The patient made an uncomplicated recovery, her haemothorax resolved and she was discharged after seven days. Follow-up examination including an MRI scan of the spine, 13 months later, showed no evidence of recurrence of the tumour or of other pathological changes. **Discussion.** The unusual feature of our patient was the presentation with symptoms of dyspnoea and a haemothorax, making the initial diagnosis difficult. A CT scan helped, but the final diagnosis of ABC was established only after surgical exploration and histological examination. There are two possible explanations of the haemothorax. First, penetration of a segmental vessel by the expanding tumour; however, exploration did not reveal such a bleeding vessel. Secondly, spontaneous rupture of the ABC following perforation of its outer shell; to the best of our knowledge this complication has never been reported. On the contrary it is well documented that ABC, although it may grow at an alarming rate, remains well contained by a rim of reactive bone (Enneking 1983); if this is absent, the thickened peristome and the compressed soft tissues circumscribe the lesion (Spjut et al 1971) and therefore no spontaneous bleeding occurs. However, profuse bleeding following evacuation of the blood clot from within the cavity does occur. The source of this bleeding seems to be the highly vascular lining that forms the inside wall of the reactive bone (Schajowicz 1981). In our case the thin shell of the tumour was ruptured at one site and we therefore assume that a spontaneous evacuation of the clot took place into the pleural cavity and caused the haemothorax.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

**REFERENCES**


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**A METHOD OF DIAGNOSING SMALL AMOUNTS OF FLUID IN THE KNEE**

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A normal knee contains 1 to 4 ml of synovial fluid (Ragan 1946). Any irritation leads to a synovial reaction which increases the amount of fluid; this serves as a sensitive, though nonspecific sign of knee injury or internal derangement.

A large amount of fluid can be detected by observation of the patient and by the ‘patellar tap’ test, a small amount by the ‘fluctuation test’ (squeezing the suprapatellar pouch and fluctuating the fluid from one side of the knee to the other) and an intermediate amount by observing one side of the straight knee while stroking the other side (‘the bulge test’ or ‘stroke test’), or by measuring the circumferences of the suprapatellar pouch (Nicholas et al 1976).

Since 1977, we have been using a method, not previously described, for detecting excess fluid in the knee. It is simple, accurate and capable of detecting a minimal excess of fluid. The patient lies supine; on flexing the knee a few degrees an indentation becomes clearly visible on the lateral side of the patellar ligament (Fig. 1). At a certain increased angle of flexion, this indentation disappears (Fig. 2). The angle at which this occurs depends on the amount of fluid which protrudes as the available joint space becomes reduced during flexion.

First the normal knee is flexed until the recess vanishes, then the affected knee is flexed to the same angle. If the indentation in the affected knee fills up at a lesser angle of flexion, this indicates a relative excess of fluid. If, at this angle of flexion, the thumb and index finger are placed on the two sides of the patellar ligament, the fluid can be moved from one side to the other and this is readily palpable.

The test is based on the relative weakness of the part of the capsule which is lateral to the patellar ligament (Smillie 1980) and on the negative pressure in synovial joints (Ropes and Bauer 1953) which causes the recess to appear when there is no excess fluid. The findings can easily be verified by observing the indentation in a flexed
normal knee, then watching it disappear as fluid is injected. We have now used the test on hundreds of patients and regard it as a simple and accurate method of detecting even a minimal excess of synovial fluid in the knee.

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FRACTURE-DISLOCATION OF THE TRIQUETRUM TREATED WITH A HERBERT SCREW

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Triquetral fracture-dislocation has not, as far as we know, been reported. There are pitfalls in diagnosis and treatment can be demanding. We report such a case.

Case report. A 30-year-old man fell onto his dominant, right hand while playing football. His wrist was extended and slightly ulnar-deviated. He was seen in the accident and emergency department on the day of injury complaining of pain over the ulnar side of his wrist; movements were limited. Radiographs were taken and an undisplaced triquetral fracture was diagnosed (Figs 1 and 2). A short arm back slab was applied which relieved his pain.

Two weeks later he was seen in the fracture clinic and examined out of plaster. There was a palpable swelling over the dorsum of his wrist and movements were still very restricted. Further radiographs, including an oblique film, showed the proximal half of the triquetrum lying dislocated, in a dorsal position (Fig. 3). The injury was exposed from a dorsal approach along the radial border of extensor carpi ulnaris. There was complete soft-tissue disruption from the triquetrum proximally although distally the fracture was beginning to unite. The dislocation was reduced and stabilised with a 16 mm Herbert screw. Following fixation there was gross triquetrolunate instability and so Kirschner wires

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