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Laparoscopic appendicectomy

SIR—Tate and colleagues' (Sept 11, p 633) report emphasises the need for well-conducted, prospective randomised trials to evaluate new surgical approaches. Although a learning curve is inherent in the development of any new operative technique, it is also important that accompanying morbidity and mortality are accurately documented by such studies.

A 58-year-old man was referred for investigation of fever and weight loss of two months' duration. Mediastinal lymphadenopathy was detected on a plain chest radiography. A previous chest film obtained at the time of a laparoscopic appendicectomy eight months earlier was normal. Appendicectomy had been reported as technically difficult in view of caecal thickening, which was presumed to be secondary to accompanying inflammation. Other than persistent mild incisional discomfort, the patient had had no other abdominal symptoms postoperatively. Computed tomographic (CT) scanning of the chest revealed widespread bilateral hilar and mediastinal lymphadenopathy, suggesting a differential diagnosis of lymphoma, sarcoidosis, or metastatic disease. A cervical mediastinoscopy was done to establish a tissue diagnosis. Biopsy showed adenocarcinoma, with histological features suggestive of a colorectal primary turnover. Further imaging of the lower gastrointestinal tract (barium enema, CT scanning) showed a large caecal carcinoma with extensive retroperitoneal lymphdenopathy.

This case illustrates two points. First, laparoscopic techniques may not necessarily increase diagnostic yield, as is advocated by their proponents. Although the laparoscopic appendicectomy had been done by an operator experienced in this technique, the presence of co-existing inflammatory disease may well have obscured any additional pathology. With the open technique, the caecum would be palpated during mobilisation, possibly leading to an earlier diagnosis of the associated caecal tumour. Second, although metastases to mediastinal lymph-nodes are frequent with intrathoracic malignant disease, it is unusual for an intra-abdominal tumour to present in this manner.

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SIR—Tate and colleagues report a prospective, randomised trial comparing laparoscopic with open appendicectomy. They show no difference in postoperative pain between the two groups. Unfortunately, they do not state what analgesia was given either before or during surgery and so no inferences can be made from their results with respect to postoperative pain relief. With any study intending to compare pain levels after surgery it is essential to standardise preoperative analgesia especially with the increasing interest in pre-emptive analgesia[1] and related studies.[2] Additionally, their conclusion that their findings do not support widespread adoption of a laparoscopic alternative to a traditional operation is debatable. In fact their work confirms my view that, for certain procedures, the laparoscopic approach offers no advantages.

As the training of surgeons in laparoscopic surgery improves and the results of prospective, randomised trials appear, I have no doubt that for specific operations the laparoscopic approach will become the technique of choice. This choice may apply to operations such as cholecystectomy or inguinal hernia repair, whereas for appendicectomy and perhaps major abdominal procedures for carcinoma, enthusiasm for laparoscopy may be tempered. Unfortunately, in getting to this stage, many patients will have had complications resulting from this minimally invasive surgery. There are all too many intensive care specialists who know of several admissions to their units necessitated by those complications.

Laparoscopic surgery was introduced without any prospective, randomised trials. It was merely the surgeons' enthusiasm for something new and the worry of being left behind if they did not master the technique, that led to the explosion in popularity of this minimally invasive surgery. Now is the time for a more rational approach.

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Chronic fatigue, ME, and ICD-10

SIR—The nosological status of fatigue syndromes continues to cause debate and controversy.[1] The inclusion in the tenth revision of the International Classification of Diseases (ICD-10)[2] of benign myalgic encephalomyelitis as a synonym for postviral fatigue syndrome (G93.3) under Diseases of the Nervous System seems to represent an important moral victory for self-help groups in the UK[3] who promote the notion of myalgic encephalomyelitis (ME) despite almost universal scepticism of the appropriateness and accuracy of this term among neurologists. Moreover, it is unlikely to lead to advances in our understanding of the condition. First, sufferers pleased to avoid the imagined stigma that a psychiatric diagnosis might carry will nevertheless balk at the inclusion of benign in the title. Second, there is no evidence of an inflammatory process affecting the central nervous system; indeed, if there were, they would be classified with other encephalitides (G04). Third, diagnostic confusion is bound to arise since the nineteenth-century term neurasthenia[4] remains in the Mental and Behavioural Disorders chapter under Other Neurotic Disorders (F48.0).[5] Neurasthenia would readily suffice for ME since it is characterised by a main complaint of fatigue, both mental and physical, with other somatic symptoms and mental phenomena like worry and depression present, but in minor degrees. Further instructions urge clinicians to record whether the syndrome develops in the aftermath of physical illness, especially viral infection, yet postviral fatigue syndrome is given as an exclusion criterion. Thus, the two definitions, though identical, are mutually exclusive.

