In the meantime the Board of Faculty remains our academic body whose aims are to foster and encourage education and high standards of practice. No doubt it is supported in these activities by all those elected to the board. To suggest that any elected member who supports the concept of a separate college is acting disruptively is surely casting an unjust slur on individuals. If some of these individuals genuinely believed that the best way for anaesthetists to further the activities required of their academic body is through a College of Anaesthetists, then many of us believe that they are being truly loyal to their principles, their electors, and their specialty.

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SIR,—As a successful candidate in the recent election to the Board of Faculty of Anaesthetists of the Royal College of Surgeons of England may I reply to the letter from Professor D Campbell and others (4 March, p 574)?

The letter suggests that those elected to the board will be "willing to disregard their declaration of loyalty to that body which, in fact, they clearly plan to disrupt." May I reassure the authors and others that I have no prior intention to "disrupt" the board and shall pursue the highest standards of representation in academic anaesthesia to the best of my ability and according to the dictates of my conscience.

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SIR,—I cannot muster the authority of the platoon of professors who were signatories to the letter under the above heading (4 March, p 574). However, I can say that feeling among my colleagues in this locality is strongly pro-college, particularly among senior registrars and recently appointed consultants. Among such colleagues the letter from the Royal College of Surgeons signed by the president and deans produced a very strongly adverse reaction, giving rise to such expressions as "humbug," "mealy-mouthed," and worse.

The most important aspect of this affair is that the Faculty of Anaesthetists is not self-accounting and the moneys raised by and from anaesthetists is spent by a body with a large majority of surgeons, and it is this which has determined a very large number of anaesthetists to break away.

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SIR,—Professor Donald Campbell and others (4 March, p 574) state that they find the editorial in *Anaesthesia* and the appeal letter from the Anaesthetists' Academic Foundation misleading in that both give the impression that the proposal to form a separate college is generally accepted by anaesthetists. As a signatory to that letter I do indeed believe that the majority of anaesthetists wish to establish a college of their own, provided it is financially feasible, since full equality and independence with the Royal College of Surgeons of England have proved impracticable. All the evidence (including a referendum in 1972 and subsequent discussions at annual general meetings and linkman conferences) supports this belief, but there is the additional safeguard (mentioned in the appeal letter) that the trustees are charged with seeing that the money is used in a way which accords with the wishes of the majority of the specialty.

Although the appeal is designed to raise funds for the foundation, the response it attracts will—given its objective—indicate the support for that objective. In a democratic society one has to accept that, while there are bound to be those who dissent, the will of the majority must prevail. It is to be hoped that this is conceded and opposition will not remain "adamant" if its numerical strength is less than its obvious ardour.

The message to those who have considered all the facts and accept the desirability of establishing an independent College of Anaesthetists is clearly to respond to the appeal letter as soon and as generously as possible to assert their commitment.

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Cimetidine prophylaxis after renal transplantation

SIR,—The paper by Dr R H Jones and others (18 February, p 398) concerning the use of cimetidine to prevent upper gastrointestinal haemorrhage after renal transplantation contains misleading statements regarding the pathophysiology of peptic ulcer disease in transplant patients, and I cannot agree with the recommendation that cimetidine should be given as a matter of routine after transplantation.

The authors state that the recent findings of McGeown *et al*[1] in renal transplant recipients reinforces the view that patients treated with steroids have a significantly higher incidence of peptic ulceration. Nowhere does the quoted paper state this point of view or allow such a conclusion. A comprehensive review of the literature[2] convincingly shows a higher prevalence of peptic ulcer disease in patients with chronic renal failure—especially those undergoing dialysis. Furthermore, there is experimental evidence of a mechanism whereby the two diseases are connected.[3] Peptic ulcer in transplant recipients is therefore a complication of the chronic uraemia which has gone before. Post-transplant steroid therapy brings the ulcer disease to light by causing bleeding and perforation, and its contribution to de-novo ulcer disease is probably minimal. Pre-transplant endoscopy identifies patients with ulcer disease, who are therefore at risk of bleeding after transplantation and likely to benefit from prophylactic cimetidine. More often, however, such bleeding is due to gastric erosions, and endoscopy is of no value in predicting which patients will develop this lesion. However, the majority of such gastric erosions occur when massive doses of prednisone are given for acute rejection episodes and also (in some centres) in the early post-transplant phase. It is worth noting that in the series reported by Dr Jones and his colleagues the prednisone dosage started at 10 times that used in the Belfast series[1] and continued at a relatively much higher dosage for the first four months; the respective incidence of gastrointestinal bleeding in the two series was 18% and 7%.[4]

Prevention of upper gastrointestinal haemorrhage after renal transplantation may therefore be achieved by (1) pretransplant endoscopic assessment; (2) sparing use of steroid; and (3), for those with ulcer, use of cimetidine in the early post-transplant stage and also when antirejection treatment is necessary. It does not make sense to add a second drug to prevent the undesirable side effects of the first without first examining other ways of minimising the side effects of the first drug. Surely the questions should be: are massive doses of prednisone really necessary, and do the benefits (if any) outweigh the substantial hazards? A comparative trial of different prednisone regimens is at least as necessary as further trials with cimetidine.

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[1] McGeown, M G, *et al*, *Lancet*, 1977, **2**, 648.
[2] Doherty, C C. In preparation.
[3] Doherty, C C, *Kidney International*. In press.
[4] Doherty, C C, *et al*, in *Proceedings of the XIVth European Dialysis and Transplant Association*, 1977, p 386.

Cimetidine and serum prolactin

SIR,—Dr S K Majumdar and his colleagues (18 February, p 409) report normal serum prolactin concentrations in five male patients treated with cimetidine 1 g/day for between one and six months. We have studied the prolactin response to cimetidine in healthy male subjects.[1] An intravenous bolus injection of cimetidine 400 mg resulted in high blood concentrations of cimetidine. The measured peak mean concentration (±SEM) was achieved 2½ min after injection (84·6±7·4 μmol/l (2·13±0·19 mg/100 ml)) and there was a concurrent three-fold increase in serum prolactin which returned to pretreatment values after 70 to 95 minutes. These data are in agreement with those from Carlson and Ippoliti.[2] We found no increase in serum prolactin when subjects were treated with bromocriptine prior to injection of cimetidine 400 mg, nor in subjects given single oral doses of cimetidine 800 mg, after which peak mean blood cimetidine concentrations (14·0±3·5 μmol/l (0·35±0·09 mg/100 ml)) was <20% of that achieved after intravenous injection.

The serum prolactin concentration has been abnormally increased in only three of seven patients with gynaecomastia or galactorrhoea in whom it was measured. We concluded that at high blood concentrations cimetidine may be acting directly or indirectly at the dopamine receptor in the pituitary to produce hyperprolactinaemia or on the uptake of prolactin in peripheral tissues. Hyperprolactinaemia may be a rare idiosyncratic response at the lower blood concentrations normally associated with oral therapeutic dosage regimens of cimetidine.

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[1] Burland, W L, *et al*, *British Journal of Clinical Pharmacology*. In press.
[2] Carlson, H E, and Ippoliti, A F, *Journal of Clinical Endocrinology and Metabolism*, 1977, **45**, 367.