Letters to the Editor

Helicobacter pylori – our knowledge is growing

Sir,

I enjoyed the leading article on *Helicobacter pylori* by Dr Colin-Jones published in your October 1990 issue.¹ However, the statement that Marshall and Warren were the first to describe these organisms in the stomach is inaccurate. They were indeed the first to associate these organisms with gastritis and generate the current interest in this organism.

Spiral bacteria in animal stomachs was probably first reported by Bizzozero² in 1893. The presence of these organisms in human stomachs was confirmed by Krientz³ in 1906. In 1938 Doenges⁴ found these organisms to be present in 43% of 242 human autopsy specimens of the stomach. Recently they have also been detected in ectopic gastric mucosa in Meckel's diverticulum.⁵

These organisms became known as campylobacter-like organisms because of their similarity to other members of the genus following the work of Marshall and Warren^{6,7} in 1983–84. Later the name *Campylobacter pyloridis* was used and shortly afterwards was changed to *Campylobacter pylori*. More recently the name *Helicobacter pylori* has been suggested⁸ due to the findings of fundamental structural differences between this organism and members of the campylobacter genus.

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Helicobacter pylori – our knowledge is growing

Sir,

Dr Colin-Jones gives a good summary of present knowledge of *H. pylori* infection in the 'West' in his leading article.¹ However, it is important to remember that *H. pylori* infection is worldwide and recommendations made for 'Western' populations cannot necessarily be applied to other populations.

In Africa, over 70% of the population have antibodies to *H. pylori*, with 45–82% infected before the age of 10.^{2,3} The latter findings are from the northern savannah of Nigeria where peptic ulcer is uncommon.⁴ The pathological role, if any, of *H. pylori* in this population is unclear.

The *H. pylori* status has only been determined in a small number of Africans with peptic ulcers, so far all have been infected. However, with such a high prevalence of infection in the normal population, the problem of eradication and an unknown reinfection rate it is difficult to recommend anti-*H. pylori* therapy.^{5–7}

The largely rural population in Africa often does not attend for outpatient follow-up, and may not re-present until they have a life-threatening ulcer complication. Operation is probably the best definitive treatment for those with proven ulceration.

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Testicular teratoma and peripheral neurofibromatosis

Sir,

We were interested in the case recently reported by Hilton *et al.*¹ of a patient with neurofibromatosis developing

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.