Anaesthesia: The Final Act" (24 May, p. 408) have fallen into the same trap that many, including myself when a member of the British Dental Association Anaesthetic Subcommittee,¹ have fallen into previously. None has defined "anaesthesia," "operator-anaesthetist," "qualified," "singlehanded," or even "emergency," and most have used a document² which was statistically suspect and rejected as not being authoritative as long ago as 1967.³ Those involved in this controversial and emotive topic usually have their own interpretation and often diversified mental pictures of "anaesthesia in dentistry."

As one often privileged to lecture on the subject to postgraduates, may I suggest that the presence of "a team" used to working together, all trained in the care of the unconscious patient and resuscitation procedures, with constant pulse and respiratory monitoring in operating theatres specifically designed for such work, may be ideal. Logically, the patient would be at an advantage over one who was being relieved of pain by a dental surgeon with no anaesthetic training in a sparsely equipped surgery in a conventional dental chair with a visiting anaesthetist who seldom gives, and has little interest in, a dental anaesthetic. It is not difficult to find consultant anaesthetists and their wives and general medical practitioners and their families who have experienced and enjoyed the former environment or, for that matter, dentists who are forced reluctantly to accept the latter.

I submit that the team's training, experience, skill, and equipment are the criteria, and such loose terminology as "fully qualified" means little in our present training set-up. Allowing patients to "go to sleep" for their dentistry (be it anaesthesia, sedation, relative analgesia—call it what you may) is now such a well-established practice in Britain that the public is unlikely to be deprived of it easily. The heart of the matter is not "operator-anaesthetist" but the lack of a real will by the establishment to improve training facilities.

Eleven years ago I, with many others, pleaded for better undergraduate and postgraduate training.⁴ The need was officially expressed in the document² referred to in your leading article—every subcommittee on dental anaesthesia has been requesting it for over a decade—and the Society for the Advancement of Anaesthesia in Dentistry has pressed for it since its inception. More than a year ago the dean of dental studies, lecturers in dental surgery, anaesthetics, and dental anaesthetics, and the professor of community care and general practice of the University of Sheffield put forward a scheme to improve training,⁵ and what has been done?—effectively nothing! In the interests of all, is it not time to remove buried heads from the sand, break partisan barriers, act, and stop uttering platitudes?—I am, etc.,

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¹ *British Dental Journal*, 1968, 124, 206.

² Ministry of Health, *Dental Anaesthesia, Report of Joint Subcommittee of the Standing Medical and Dental Advisory Committee on Dental Anaesthesia*, London, H.M.S.O., 1967.

³ British Dental Association, *A.G.M. Report*, 1968.

⁴ *The Teaching of General Anaesthesia for Dental Surgery*, Symposium, 4 April 1964, Royal College of Surgeons of England.

⁵ *British Dental Journal*, 1974, 136, 155.

Giardiasis in Families

SIR,—Adoption of children from outside Europe is common in Sweden and these children, as part of a health check, have their faeces examined for parasites. A not infrequent finding is *Giardia lamblia* cysts. One child seen here recently was repeatedly positive for this parasite in spite of treatment. The adopting parents were examined and found to be also positive; all three persons were treated and there has been no further trouble.

Because of this finding we now regularly check family members when an adopted child is positive for *G. lamblia*. To date this year five out of a possible six families have had some or all other members affected, with or without symptoms. The infestation rate varied between 50% and 100%. In one case the *G. lamblia* infection may have come the other way: the adopting parents had visited the Soviet Union some time before travelling to Thailand to collect the child. In addition, all the immediate family of a Swedish mentally retarded child with giardiasis were found to be infested with the parasite and Aust Kettis and Thorén¹ have recently described an Israeli child apparently responsible for infecting five other families, one of these at second hand.

These observations emphasize the infectiveness of *G. lamblia*, show the necessity of checking contacts of patients with giardiasis, and also draw attention to the problems which may arise if the condition is missed.—I am, etc.,

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¹ Aust Kettis, A., and Thorén, G., *Scandinavian Journal of Infectious Diseases*, 1974, 6, 349.

