm as evaluated from the monitored electrocardiogram, for two to three minutes, usually followed by bradyarrhythmias from lower pacemakers.

Meuris and coworkers1 have reported in detail the various arrhythmic sequels in seven patients with myocardial rupture of the left ventricular free wall, followed by tamponade. In six cases similar electrocardiographic events were recorded, ie slowing of the sinus rhythm, followed by nodal rhythm. It is suggested that slowing of sinus rhythm followed by nodal rhythm are electrocardiographic changes indicative of acute cardiac tamponade.

Table 2—Clinical, Pathologic and Electrocardiographic Findings in Seven Cases with Cardiac Rupture and Tamponade after Acute Myocardial Infarction.

<table>
<thead>
<tr>
<th>Case</th>
<th>Infarct</th>
<th>Findings at Autopsy</th>
<th>Size of Infarct (mm)</th>
<th>Arrhythmias before, and within 3 minutes after Onset of Bradycardia*</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>Age, yrs</td>
<td>Sex</td>
<td>Age</td>
<td>Site</td>
</tr>
<tr>
<td>------</td>
<td>----------</td>
<td>-----</td>
<td>-----</td>
<td>------</td>
</tr>
<tr>
<td>1</td>
<td>72 F</td>
<td>1-7</td>
<td>Anterior</td>
<td>40</td>
</tr>
<tr>
<td>2</td>
<td>57 M</td>
<td>30</td>
<td>Anterior</td>
<td>50</td>
</tr>
<tr>
<td>3</td>
<td>73 F</td>
<td>1</td>
<td>Anterior</td>
<td>45</td>
</tr>
<tr>
<td>4</td>
<td>85 M</td>
<td>1-2</td>
<td>Anterior</td>
<td>40</td>
</tr>
<tr>
<td>5</td>
<td>71 F</td>
<td>1</td>
<td>Inferolateral</td>
<td>45</td>
</tr>
<tr>
<td>6</td>
<td>63 M</td>
<td>1-2</td>
<td>Inferolateral</td>
<td>50</td>
</tr>
<tr>
<td>7</td>
<td>62 M</td>
<td>1</td>
<td>Anterior</td>
<td>45</td>
</tr>
</tbody>
</table>

*Estimated percentage of left ventricular myocardium.
**ST = sinus tachycardia; NR = nodal rhythm; A-V H = second degree atrioventricular block; IR = idioventricular rhythm; VEB = ventricular ectopic beat; SR = sinus rhythm; SVEB = supraventricular ectopic beat; SB = sinus bradycardia.

This study reports and evaluates the electrocardiographic findings in seven patients with cardiac rupture and tamponade from periods with continuous ECG recording and attempts at emergency surgical treatment in this CCU.3

MATERIAL AND METHODS

The monitored electrocardiogram for each patient in the CCU was in addition recorded continuously with an ink-jet electrocardiograph (Mingograph 81, Elema-Schonander, Stockholm, Sweden) with a paper speed of 10 mm per second. All 32 deaths during these periods of continuous ECG-recording have been analyzed for terminal events, and constitute the present case material. Autopsy was performed in each case.

The material is presented in Table 1, together with important clinical features during the terminal phase, and findings of rupture or tamponade at autopsy.

The ECG-recordings have been studied for all 11 patients without prolonged shock, frank pulmonary edema or ventricular fibrillation initiating the terminal phase. In these 11 cases cardiac rupture could be suspected. Autopsy confirmed myocardial rupture with tamponade in seven of these, aortic rupture with tamponade in one, no rupture or tamponade in the remaining three subjects.

RESULTS

Pertinent clinical, pathologic and electrocardio-
graphic findings are given in Table 2 for the seven patients with myocardial rupture and tamponade. In all seven cases abrupt bradycardia appeared in close relation to unconsciousness from hemodynamic deterioration. The arrhythmias within five minutes before, and three minutes after the onset of sudden bradycardia are also given in Table 2. The QRS-rates for the same time intervals are shown graphically in Figure 1, together with the time for any baseline disturbances before the bradycardia. In addition, any change in the amplitudes of the persistent QRS-complexes have been given. The onset of bradycardia has arbitrarily been taken as the reference point in the diagram. In cases 2, 3, 4, 6 and 7 this was known to have occurred within one minute from sudden unconsciousness although the exact starting point for this was not possible to relate to the ECG.

Baseline disturbances as seen with skeletal muscle activity seemed to be a common feature preceding the onset of bradycardia. In case 1 there were interruptions in the ECG, and in case 4 baseline evidence for muscular activity was abundant, as the patient was on the bedpan, from which he fell. Therefore baseline disturbances are not indicated for these two cases. For the same reasons QRS amplitudes could not be evaluated in these cases. In the remaining five cases a decrease in QRS amplitudes of the sustained rhythm was seen, ranging from 15 percent to 38 percent.

Systematically sampled ECG-recordings for six of the patients are given in Figure 2, case 1 being excluded because of poor technical quality. Examples of the ECG-recordings showing some of these features are given in Figure 3. Sudden onset of bradycardia and reduction of the QRS amplitudes are illustrated.

