evidence, has emphasised the need to initiate now radical changes in the American diet, and among the steps it considers necessary to facilitate such changes has put in the first place "health and nutrition education in the classrooms and cafeterias of our schools."

Probably as a result of previous very vigorous health educational efforts American coronary mortality has for years been going down: only now may ours be starting to show a similar trend. For how much longer are we going to sit twiddling our thumbs and the knobs in our coronary care units?

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Is this uncertainty about the value of neurosurgery in this condition confined entirely to those over 59? Why does the report nowhere allow comparison with younger patients or with the outcome of those on whom angiography was not performed or unsuccessful?

Finally, with great temerity, may we question the authors' repeated assumption that those over 59 are in some way intrinsically of less value than younger patients?

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SIR,—I should like to congratulate Dr B V Martindale and Mr J Garfield on their paper (25 February, p 465). It is good to hear a note of restraint at a time when neurosurgeons everywhere are so preoccupied with technique and technology. The operating microscope, colour television, and videotapes threaten to become more important than the patients—the means more important than the ends.

I became convinced several years ago that it was safer to manage ruptured intracranial aneurysms in patients over 60 without surgery and all my experience since has supported this conviction. It is most valuable to have statistical evidence for a conservative approach in this group of patients.

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Testing monocyte function

Subarachnoid haemorrhage in patients over 59

SIR,—In reply to the article on this subject by Dr B V Martindale and Mr J Garfield (25 February, p 465) it must be emphasised that patients seen by surgeons need not necessarily undergo surgical operation but may require special investigations for both prognosis and management. With the increasing use of β -blocking agents in the treatment of the effects of intracranial arterial aneurysms and atherosclerosis there is an increasing need to discover in any given patient the exact nature of the intracranial arterial disease. Not all intracranial arterial aneurysms are suitable for surgical ablation, but the surgical neurologist is an eminently suitable person to discover the diagnosis and continue the management of such patients. Furthermore, the relief of intracerebral haemorrhage is occasionally very wise after the age of 60 years, giving patients good health and useful lives for many years afterwards. Finally, the cases of middle cerebral aneurysms over the age of 59 quoted in the article show a useful success rate with surgery (six out of eight cases) and the figures quoted for aneurysms involving the intracranial part of the internal carotid artery show a disappointing success rate not necessarily equal to those in series from other centres (but no indication of the exact operative technique is given).

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SIR,—The report by Dr B V Martindale and Mr J Garfield (25 February, p 465) demonstrates a refreshing desire to examine the possible diversion of resources from neurosurgery to the basic requirements of the elderly, but the report poses more questions than "Are intracranial investigations justified?"

Figures in the text indicate that of 28 highly selected patients subjected to surgery, 16 (57%) were capable of being independent in one year. Of 69 patients managed conservatively because of "unfavourable" or "adverse" factors, 29 (42%) were similarly alive and independent at one year.

Outline of method of measuring phagocytosis and intracellular killing of *C. albicans*.

at 30°C (instead of overnight at 37°C) gives a constant number of candida for 90 min when reincubated with serum at 37°C. Thus during this interval a microbiological assay gives reliable determination of the number of viable candida cells. After incubation of equal numbers of either granulocytes or monocytes with candida and serum at 37°C under slow rotation the decrease in the number of extracellular candida counted in a haemocytometer proved to be a reliable measure of phagocytosis. The decrease in the number of viable candida cells measured by a microbiological method after disruption of the cells accurately reflects the intracellular killing.

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¹ Territo, M C, and Cline, M J, *Journal of Immunology*, 1977, **118**, 187.

² Lehrer, R, and Cline, M J, *Journal of Bacteriology*, 1969, **98**, 996.

³ Schmid, L, and Brune, K, *Infection and Immunity*, 1974, **10**, 1120.

⁴ van Furth, R, et al, in *Handbook for Experimental Immunology*, ed D Weir, 2nd edn, ch 32. Oxford, Blackwell, 1973.

⁵ Leijh, P C J, et al, *Infection and Immunity*, 1977, **17**, 313.

Induction and neonatal jaundice in infants of diabetics

SIR,—In the management of diabetic pregnancy success is usually measured by a low perinatal mortality rate, which in good centres varies between 3 and 10%.^{1,2} However, neonatal problems are still common. Premature delivery may partly explain this finding, as even when optimal control of maternal diabetes and a normal infant weight for gestational age are achieved the neonatal morbidity rate remains around 50% among offspring of insulin-dependent diabetics.³ In recent correspondence in your columns (7 January, p 50; 20 January, p 239) the role of oxytocin and induction of delivery in relation to neonatal jaundice has been discussed.