Fibre Content of Bread

SIR,—Mr. C. L. Copeland (31 May, p. 503), who is the Executive Director of the Flour Advisory Board, alleges that there were two inaccuracies in the B.B.C. T.V. Horizon programme on roughage.

For some two years in 1972-74, led by my surgical colleague Mr. Denis Burkitt, F.R.S., a group of doctors, all of whom are contributors to our book,¹ have been the guests of Mr. Copeland and his scientific staff, joining in mutual friendly discussions. For our part we have learnt much during these discussions and in our papers have produced subsequently the millers' own assessment of the crude fibre content of the flour in the middle of the last century, though the best authorities had previously stated that even "the best quality 'white flour' prior to the middle of the nineteenth century was a wholemeal flour, produced by stone grinding, from which the coarse particles of the bran were removed by 'bolting' through fine linen or woollen cloths."² Possibly the producers of the B.B.C. programme can be excused for relying too exclusively on this earlier statement and not referring to a recent communication by Dr. M. A. Eastwood and the millers.³

Mr. Copeland states that the second inaccuracy concerned the date of the change in the fibre content of the war-time bread. He writes that "the higher fibre content of flour was not introduced until April 1942"

and refers to "official records" but cites no reference. On 26 October 1939 the extraction rate was raised from the previous peace-time level of 70% to 73% and on 27 April 1941 to 75%, thereby increasing the crude fibre content from 0.10% to 0.15% (unpublished Ministry data, courtesy of Dr. C. T. Greenwood, National Association of British and Irish Millers). Early in 1941 National flour 85% extraction, crude fibre at first varying from 0.4 to 1.3%,⁴ was milled throughout Britain, but only a small minority ate it as it was not liked by the public and was strongly opposed by the millers. In April 1942 the British Government made the National flour compulsory. In my opinion, stated in detail elsewhere,⁵ the cereal crude fibre content rose significantly during 1941; this did not wait until 1942 as stated by Mr. Copeland.

Mr. Copeland is indeed correct in stating that a "lower incidence of diseases from 1939" could not be attributed to an increased fibre content of the war-time flour, which occurred in 1941, or according to him in 1942. Death rates from diverticular disease, however, which had been almost stationary since 1937, rose again from 1953 until about 1960, this rise coinciding with the termination of the compulsory National flour in 1953. Diabetes mellitus death rates fell by 55% from 1941 until 1953-56; this coincided completely with the years of compulsory consumption (1942-53) and voluntary consumption (1941 and 1954-56) of the high-fibre National flour.⁶—I am, etc.,

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¹ *Refined Carbohydrates and Disease: the Implications of Dietary Fibre*, ed. D. Burkitt and H. Trowell, London, Academic Press, in press.

² Hollingsworth, D. F., Drummond, J. C., and Wilbraham, A., in *The Englishman's Food*, p. 43. London, Cape, 1958.

³ Eastwood, M. A., et al., *Lancet*, 1974, 1, 1029.

⁴ *Nature*, London, 1942, 149, 460, and 150, 538.

⁵ Trowell, H., *Proceedings of the Nutrition Society*, 1973, 32, 151.

⁶ Trowell, H., *Lancet*, 1974, 1, 998.

Vagotomy, Antrectomy, and Duodenal Ulcer

SIR,—We would like to comment on several points which were raised by Drs. P. Madsen and O. Kronborg in their letter (21 June, p. 688) about our paper "Highly Selective Vagotomy for Duodenal Ulcer: Do Hypersecretors Need Antrectomy?" (29 March, p. 716).

