After a somewhat belated diagnosis of carcinoma (should not all rectal bleeding be considered due to malignant disease until *proved* otherwise?) I found myself with a colostomy and many of the recognised complications of abdominoperineal resection. Surprisingly I have found the colostomy the least of the problems and, after an initially traumatic period of adjustment, find its presence, far from being "an affront difficult to bear," only a slight inconvenience. I suspect that the reason for this may be found in the attitudes of those around me, and in particular those of my family and the various doctors and nurses who have looked after me. There is no shortage of evidence suggesting that adjustment to disease is in part dependent upon attitudes and milieu. I would submit that rather than making general statements upon the quality of life after radical surgery or other treatments we should, as a profession, assess each case individually, looking at the patient's personality, his social situation, family, etc, when trying to assess how such a person may cope. I am sure the same applies when deciding what to tell the patient about his malignant disease.

The value of 'ostomy associations will also vary between patients, for what may be "unhealthy introspection" for one may be a lifeline of support for another. The various problems related to colostomies referred to in the article seem to be more familiar to association members than to the medical profession, and thus so are the solutions. In this respect, how sad it is that so many surgeons refuse to make use of stoma therapists who share that source of knowledge.

Where I am most in agreement with Professor Dudley is in his statement that doctors or nurses still need a one-to-one relationship with their stoma patients. It is remarkable how much variation there can be from one professional to another in his attitude to patients with malignant disease, and I suspect that very many doctors have no idea how important their every word and gesture can be. Do we realise that our patients can dwell for hours and days on the significance of a particular statement we may have made?

By all means perform the surgery most likely to preserve life, but it is only then that the real work begins. How much more meaningful to me now is the statement made by one of the surgeons at my own alma mater—"any fool can do an operation, it's the pre- and post-operative care that counts."

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SIR,—I found myself in general agreement with Mr Brian Harcourt's approach to the treatment of cataract (29 April, p 1121) and was pleased to note that intraocular lens implants to rehabilitate the aphakic patient were considered. I would question, however, the arbitrary age of 65 as representing some kind of watershed in this matter. Either a patient needs pseudophakic surgery or he doesn't, and age is irrelevant. Indeed, with great respect to Mr Harcourt, my experience is that it is the younger patients, notably those with unilateral cataract/aphakia, whose need is greatest in order to achieve optimum visual performance. I suspect that many elderly patients with bilateral cataracts are adequately cared for by conventional cataract surgery followed by careful refraction and dispensing of aphakic

spectacles, which enable them to feed themselves, find their way to the bathroom, look at television, and read. Not that I would deny them the benefits of pseudophakia, far from it.

Mr Harcourt also expresses reservations about the long-term effect of the plastic within the eye. How long is long? I have under my care a patient who received a posterior chamber lens from Mr Harold Ridley in 1952 and an anterior chamber lens from me 20 years later. Both eyes are splendid and I know of many others. One thing we can be sure of is that the plastic (in this case Perspex CQ, made by ICI) is harmless within the eye up to 29 years.

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SIR,—I wonder why Mr Brian Harcourt (29 April, p 1121) thinks ophthalmic surgeons "sensibly" avoid calling a lens opacity a cataract. Certainly I, for one, was angry to be told by a friend that I had one after years of trying to get an adequate prescription for my right vision—I have always had moderately severe mixed astigmatism.

Events caught up in that before I got round to surgery my left lens started a central opacity—more or less blind within days. And so to bilateral surgery at a two-month interval—so successful that I wear hard contact lenses to provide all-round 6/6, 6/5 vision. I wish I had had more positive advice years earlier.

Putting the first lens in did bother me a lot. I cut the bottom half off one side of an old pair of frames and had a reading lens put in the remaining side, which I then used with the aid of a mirror for the close-up exercise of putting in the first lens, using that one for putting in the other.

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SI, moles, and drugs

SIR,—A recent leading article (18 March, p 668) drew attention to some of the difficulties encountered when expressing drug concentrations in molar units. Our experience, working in an establishment which undertakes over 20 000 drug assays annually as well as providing information on the treatment of drug intoxication, suggests that such a departure engenders considerable confusion among both clinicians and laboratory staff.

