and response to treatment at 2, 4, 6, and 13 weeks. Clinical response was defined as complete re-epithelialisation of all lesions at the end of treatment and no reactivation or mucosal involvement during 52 weeks of follow-up. Anaemia was considered as a haematocrit of less than 36% (men) or less than 32% (women) and methaemoglobinaemia was defined clinically by the presence of cyanosis with no evidence of heart or lung disease.

Most patients were adults (range 17–39 years), men (10/11), and had one to three cutaneous lesions. Mean duration of lesions at the time of enrolment was 2.1 months. Two of the six patients who completed the course of treatment had a favourable clinical response. Four patients were clinical failures: lesions of three failed to re-epithelialise and a fourth responded initially but developed parasitologically confirmed mucosal involvement at 30 weeks. Of the remaining five patients: one was withdrawn because of adverse effects (anaemia, methaemoglobinaemia, and headache), one dropped out because of headache, and three were lost to follow-up after 2, 5, and 6 weeks of treatment. Anaemia (8/11) and methaemoglobinaemia (5/11) were the most frequent adverse effects, probably due to the high dosage used. Nausea (4/11) and vomiting (2/11) also occurred. *Leishmania (V) panamensis* was identified from each of the six cases where parasite isolation was successful.

Although the results of treatment of a small number of patients in an uncontrolled trial do not provide conclusive results, dapsone does not appear to be a promising alternative in the treatment of cutaneous leishmaniasis in Colombia.

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Lack of association between 5-HT_{2A} gene promoter polymorphism and susceptibility to anorexia nervosa

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It has been suggested that there may be an aberration in the serotonergic (5-HT) system of those with anorexia nervosa and data from Collier and colleagues¹ demonstrate a putative association between a polymorphism in the promoter region of the gene for 5-HT_{2A} and genetic predisposition to the disease. We have attempted to replicate these data.

We compared a cohort of 152 patients with anorexia nervosa (ICD 10 criteria) with 150 normal female controls. Patients were all white females from the Yorkshire Centre for Eating Disorders. The control cohort was obtained from a number of sources, but did not have a personal or immediate family history of an eating disorder or other psychiatric illness. Written informed consent was obtained

	Genotype			Allele usage	
	–1438A/A	–1438A/G	–1438G/G	–1438A	–1438G
Controls (n=160)	30 (0.2)	67 (0.45)	53 (0.35)	127 (0.42)	173 (0.58)
Anorexia nervosa (n=152)	39 (0.25)	68 (0.45)	45 (0.30)	146 (0.48)	158 (0.52)

Genotype and allele usage of –1438G/A polymorphism in anorexia nervosa

and peripheral venous blood sampled. All individuals were asked to complete identical questionnaires. These were independently assessed before inclusion into the study. DNA was prepared by standard protocols. PCR primers were synthesised according to the sequence published by Collier et al¹ and used to amplify a 468 bp fragment of the promoter region of 5-HT_{2A}. Genotypes were determined by digesting the resultant 468 bp product with *MspI* and resolving on a 2% agarose gel. Statistical analysis was performed with “Clump” to identify any differences between both genotype and allele frequency in the two populations.

We were unable to identify any association between genotype or allele usage at this locus and anorexia nervosa (table: genotype usage $\chi^2=1.82$, $p>0.1$, $df=2$; allele usage $\chi^2=1.97$, $p>0.1$, $df=1$). These data indicate that this polymorphism is not involved in the genetic predisposition hypothesised in anorexia nervosa in our population.

Restriction-fragment length polymorphisms are bi-allelic and in order to satisfy Hardy-Weinberg equilibrium these alleles will tend to be seen frequently in the general population. Thus, it is unlikely that a shotgun approach using such markers will shed any light on the genetic component of a complex disease such as anorexia nervosa.

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Recovery of axonal transport in “dead neurons”

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It is generally believed that a neuron is highly sensitive to hypoxia or glucose deprivation and that cerebral ischaemia of more than several minutes results in irreversible brain neuron damage. This view has been challenged,¹ and we now present evidence for survival of human brain neurons up to 8 h after death such that they still have the potential to recover their functions of energy metabolism and axonal transport.