First of all, we are glad that they have not recommended antrectomy (plus vagotomy) as an alternative to H.S.V., but we fear that many will conclude that antrectomy is indeed necessary in a considerable proportion of patients in the light of their staggering recurrence rate of 22% after H.S.V.¹

Drs. Madsen and Kronborg express surprise that we did not comment on the high frequency (51%) of positive insulin tests which we found in our patients one year or more after H.S.V. This is easily explained: we thought it was irrelevant. Far from hiding the fact, we were the first to describe it² and indeed we are about to publish a sequel which shows that 3-5 years after H.S.V. almost 100% of patients have developed positive responses to insulin. But that does not seem to matter, because in Leeds,³ some parts of Copenhagen,³ and

Reference	No. of Patients	% with Positive Insulin Tests One Week After H.S.V.	% Developing Recurrent Ulcer	Comment
Kronborg and Madsen ¹	50	58	22	High incidence of incomplete vagotomy; high incidence of recurrent ulceration.
Wastell <i>et al.</i> ⁵	16	28	13	Intermediate incidences both of incomplete vagotomy and of recurrent ulceration.
Kennedy <i>et al.</i> ⁴	50	4	2	Low incidence of incomplete vagotomy, low incidence of recurrent ulceration.
Johnston and Goligher (unpublished)	100	3	1	Low incidence of incomplete vagotomy, low incidence of recurrent ulceration.

Belfast,⁴ at any rate, it has been found that very few of these patients develop recurrent ulceration.

What does matter, in our opinion, is the insulin test that is performed 5-10 days after H.S.V. (see table) because the results of this test seem to have a considerable bearing upon the subsequent incidence of recurrent ulceration. Drs. Madsen and Kronborg say that their acid secretion figures obtained from this test "revealed no relationship between the completeness of H.S.V. . . . and recurrent ulceration." They seem to deduce from this that no such relationship exists. An alternative explanation—and a much more likely one in our view—is that the method which they employed was not adequate to reveal the relationship. Specifically, we think that they need more data on larger numbers of patients. It is striking (even if not statistically significant) that no fewer than 10 of the 11 patients in Drs. Madsen and Kronborg's series who developed recurrent ulceration after H.S.V. showed some secretory response to insulin 10 days after operation and that their mean acid response to insulin (minus basal) was 4.9 mmol (mEq)/h, which is quite a big response. In contrast, the mean acid response to insulin (minus basal) after H.S.V. in our first 100 patients in Leeds was 0.1 mmol/h. If Drs. Madsen and Kronborg were to look at their own and others' results in a different way (table) we feel that they might reach a different conclusion about the prognostic value of the insulin test that is performed soon after operation. As shown in the table, Drs. Madsen and Kronborg's patients had a significantly higher incidence of positive insulin tests after H.S.V. and significantly more recurrent ulcers than our patients in Leeds or the patients of Kennedy *et al.* in Belfast. Post hoc ergo propter hoc? We certainly think the latter.—We are, etc.,

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- 1 Kronborg, O., and Madsen, P., *Gut*, 1975, **16**, 268.
- 2 Johnston, D., *et al.*, *Gastroenterology*, 1973, **64**, 12.
- 3 Amdrup, E., *et al.*, *Annals of Surgery*, 1974, **180**, 279.
- 4 Kennedy, T., *et al.*, *British Medical Journal*, 1975, **2**, 301.
- 5 Wastell, C., *et al.*, *British Medical Journal*, 1972, **1**, 28.

Variations in Leucocyte Count during Menstrual Cycle

SIR,—The recent paper by Drs. Barbara J. Bain and J. M. England (31 May, p. 473) relating variations in leucocyte counts during

the menstrual cycle to cyclical changes in endogenous oestrogen levels prompts me to report some recent experimental observations.

Magarey and Baum¹ described an increase in the phagocytic activity of the reticulo-endothelial system in patients receiving oestrogen therapy. Expanding on this initial finding I have postulated that the benefit of endocrine manipulation for certain human cancers may in part be related to stimulation of host factors.² The macrophages that populate the sinusoidal system of the liver and spleen, and in addition take part in a wide variety of cellular immune mechanisms (including the host response to cancer), arise from rapidly dividing precursors in the bone marrow. The new formed cells discharged from the marrow appear as monocytes in the peripheral blood before taking on a variety of morphological characteristics depending on which site within the reticulo-endothelial system they are eventually found.³ It would seem reasonable to assume, therefore, that oestrogen would stimulate these precursor cells with the resulting monocytosis providing an enlarged peripheral pool of phagocytic cells capable of clearing injected particulate matter from the blood.

