ing another asymptomatic period or that her disease is improving spontaneously now that she is an adolescent. However, all her asymptomatic periods were associated with a further decrease in her activities until she was confined to home prior to her last hospitalization. Also, serial ECGs showed a progressive increase in the atrioventricular junction disease and more bizarre tachyarrhythmias. Currently the patient is asymptomatic at nearly full activity. Therefore, it is likely that her present well-being is the result of therapy. On the basis of these observations, we recommend that increasing consideration be given to combined pacemaker and drug therapy in these patients.

REFERENCES


Cardiac Rupture Complicating Acute Myocardial Infarction in Presence of Normal Electrocardiogram

Michael G. Loughhead, M.B., B.S.*

Cardiac rupture following acute myocardial infarction occurs most frequently in patients with electrocardiographic evidence of transmural infarction. Unusual presentations of cardiac rupture need immediate recognition to enable successful surgical treatment. An unusual case is presented of cardiac rupture complicating acute myocardial infarction despite a normal electrocardiogram prior to the rupture.

Rupture of the left ventricular free wall causes 4 to 19 percent of hospital deaths from acute myocardial infarction.1 Due to the reduction in hospital deaths from primary arrhythmias following the development of coronary care units, cardiac rupture can be expected to cause an increased proportion of hospital deaths.2 Aggressive surgical treatment of this complication has been successful, but immediate recognition is essential.3 Recent reviews indicate that the vast majority of cardiac ruptures following acute myocardial infarction occur in patients with electrocardiographic changes of transmural infarc-

*Director, Intensive Care Unit, Brandon General Hospital, Brandon, Manitoba, Canada.

Reprint requests: Dr. Loughhead, Intensive Care Unit, Presbyterian-University Hospital, Pittsburgh 15213

Figure 1. Electrocardiogram following admission. The P-R interval is 0.19 sec, QRS duration 0.09 sec, QT interval 0.39 sec and QRS axis + 20°. The electrocardiogram is within normal limits.
nal findings (Fig 1), the white blood cell count was 17000/mm$^3$ with 88 percent neutrophils showing a shift to the left. Serum enzymes showed normal levels of SGOT and LDH, but the CPK was minimally elevated at 87 units (normal less than 70 units).

Severe chest pain recurred two hours after admission and was controlled by 10 mg of morphine given intravenously. One half hour later, he complained of severe nausea and then suddenly collapsed. The blood pressure could not be detected and the cardiac monitor revealed atrioventricular junctional bradycardia at 40/min. Despite restoration of sinus rhythm at 115/min following 0.6 mg of atropine given intravenously, the blood pressure was still unrecordable and the central venous pressure was 23 cm H$_2$O. A further electrocardiogram (Fig 2) revealed a shift in QRS axis from $+20^\circ$ to $+80^\circ$, ST depression in V$_1$.3 and peaked T waves in V$_2$.3. ST elevation of 1.5 mm and a reduction in R wave voltage in V$_3$ were now present, suggesting a lateral wall infarction. This state of electromechanical dissociation with no recordable blood pressure but an apparently useful cardiac rhythm continued until spontaneous breathing stopped and electrical asystole developed two hours later. Autopsy demonstrated acute hemopericardium due to a dissecting hematoma through the lateral wall of the left ventricle. There was severe calcific atherosclerosis of all three coronary arteries and the lateral circumflex artery was occluded by atheroma. Sections through the ruptured myocardium showed some abnormal muscle staining with minimal polymorph infiltration.

## Discussion

Cardiac rupture occurring in patients with acute myocardial infarction but without electrocardiographic changes of transmural infarction appears to be extremely rare. London and London$^1$ in reviewing 47 cases of cardiac rupture following acute myocardial infarction, noted that one of their patients had a normal electrocardiogram prior to the rupture but the time interval between was not reported. Biorck et al$^4$ reviewed 11 patients with cardiac rupture from a consecutive series of 529 patients with acute myocardial infarction and reported that 10 patients had electrocardiographic evidence of acute myocardial infarction and one had a persistent ventricular tachycardia. Friedman et al$^2$ reported that all of their patients had electrocardiographic changes of acute transmural infarction. However, this patient shows clearly that cardiac rupture can occur in the absence of any abnormality on the electrocardiogram prior to the rupture.