At the Birmingham Maternity Hospital in a prospective study of 100 diabetic women, 84 of whom were on insulin, severe neonatal jaundice (serum bilirubin >250 μ mol/l (14.6 mg/100 ml)) affected 19% of infants and was the single most common neonatal problem. When an analysis of the data is carried out it emerges that severe jaundice was most common

However, other problems are encountered when phagocytosis and intracellular killing of candida are tested with the same methods as are used for bacteria.⁴ When an overnight culture of *C. albicans* is reincubated in the presence of serum (as required for a phagocytosis test) candida grows within 30 min by budding and formation of pseudomycelium. This causes clumping of the yeast cells and makes accurate counting in a microbiological assay unreliable because of underscoring. Secondly, because the density of the yeast cells is similar to that of the phagocytic cells separation of leucocytes from candida by differential centrifugation is impossible. Consequently the assays used for bacteria⁴ cannot be used for candida without modification.

These difficulties can be overcome with a recently developed method⁵ (see figure). Preculturing of *C. albicans* for at least five days

after induction and vaginal delivery (30%). Equal numbers of infants were delivered by induction or caesarean section, but the incidence of neonatal jaundice was significantly higher ($\chi^2=5.06$; $P<0.025$) in the former group. The mean time of delivery for the induction and caesarean section groups was 37 weeks and 36.5 weeks respectively, while infants delivered by the vaginal route were on average 0.4 kg heavier.

Vaginal delivery is generally the preferred mode of delivery for diabetics,² but it is important to recognise that when premature delivery is induced by this route severe neonatal jaundice is a common problem. On the other hand caesarean section has been associated with other neonatal complications which also represent a serious hazard to the newborn of the diabetic.

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¹ White, P, *American Journal of Obstetrics and Gynecology*, 1978, **130**, 228.

² Drury, M I, Greene, A T, and Strojge, J M, *Obstetrics and Gynecology*, 1977, **49**, 519.

³ Soler, N G, and Malins, J M, *Diabetes*, 1977, **26**, suppl 1, p 260.

College of Anaesthetists

SIR,—Since 10 distinguished anaesthetists have chosen to use your columns as a platform to discuss this controversial issue (4 March, p 574) I would be grateful for the opportunity to comment upon their letter.

The authors suggest that a letter recently circulated among anaesthetists by the Association of Anaesthetists "appears to have been prepared and printed" before the letter calling for unity was sent by the president and faculty deans of the Royal College of Surgeons of England to all fellows. If this is indeed the case, anaesthetists have a right to know the circumstances likely to have produced such a clumsy clash. The dean of the faculty sits by right not only on the council of the association but on all its important subcommittees. To my personal knowledge, since the "college referendum" in 1971/72, when I was honorary secretary, until at least last year successive deans have been closely consulted within the association on all matters relating to "the college issue" and their views accorded, quite rightly in my opinion, a disproportionate degree of weight when important decisions have been reached. On the other hand it is the president of the Royal College of Surgeons, not of the Association of Anaesthetists, who sits by right on faculty board meetings, and one can only assume that this arrangement is the cause of any lack of liaison within our own specialty. Perhaps our new "independent" faculty will find it possible to work as closely with its parent body, the association, as it does with its partners.

Your correspondents also appear to disapprove the association's encouragement to voters to elect to the board of the faculty those known to be in favour of a separate college. I had hardly supposed that my receipt of the college's letter a week or so before the faculty elections was pure coincidence. Indeed, the letter claimed as one of its purposes "to bring the message of the college's unit clearly before those who elect its governing council and boards of faculty"; at worst the association's approach could be considered unsophisticated to the point of being downright straightforward.

The president of the Royal College of Surgeons and the faculty deans have a right to request our loyalty, provided their information is accurate and their intentions are both sincere and clear. In their letter they state that the college council delayed its final request for

the new charter until it was known that the association's referendum had not resulted in an "approach to the college for the creation of an independent college." They fail to record that the referendum analysis did result in an approach by the Association to the College to discuss two suggestions: (1) That anaesthetists should have the same opportunity as surgical fellows to elect representatives, on an equitable numerical basis, directly to their college council. The only objection to this which had ever been raised—namely, that the college council was concerned not only in the college government but also in the supervision of surgical academic affairs, could be easily overcome by the creation of a faculty of surgery. The association recognised that such a change would take time, but felt that if true equality of fellows were ever to be seen to have been achieved a commitment along these lines was required. (2) That should (1) be unacceptable to the college eventual separation seemed inevitable. If this were the case would the college support the creation of an independent College of Anaesthetists within the existing college's physical framework?