To test this hypothesis I have studied the proliferation of macrophage precursors in mice treated with oestrogen. Male Swiss T.O. mice were injected intraperitoneally with 10 µg of oestradiol benzoate in 0.1 ml of arachis oil. Twenty-four hours after the injection the mice were killed and bone marrow was harvested from the femurs under aseptic conditions. In each experiment the marrow from three animals was pooled and aliquots containing 10⁵ nucleated cells were added to each of a replicate series of 10 tissue culture plates containing a semisolid medium prepared according to the method of Bradley and Metcalf.⁴

After seven days' incubation under controlled conditions the numbers of macrophage colonies per plate were counted, giving an index of the number of monocyte/macrophage precursors proliferating at the time of sacrifice. Control animals injected with 0.1 ml of arachis oil were studied in the same way. The experiment was repeated four times, providing 40 observations. The results are summarized in the table:

	Mean Colonies per Plate (±S.D.)
Oestrogen group	127±63
Controls	191±86

t=3.68, P<0.01

The results suggest that oestrogen in the dose used, far from increasing the number of macrophage precursors dividing, may actually have the reverse effect. The only

explanation for this unexpected finding that occurs to me is that oestrogen promotes the release of mature monocytes from the marrow reserve (as suggested by Drs. Bain and England) and that the increased peripheral pool of monocyte/macrophage produces an inhibitory factor, as has already been postulated by Ishikawa *et al.*⁵

Whatever the complete explanation for the effect of oestrogen on the mononuclear phagocytic system, there is no doubt in my mind that the endocrine system plays an important role in regulating cellular immunity and that the inter-relationship of these systems remains to be unravelled. Such research would be of the utmost value to clinical oncologists and transplant surgeons, as well as to haematologists.

I wish to acknowledge the invaluable technical assistance of Mrs. Maree Breese and the generous financial support of Tenovus, Cardiff.

—I am, etc.,

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Welsh National School of Medicine,
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- 1 Magarey, C. J., and Baum, M., *British Medical Journal*, 1971, **2**, 367.
- 2 Baum, M., in *Host Defence in Breast Cancer*, ed. B. A. Stoll, p. 130. London, Heinemann, 1975.
- 3 Leder, L. D., *Blut*, 1967, **16**, 86.
- 4 Bradley, T. R., and Metcalf, D., *Australian Journal of Experimental Biological and Medical Science*, 1966, **44**, 287.
- 5 Ishikawa, Y., Dluznick, D. H., and Sachs, L., *Proceedings of the National Academy of Sciences of the U.S.A.*, 1967, **58**, 1480.

Rewards of the Academic Career

SIR,—Dr. R. A. North says (7 June, p. 555) that "academic professorships" are paid almost as well as "consultantships." They are not. In this university the differential is at least £3500 per annum in favour of the consultant.—I am, etc.,

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Dying for a Number

SIR,—"No man ever died for a number" (Dr. J. S. Bradshaw, *Personal View*, 14 June, p. 611). Oh didn't he?

Just read what Sir John Fortescue, not normally a writer much given to emotional writing, had to say about the Battle of Albuera (1811). This was a "soldiers' battle" if ever there was one. The Portuguese had made Beresford a marshal. God had not made him a great general. It was all up to the soldiers; "and hence it was that when one man in every two, or even two in every three, had fallen in Hoghton's Brigade, the survivors were still in line by their colours, closing in towards the tattered silk which represented the ark of their covenant—the one thing supremely important to them in the world." For me, these are among the most moving words ever written in the English language.

The King's or Queen's colour is important; but the ark of the covenant is the regimental colour. In those days of our small army's growing fame this, and many another scrap of tattered silk, bore a number.—I am, etc.,

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