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 McCaw³ 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 neurofibromatosis⁵ represent a likely example of a malignancy occurring in tissue which has developed close to the neural crest.

Urogenital rhabdomyosarcoma⁶ 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 (3.6×10^{-8}) 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.

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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?

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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 x 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.