In the patient with acute myocardial infarction due to dissecting aortic aneurysm, sudden unconsciousness occurred together with sustained QRS activity, and fatal outcome. At autopsy aortic rupture was found to have resulted in cardiac tamponade, and simultaneous electrocardiographic findings included sudden bradycardia, baseline disturbance and reduction of QRS amplitudes.

In two patients with prolonged shock during the terminal phase, the autopsy revealed myocardial lacerations reaching almost through the free left ventricular wall. There was no hemopericardium, and no tamponade, and the QRS rates during the final 30 minutes did not show sudden onset of bradycardia. The described bradycardia thus seems to be due to the sudden cardiac tamponade rather than the myocardial lacerations as such.

In the three remaining patients rapid hemodynamic deterioration occurred with persistent QRS-activity. At autopsy no rupture or tamponade was found whereas the infarcts involved 40 percent, 60 percent and 90 percent of the left ventricular
CASE 7

Figure 2. Systematically sampled electrocardiographic recordings in six patients with myocardial rupture and tamponade. The last baseline disturbance before the onset of bradycardia was taken as reference time. Samples from one minute prior, at, and every 30th second after reference time.

wall respectively. The patient with the smallest infarct also had severe aortic stenosis. No episodes of sudden bradycardia during the terminal 30 minutes were found. Also there was a less abrupt onset of the rapid hemodynamic deterioration in these patients, without sudden unconsciousness.

DISCUSSION

The present series shows a similar electrocardiographic pattern following myocardial rupture with tamponade as in the series reported by Dissman and co-workers and Meurs and associates. In three out of 13 patients in the former series severe arrhythmia or shock was present at the time of rupture and in eight of the remaining ten, bradycardias followed within three minutes after estimated rupture. These include sinus bradycardia, slow idioventricular rhythm, high degree A-V block and probably also nodal rhythm. In one patient ventricular fibrillation occurred. However, the ECG was monitored and not recorded, and detailed description of the sequence of arrhythmias has not been given.

In the series of Meurs and co-workers the ECG was recorded as well as monitored. They found in six patients with rupture of the free left ventricular wall, followed by tamponade, a persistent sinus rhythm, usually for 15 to 25 seconds and followed by slowing heart rate during a period of nodal rhythm. They also noted sudden alterations of the baseline of the ECG indicating sudden changes in respiration and muscular activity or both. They did not find any decrease of the amplitude of the QRS complex. Our findings thus confirm some of these findings and stress the appearance of the very sudden bradycardia as well as showing a decrease in the QRS amplitude.

In the present study the analysis of the ECG during the half hour preceding the rupture has not revealed any significant change in rhythm or rate. At the time of acute tamponade, as evidenced by sudden unconsciousness, all seven patients showed abrupt onset of bradycardia, usually in close association with baseline disturbance and reduction in QRS amplitude. In the case with acute myocardial infarction secondary to a dissecting aortic aneurysm with rupture of the latter into the pericardium, baseline disturbance, QRS amplitude reduction and sudden bradycardia were similarly closely related to sudden severe hemodynamic deterioration with unconsciousness. In the two cases with nonpenetrating lacerations of the myocardium the analysis of the ECG failed to reveal any episode of sudden bradycardia during the terminal 30 minutes. These 11 patients therefore support the hypothesis that it is the tamponade, and not the myocardial laceration per se, which is the cause of the bradycardia.

The presence of cardiac receptors for vagal reflexes involved during distension of the pericardium have been investigated previously. This may therefore well be the mechanism involved in causing the bradycardia seen in the patients we
bradycardia, the last baseline disturbance before this, and the onset of QRS-amplitude reduction can be established from the ECG. In no case did they occur more than 90 seconds apart. It is not possible to establish without additional hemodynamic surveillance the distinct time association between the development of hemodynamic deterioration and the electrocardiographic changes.

The problem of overdiagnosis is raised by the fact that three patients who showed rapid hemodynamic deterioration associated with sustained QRS configuration, did not have rupture and tamponade at autopsy. These three patients did not, however, show any abrupt bradycardia and their hemodynamic deterioration seemed less rapid and was without sudden unconsciousness.

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III. Attempts at Emergency Surgical Treatment

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E. Orntius, M.D.;** A. Sjögren, M.D.;** and B. Werner, M.D.+ The constant observation of patients in coronary care units (CCUs) permits a rapid diagnosis of rupture of the free left ventricular wall and resulting cardiac tamponade. This has allowed for a surgical trial in three patients to relieve the tamponade and to repair the rupture. No patients survived hospitalization. One patient died of a new rupture six hours after successful repair of the original one. The next patient survived for one month but suffered from severe brain damage as a consequence of excessive bleeding during the intervention. The third patient died during the attempts of suturing a new rupture of the myocardium, three hours after the first rupture. The experience shows that a CCU staff can 1) quickly and correctly diagnose a rupture with tamponade, 2) relieve the tamponade and 3) stop the gross bleeding. The main problem does not seem to be to prevent the myocardial sutures from cutting but rather the occurrence of new ruptures. Further attempts with varying techniques seem justified.

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