The presentation of cardiac rupture in this case is otherwise typical. Recurrence of severe chest pain followed by sudden electromechanical dissociation, often accompanied by sinus or junctional bradycardia, appears to be the most common presentation of this complication.$^3,5,6$ The occurrence of new ST segment changes associated with peaking of T waves is seen frequently with acute hemopericardium.$^7$ A combination of these clinical and electrocardiographic changes appears to be a characteristic syndrome and should lead to immediate recognition of this complication.$5,8$ This is of more than academic interest, as Biorck et al$^1$ noted in their series that in no case was the rupture associated with massive infarction and surgical repair should therefore be feasible.

Similar observations have been made by London and London$^1$ who reported that shock or congestive failure occurred in only one-third of their cases, and by Naeim et al$^6$ who reported only a 7 percent incidence of massive infarction in their series of 44 cases. A recent report by Cobbs et al$^3$ describing the first two long-term survivors of this hitherto invariably fatal complication should stimulate further interest in an aggressive surgical approach to salvage these hearts "too good to die." In the present case, immediate surgical repair may well have saved the patient, as he survived two hours after the rupture, but as the nearest cardiac surgical unit is 130 miles away this was not attempted. In retrospect, this was wrong. Immediate surgical repair should have been attempted. Survival for a period long enough to allow appropriate treatment is probably not as rare as previously thought, as Cobbs et al$^3$ were able to operate on 3 of 16 patients seen over a 5-year period. In their two

---

Figure 2. Electrocardiogram following rupture. ST depression is present in V$_1$.3, peaked T waves in V$_2$.3. V$_6$ shows 1.5 mm ST elevation and reduction in R-wave voltage.

---

372 MICHAEL G. LOUGHEAD
successful cases, 20 and 40 minutes respectively had elapsed prior to resuscitation attempts.

One of the successfully treated cases of Cobbs et al. had only minor nonspecific ST segment and T wave changes on the electrocardiogram prior to the rupture and they emphasized the importance of remembering that electrocardiographic evidence of acute myocardial infarction may be absent. Their success and this case demonstrate that a normal or only slightly abnormal electrocardiogram should not deter immediate surgery if the characteristic syndrome of cardiac rupture should develop.

REFERENCES

Pulmonary Interstitial Fibrosis following Near-Drowning and Exposure to Short-term High Oxygen Concentrations*

Frederick L. Glauser, M.D. and William Richard Smith, M.D.

Following near-drowning in fresh water, a 19-year-old man experienced severe adult respiratory distress syndrome, necessitating ventilatory support with positive end-expiratory pressure and high oxygen concentrations. Post-extubation, his course was highlighted by persistent hypoxemia and interrupted by a lung abscess which responded promptly to antibiotics. Pulmonary function tests were consistent with severe restrictive disease and chest radiograph revealed persistent bilateral alveolar and interstitial infiltrates. An open lung biopsy on the 26th hospital day showed interstitial fibrosis. Over the ensuing two months, the chest radiograph and pulmonary function tests returned towards normal. We attribute the pulmonary fibrosis to incomplete resolution of the alveolar interstitial pathology secondary to the near-drowning and exposure to high oxygen mixtures.

The acute clinical and physiologic sequelae attendant upon near-drowning are well described.1-5 Most abnormalities reverse rapidly with appropriate therapy; the majority of patients are discharged from the hospital within one week. It has been assumed that the pulmonary pathology, consisting of alveolar and interstitial edema with varying degrees of alveolar and capillary cellular damage,6 reverses rapidly and leaves the patient with normal lung architecture and function. That this may not always be true is pointed out in this report, in which fresh water near-drowning and short-term high dose oxygen exposure led to pulmonary fibrosis as evidenced by pulmonary function tests and lung biopsy.

CASE REPORT

A 19-year-old caucasian man was discovered face down in a swimming pool on May 26, 1974. He was taken to a local hospital where an oropharyngeal airway was placed. On subsequent transfer to the Orange County Hospital, he was intubated with an oral endotracheal (ET) tube and placed on 100 percent oxygen by volume ventilator. The patient had never had any pulmonary problems and had a normal preemployment chest radiograph two months prior to admission.

Physical Examination

The blood pressure was 110/60 mm Hg, pulse 160/minute and regular, rectal temperature 39.9° C. The patient was in severe respiratory distress with pink frothy fluid exuding from the ET tube. Auscultation of the chest revealed multiple fine and coarse inspiratory rales bilaterally.

*From the Department of Medicine, University of California, Irvine.