I understood that both these proposals met with unequivocal rebuff and on these grounds switched my own allegiance from "a satisfactory long-term solution within the college" to "the inevitability of independence" and the establishment of an academic foundation to test its feasibility. Am I wrong? Can I expect the 10-year experiment to move towards one or other of these aims? Even today's experiments should have some purpose other than to occupy time. It is long since the winds of change blew a bit everywhere. Fearful of too radical an outcome, it is nevertheless time to put just the faintest breeze up Lincoln's Inn Fields.

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Papilloedema

SIR,—Your leading article (4 February, p 263) does well to reiterate that papilloedema from increased intracranial pressure was shown by Parsons¹ to be non-inflammatory. Along with this, and with insufficient emphasis, is the complete lack of interference with vision except in advanced cases, and this is its main differentiating feature from inflammatory papillitis as in multiple sclerosis. Because of this good vision the discussion on axonal transport is largely academic, as blockage of axonal transport is not consistent with continuance of normal function. Such blockage may be all very fashionable and play a big part in papillitis and ischaemic papillopathy. It probably plays some part in advanced papilloedema when vision does fail, but I maintain that it has no place in early papilloedema or in its causation. Clinically, visual loss is only a late effect and not an early one unless from incidental involvement of visual pathways by the tumour.

I had thought from your article that a reference² had been found which preceded my physiological theorising^{3,4} that there would be stasis anoxia of retrolaminar tissue from shunting of blood to the easier prelaminar tissue in states of raised cerebrospinal fluid pressure and that this shunt led to a breakdown of local autoregulative control which aggravated the effects. But no, the reference² related only to ischaemia from increased intraocular pressure,

which is prelaminar and practically the reverse of papilloedema. Much as I deprecate the suggested involvement of axonal transport in plerocephalic papilloedema up to the stage of loss of function, which is what usually pertains, my theory does at least offer a rational explanation for anoxic blockage of flow in advanced cases, and as far as I can see it is only when there is some degree of visual failure that one can get blockage of axonal flow.

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¹ Parsons, J H, *Pathology of the Eye*, vol IV, p 1349. Oxford University Press, 1908.

² Obstbaum, S A, and Podos, S M, *Investigative Ophthalmology*, 1974, **13**, 81.

³ Primrose, J, in *Vision and Circulation*, ed J S Cant, p 29. London, Kimpton, 1976.

⁴ Primrose, J, *Documenta Ophthalmologica*, 1976, **40**, 391.

Eruption during treatment with oxprenolol

SIR,—In the past two months we have seen two patients who have developed a distinctive eruption while receiving treatment with oxprenolol (Trasicor).

The two patients, a 77-year-old woman and a 49-year-old man, developed their rashes after receiving treatment for five months and two months respectively. The lesions consisted in both cases of well-defined red rings, some of which were scaly or eroded, on the trunk, face, and limbs. Psoriasiform features were seen on the lower limbs in the second case. Histological examination revealed lichenoid changes with basal cell liquefaction degeneration, intraepidermal necrotic cells, and colloid body formation.

Within the past two months we have also received biopsy specimens from two further patients developing rashes during oxprenolol treatment which have shown similar histological changes.

A rather similar eruption due to oxprenolol has previously been described;¹ our cases will be more fully reported in the near future. They may reflect increased usage of oxprenolol because of anxiety about the safety of other beta-blockers. A preliminary communication is being made here because we are concerned that this probable side effect should be recognised early, perhaps allowing more serious side effects to be avoided.

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¹ Holt, P J A, and Waddington, E, *British Medical Journal*, 1975, **2**, 539.

Psychological evaluation in cases of self-poisoning

SIR,—Dr R Gardner and his colleagues at Addenbrooke's Hospital, Cambridge (17 December, p 1567) suggest that patients who take a drug overdose deliberately need not be referred routinely to a psychiatrist but that physicians should decide if specialist psychiatric advice is necessary. I should like to present some information which I feel is relevant to their suggestion.