It is regrettable that the term chronic fatigue syndrome (CFS) was not adopted since this avoids unsubstantiated pathophysiology implied by myalgic encephalomyelitis and is neutral regarding aetiology. CFS avoids artificial separation between mental and physical symptoms and treats them both as inherent to the syndrome. Furthermore, the WHO may have made use of converging case definitions from both the Centers for Disease Control in the US[6] and the Oxford criteria from the UK,[7] which have proven reliability as well as clinical and research usefulness. Applying more stringent criteria for CFS in the hope of revealing a more neurological sub-group succeeds only in strengthening the association with psychiatric disorders.[8] Finally, considerable research effort has yet to

identify any grounds for separating postviral (or bacterial or protozoal) fatigue, including ME, from the broader CFS other than what sufferers call their condition. The role of viruses may turn out to be an important trigger, but classifying disease in general on this basis is not seriously contemplated.

Whereas the director of the WHO Division of Mental Health states in his introduction to the ICD-10 that "a classification is a way of seeing the world at a point in time", we believe that this latest attempt to classify fatigue syndromes will prevent many people from seeing the world as it actually is.

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Stressful life events and Graves' disease

SIR—In his Sept 4 commentary Rosch quite rightly concludes that despite much suggestive data, the role of stressful life events in the aetiopathogenesis of Graves' disease is controversial. However, the occurrence of thyrotoxicosis and exophthalmos and typical histological changes of Graves' disease in "wild rabbits within hours of vigorous pursuit" that he cites as supporting the notion of "stress thyrotoxicosis" is one of those charming medical legends that on close scrutiny prove to be incorrect.

Kracht and Kracht[1] reported in 1952 that wild rabbits, caged in a laboratory and exposed to barking dogs, lost weight quickly, and at necropsy showed thyroid hyperplasia. Owing to the primitive state of knowledge at the time, no specific tests of thyroid function were done in those animals. In 1953, G W Harris, then professor of physiology at the University of London and renowned as the originator of the hypophysial-portal chemotransmitter hypothesis of anterior pituitary control, sought to apply a new method of measuring rabbit thyroid function developed in his laboratory by C von Euler and K Brown-Grant to confirm the Krachts' claim. As a new postdoctoral fellow, I accompanied Harris to Dorking, Surrey, where, with the help of a professional rabbit-catcher and a trained ferret, we netted five wild rabbits. The rabbits were transported in sacks to the neuroendocrine laboratory of the Maudsley Hospital and caged for observation.[2] We were unable to confirm the Kracht and Kracht report. When tested by measurement of their rate of radioactive iodine discharge from the neck (a highly stressful procedure that involved restraining the animals for several minutes twice a day), the rabbits' thyroid hormone release proved to be suppressed, as is the case in laboratory rabbits similarly studied. The animals did in fact lose weight for the first week but for the succeeding 6 weeks either maintained or gained body weight. Transient widening of the palpebral fissure but not exophthalmos was noted. Indeed, the characteristic pituitary-thyroid response to stress in laboratory animals and in man is that of suppression, probably due to inhibition of thyroid-releasing hormone and increased somatostatin secretion from the hypothalamus. The histological finding of hyperplasia in wild rabbits is uninterpretable because the dietary intake of iodide and naturally occurring goitrogens is not controlled. The experiment could not be repeated the next year because of the myxomatosis epidemic that virtually wiped out the wild rabbit population in Europe, including the UK.

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SIR—Rosch attributes the sentence "the safest answer to the question of whether stress causes Graves' disease is: perhaps" to us.[1] This sentence did not appear in our report, but did so in another by Harris and colleagues.[2]

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1 Sonino N, Girelli ME, Boscaro M, Fallo F, Busnardo B, Fava GA. Life events in the pathogenesis of Graves' disease. *Acta Endocrinol* 1993; **128:** 293–96.
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The vanishing ECG

SIR—In our centre, about 80 electrocardiograms (ECGs) were stored in 35 plastic folders in an ordinary cupboard at controlled room temperature. After three months, all printout tracings had completely disappeared without modification of the colour or grid of the paper. To reveal the vanished ECGs, the ECG paper was heated by passing it through a laser printer, resulting in all cases in the appearance of a white ECG tracing on a black background.

The numbers of records originating from medical devices (ECG recorders, blood pressure monitors, echographic recorders, defibrillators) and fax machines that use a thermal recorder printhead are increasing. Although device directions usually recommend that exposure of thermosensitive paper to bright light should be avoided, other paper storage conditions are not specified—in particular, there is no warning about storage in plastic folders or warm environments. Since European good clinical practice guidelines oblige physicians to keep all source data obtained during investigational protocols for 15 years, we strongly recommend systematic making of paper copies or scanning for computer storage to prevent loss of data presented on heat-sensitive paper.

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CORRECTIONS

Event-related potentials in vegetative state—In this letter by Marosi and colleagues (June 5, p 1473) the fourth author's name should have been P Bramanti.

Tuberculosis and immunodeficiency in HIV-1-infected patients in Africa—In this letter by A M Elliot and colleagues (Oct 23, p 1053), the percentage of patients with lung disease only who had oral candida was 10% (not 20% as shown), and the percentage of those with pleural disease only who had fever for greater than 1 month was 24% (not 34%).