testicular teratoma. The authors are confident that neurofibromatosis . . . has been associated with a number of malignant neoplasms both of neural and non-neural origin . . . .

However, the long term follow up of neurofibromatosis conducted by Sorensen et al. 2 (and quoted by the authors as supporting the hypothesis of an increased malignancy risk in neurofibromatosis) in fact concludes that there is little, if any, increased risk of tumours of non-neural crest origin occurring in these patients. Hecht and McCaw 3 also raised several important points in relation to the supposed association of malignancy and neurofibromatosis. There seems little doubt that sufferers from this condition do have an increased tendency to the development of malignancies. This has been estimated as a 7% risk in the preadolescent and a 20% risk in those over 21 years. 4 This increased risk, despite the number of so-called 'associated tumours', is made up almost entirely of tumours of neural crest origin. It may well be that the malignant potential in neurofibromatosis is restricted to tissues derived from or near to the neural crest. The three cases of Wilms' tumour reported in association with neurofibromatosis 5 represent a likely example of a malignancy occurring in tissue which has developed close to the neural crest.

Urogenital rhabdomyosarcoma 6 would be another. The risk of cancer developing in any individual member of the population as a whole is approximately 25%. People suffering from neurofibromatosis are presumably subject to at least this level of risk and will be prone to the usual variety of tumours seen in a normal population. The authors' calculation of the chance of an individual having both neurofibromatosis and testicular teratoma (3.6—6 × 10⁻⁶) is correct. However, to imply that this single case report has any statistical significance would represent a type 1 statistical error. We believe this association must be considered unproven.

Neil Oakley
Brian Todd
Department of Orthopaedics,
Royal Hallamshire Hospital,
Sheffield, UK.

The photic sneeze

Sir,

I read with interest the leading article on the photic sneeze. 1 A related problem is how to abort an imminent sneeze. Study of an effective stimulus could provide an insight into the physiology of the reflex. Paratroopers are advised to press firmly on the soft tissue overlying the intermaxillary suture, presumably sending somatic afferent impulses via the second division of the fifth cranial nerve resulting in a central inhibitory effect. In civilian life, the involuntary blepharospasm accompanying a sneeze poses a potential danger whilst driving, and knowledge of an inhibitory manoeuvre would be helpful. In these circumstances one cannot avoid photic stimulation and I have found infra-nasal pressure ineffective. Could this be because I am a photo-responder?

D.S.C. Rose
Department of Histopathology,
St. Mary's Hospital Medical School,
London W2 1PG, UK.

Reference


Occult spontaneous intramuscular haemorrhage as a complication of heparin for unstable angina

Sir,

We read with interest the report of spontaneous iliopsoas haemorrhage following streptokinase therapy for myocardial infarction. 1 We report two similar cases of occult spontaneous intramuscular haemorrhage following heparin therapy for unstable angina, which can remain undetected until either the effects of profound blood loss become manifest, or symptoms of an acute compartment syndrome arise.

Case 1 A 78 year old man was admitted with an acute inferior myocardial infarction. He was treated with streptokinase (1.5 million units), aspirin 150 mg daily and started on high dose subcutaneous heparin (12,500 units twice daily). He initially progressed well, but 3 days later developed recurrent chest pain which resolved with nifedipine and an isosorbide dinitrate infusion. On the sixth day of admission he complained of pain and stiffness in his right buttock. A large tense swelling in the right gluteal muscle was noted. There was no history of trauma or intramuscular injections. He became hypotensive, and the haemoglobin concentration fell from 11.0 g/dl to 7.5 g/dl. The kaolin partial thromboplastin time was 136 seconds, control 46 seconds. An ultrasound examination revealed a right gluteal muscle haematoma, measuring 8 × 6 cm. The heparin was stopped, and the clotting returned to normal. He remained hypotensive despite a blood transfusion and developed cardiogenic shock. He died 10 days after admission. It was felt that the gluteal haemorrhage had contributed significantly to his demise.
Case 2  A 53 year old man with known coronary artery disease was admitted with unstable angina. This failed to respond to isosorbide dinitrate infusion and nifedipine, and a continuous heparin infusion (30,000 units per 24 hours) was started. Three days later he developed pain in the left groin and hip, difficulty in extending the left leg, and numbness over the anterior thigh. Examination showed a fixed flexion deformity of the left hip, with severe limitation of hip extension due to pain. The left knee jerk was absent, and diminished sensation over the left anterior thigh was confirmed. A computed tomography scan of the abdomen showed a large fluid collection, presumably a haematoma, in the left psoas and iliacus muscle measuring 10 × 6 cm (Figure 1). This was compressing the left femoral nerve, producing a femoral neuropathy. The heparin infusion was stopped, and the neurological deficit resolved after 3 weeks' intensive physiotherapy. A repeat computed tomography scan showed complete resolution of the haematoma.

![CT scan of abdomen (Case 2).](image)

Occult haemorrhage into the iliopsoas or gluteal muscle has been reported previously following heparin therapy. In both situations, acute compartment syndrome complications such as tissue necrosis, nerve damage and vascular ischaemia may occur, and may often be the presenting feature of the haematoma. Ultrasound or computed tomography scanning is invaluable in diagnosis, and, as in Case 2, serial scans may show complete resolution.

In neither case reported above was there an intramuscular injection, or other local trauma, to precipitate the bleeding. Perhaps the concurrent use of vasodilator therapy produces local vascular changes which predispose to prolonged intramuscular bleeding. The potential benefit from heparin therapy in unstable angina has to be balanced against the increased risk of this and other bleeding complications.

Stephen R.D. Johnston
Hugh M. Mather
Ealing General Hospital,
Uxbridge Road, Southall,
Middlesex UB1 3HW, UK.

References


Venepuncture for calcium assays: should we still avoid tourniquet?

Sir,

Dr McMullan and colleagues have demonstrated convincingly that the application of a standard tourniquet for up to 10 minutes caused an increase in serum proteins and total calcium, but that calcium adjusted for albumin did not change.

We should, however, like to sound a note of caution. Individuals may show significant differences between the slopes of their regression lines of serum calcium on albumin if the venous occlusion is accompanied by forearm exercise, when adjustment of calcium for albumin may be inaccurate. The probable reason is that there is a variable production of lactic acid and lowering of pH within as short a period as one minute of occlusion with fist-clenching and this causes variable dissociation of protein-bound calcium which is lost with plasma water through the capillary wall by ultrafiltration.

Thus, although a tourniquet can be used if the total calcium is adjusted for albumin, it is imperative to avoid forearm exercise.

B.E. Walker
R.B. Payne
Departments of Medicine and Chemical Pathology,
St. James's University Hospital,
Leeds LS9 7TF, UK.