The evidence was obtained from our study of more than 30 postmortem human brains. Postmortem delay is usually 3–6 h. Brain tissues, ranging from a slice of 400 μ m to a block of 3×2×2 cm, from different areas (cortex, hypothalamus, and brainstem) were preincubated in modified artificial cerebrospinal fluid at 0–4°C for 2.3 h. Two neuronal tracers, neurobiotin and biotinylated dextran amine, which can be taken up and transported along the axon only by “living neurons”, were injected in different areas in these brain tissues, then incubated in artificial cerebrospinal fluid at room temperature (22°C) provided with 95% O₂+5% CO₂. After 6–18 h incubation, the tissues were fixed and cut into sections. The tracer was viewed by

Photomicrograph showing labelled fibres leave dorsomedial nucleus of hypothalamus (DMH), run ventrally, and project to ventromedial nucleus of hypothalamus (VMH), indicating recovery of axonal transport

an avidin-biotin-peroxidase complex (ABC) detection method. Consequently, tracing over a distance of 0.5–1.5 cm along the axon from the injection spot was observed in the different brain areas. An example is shown in the figure. The axonal transport under these conditions is an active energy-dependent process rather than passive diffusion, since no transport was visible if oxygen or oxygen and glucose were omitted during incubation. In addition, adding a physiological concentration of cortisol during incubation increased the neurons' ability to transport.

Our findings indicate that with suitable in-vitro treatment so-called dead neurons recover oxidative metabolism, energy production, and axonal transport. There appear to be some mechanisms that may protect neurons from death and, to some extent, their function may recover. It may be possible to trace anatomical connections between different brain areas in the human brain, and to culture human postmortem neurons to investigate brain function in health and disease. New strategies for treating human brain injury induced by transient cardiac arrest or stroke may be developed.

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Is laparoscopic appendicectomy a gynaecological procedure?

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Chronic pelvic pain accounts for 10% of visits to a gynaecologist and over 40% of diagnostic laparoscopies. Although appendicitis is one of the differential diagnoses of pelvic pain, it is unusual for gynaecologists to remove the appendix, although the first laparoscopic appendicectomy was done by a gynaecologist in 1983.¹ The technique has since been widely adopted by many surgeons for the treatment of acute appendicitis.²

We reviewed 20 women (mean age 34, range 26–59 yr) presenting at our department with chronic right-sided pelvic pain as their main complaint who underwent laparoscopic appendicectomy as part of their management. Mean duration of pelvic pain was 8.1 years (95th CI 4.7–11.6). 13 had had diagnostic laparoscopies previously and six had had laparoscopic or open surgical procedures. The appendix was involved with adhesions or looked abnormal in 18 patients. Appendicectomy was done with bipolar cautery and endoloop sutures. 17 women had other laparoscopic procedures for endometriosis, pelvic adhesions, ovarian cysts, or leiomyomas. 17 of the appendices were histologically abnormal, including one of two which looked normal; abnormal histology included fibrosis (n=8), lymphoid hyperplasia (5), chronic inflammation (3), acute inflammation (2), faecoliths (2), and actinomycosis (1). Women were reviewed 6 weeks postoperatively and 18 were followed-up for up to 6½ years (mean 2.3 years). 15 women reported complete relief or marked improvement of their symptoms at 6 weeks. 13 of this group had long-term follow up, 11 (85%) of whom had sustained relief of pain. There was no correlation between outcome and appendix histology or whether appendicectomy was combined with other procedures.

Our results confirm that an abnormal appendix is common in women with chronic right-sided pelvic pain.^{3,4} Although the clinical significance of some histological abnormalities remains controversial, short-term benefits of appendicectomy have been shown,^{3,4} and our study confirms that pain relief can be long-term, even if the appendix is histologically normal. While the presence of other pathologies confounds the assessment, pain relief following appendicectomy in our study was considerably better than, for instance, the reported symptomatic improvement after laparoscopic surgery for minimal endometriosis (61% vs 38%).⁵ The high prevalence of abnormal appendix histology in the women we report, together with good pain relief after appendicectomy, suggests that laparoscopic appendicectomy should be considered by gynaecologists in the management of such cases, even in the presence of other pelvic pathologies.

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