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Dislocation of the distal radio-ulnar joint

Sir

The common association of dislocation of the distal radio-ulnar joint with fractures of the radius is well documented. The incidence of this dislocation with radial head fractures is quoted as 1% (Essex-Lopresti, 1950, 1951; McDougall & White, 1957), and is even more common in Colle's and Galeazzi fractures (Mikic, 1975). Simple acute anterior dislocation of the distal ulnar at the inferior radio-ulnar joint without a fracture is rare, but a few cases have been reported (Cox, 1942; Curr & Coe, 1946; Rose-Innes, 1960).

A 24-year-old left-handed man was brought to the accident and emergency department following a fall on to his outstretched right hand during a game of football. He was in severe pain and there was a depression (furrow) along the distal posterior half of the ulna, with prominence of the radius at the wrist. Elbow movements were normal, but he could not perform supination, pronation or rotation of the forearm. Although he complained of numbness in the ring and little fingers, circulation was normal and there was no objective evidence of sensory loss. Radiographs were interpreted as showing rotation of the ulnar styloid and widening of the wrist joint (Fig. 1). The views were not good but could exclude fracture. The diagnosis of dislocation of the distal radio-ulnar joint was made. Unsuccessful attempts were made to reduce this dislocation using local anaesthesia and diazepam. Under general anaesthesia the clinical deformity was corrected and a check x-ray was satisfactory (Fig. 2). He was immobilized in a plaster of Paris for 4 weeks and later regained full rotation of the forearm.

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Fig. 1 Dislocation of distal ulna at the inferior radio-ulna joint. The ulna styloid is rotated through 90°.

Fig. 2 Immediate post-reduction film in POP. This position was maintained for 4 weeks.

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re Myocardial infarction and ventricular rupture

Sir

Ventricular rupture may occur as a consequence of myocardial infarction, but it is rarely thought of and usually fatal. However, the condition is now treatable under certain circumstances although early detection is essential. We have recently had two cases which illustrate the problems this condition may pose in the accident and emergency department.

- A 61-year-old female was brought into the department complaining of severe right lower chest and upper abdominal pain. Three years previously she had had a myocardial infarction. She had been attending the out-patient department with right upper quadrant pain but all investigations had been negative. She was shocked. Blood pressure was unrecordable. Heart sounds were quiet but present. Chest X-ray was normal. Electrocardiograph showed pathological Q waves in lead III. The central venous pressure was recorded as 23 mm of water. A diagnosis of massive pulmonary embolism was made and she was transferred to the cardiac laboratory for catheterization, but she suffered a cardiac arrest while in the laboratory and resuscitation was unsuccessful. A post mortem examination revealed 200 ml of clotted blood in the pericardial sac with a full thickness infarct in the anterior wall of the left ventricle. There was a small tortuous passage from the cavity of the ventricle to the epicardium.
- The second case was a 64-year-old male who collapsed in the diabetic department. He had had increasing chest pain over the previous 6 weeks. On arrival in the accident and emergency department he had no cardiac output and both pupils were dilated. Electrocardiograph showed asystole and resuscitation was unsuccessful. Post-mortem examination revealed 200 ml of blood in the pericardial cavity with a recent infarct in the inferior wall of the right ventricle. There was a 0.5-cm hole in the free wall of the right ventricle.

Rupture of a free wall of the heart is not uncommon; it accounts for 10% of deaths following myocardial infarction (Rasmussen *et al.*, 1979). A large perforation is rapidly fatal. However, if there is only a small breach in the ventricular wall then the patient may survive if the condition is quickly recognized and treated (Parr *et al.*, 1981).

The major clinical signs of cardiac tamponade are hypotension, pulsus paradoxus, quiet or absent heart sounds and markedly raised jugular venous pressure. Ultrasound is the investigation most likely to confirm the diagnosis but false negatives have been reported due to the presence of blood clot. Chest x-ray is often normal. The electrocardiogram may show tall peaked T waves in the precordial leads or the reversal of pre-existing T wave inversion.