A typical incident occurred recently. We were telephoned for guidance on the management of a patient poisoned with barbiturates. The local hospital laboratory had reported a blood barbiturate concentration of "230," which the clinician dealing with the patient assumed to be in terms of mg/l. Since an intermediate-acting barbiturate drug was involved we questioned the validity of this result. Unfortunately, we were contacted outside of normal laboratory hours and considerable delay ensued before the biochemist who had performed the assay could be traced to confirm that the units were, in fact, $\mu\text{mol/l}$ and that the concentration in mass units was 52 mg/l. By this time the patient's condition had, fortunately, improved. It can be argued that such confusion is of little consequence in the majority of poisoning cases, in which treatment is largely supportive. However, when active therapy must be instituted rapidly and where

the decision to do so hinges predominantly on toxicological data, as, for example, in paracetamol poisoning, such delays cannot be tolerated. Fortunately, plasma paracetamol levels expressed in $\mu\text{mol/l}$ are numerically greater than the equivalent in mass units (mg/l). It is doubtful, therefore, whether the adoption of the molar system has led to the withholding of antidotal therapy from such patients at risk. That the converse has occurred is more than likely, however. To cite an example, a clinician dealing with a paracetamol-poisoned patient was told that the plasma concentration in a sample taken 5 h after ingestion was "600." Being aware of the recommendation by Prescott *et al*¹ that intravenous cysteamine should be administered to patients presenting with levels of 300 mg/l or more at this time he telephoned to ascertain the cysteamine dosage required. A query was raised about the laboratory result, whereupon it was learned that the equivalent mass concentration was a rather insignificant 90 mg/l. Thus the unnecessary administration of an antidote which can precipitate considerable distress was fortunately avoided. The toxicological criteria for instituting parenteral desferrioxamine in acute iron poisoning have been similarly obscured by the change to molar units. Since we ourselves are not immune from such confusion and are increasingly called upon to act as mathematical interpreters, we now find it necessary to carry conversion charts at all times.

In the notable absence of a national (or supranational) ruling on this point by any authorised bodies it perhaps falls to organisations such as ours to voice the opinions of the vast majority of analysts and clinicians, who find the application of molar units in this context to be both fatuous and impracticable. By all means let us universally adopt the mass SI units of grams (or subdivisions thereof) per litre for drug concentrations. However, until such time as drugs are prescribed, or can be obtained from the pharmacist, in moles the subsequent provision of toxicological data in these terms has, in our view, no merit whatsoever.

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¹ Prescott, L F, *et al*, *Lancet*, 1974, **1**, 588.

Inhibition of fibrinolysis and abruptio placentae

SIR,—The case report by Dr B Åstedt and Professor I M Nilsson (25 March, p 756) has unfortunate implications which the authors should have emphasised. Since they run a coagulation laboratory with an international reputation, no doubt the described therapy was safe in their hands. Without such facilities fibrinolytic inhibition in pregnancy is extremely dangerous.

In pregnancy there is normally a quite profound inhibition of fibrinolysis,¹ which I have shown to depend on the hormone lactogen,² and of course this low level of fibrinolytic activity is essential to prevent thrombosis in the microcirculation if thromboplastin should be released from a damaged placenta. Deliberate inhibition of fibrinolysis in both man³ and the rabbit⁴ can lead to renal cortical necrosis.

The patient referred to seems to have been unusual. It is thought that activation of fibrinolysis during pregnancy is uncommon. Indeed this is the very reason why the classic disseminated intravascular coagulation syndromes were first recognised in human pregnancy. Could it be that this patient lacked one of the normal fibrinolytic inhibitors that are known to act either locally⁵ or in the general circulation? If this is the case then the extrapolation is unjustifiable.

I feel that we need clarification of this case or more work by an expert unit before this suggestion is put into widespread practice.

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- ¹ Lauritsen, O S, *Scandinavian Journal of Clinical and Laboratory Investigation*, 1969, **23**, 191.
- ² Wardle, E N, *International Research Communications System*, 1973, **12**, 8-8-8.
- ³ McKay, D G, *Disseminated Intravascular Coagulation*. New York, Harper and Row, 1965.
- ⁴ Wardle, E N, and Wright, N, *American Journal of Obstetrics and Gynecology*, 1973, **115**, 17.
- ⁵ Stark, J M, *Lancet*, 1978, **1**, 417.

* * We sent a copy of this letter to Dr Åstedt and Professor Nilsson, whose reply is printed below.—Ed, *BMJ*.

SIR,—Judging from our experience the fears entertained by Dr Wardle about the risk of inhibition of fibrinolysis in pregnancy go too far.

We are well aware of the fact that fibrinolysis decreases during pregnancy¹ but rapidly returns at delivery after separation of the placenta, and we have also shown the significance of the placenta in the depression of fibrinolytic activity.² Our case of abruptio placentae associated with activation of the fibrinolytic system (25 March, p 756) is not an example of an uncommon complication. The patient did not lack any of the known inhibitors. In a report by Edgington,³ who used the cleavage-associated neoantigen of fibrinogen for differentiating between degradation products derived from fibrinogen and fibrin, he found in all his cases of abruptio placentae that the degradation products were derived from fibrinogen only, indicating activation, above all, of the fibrinolytic system. This is in agreement with our findings in a series of 14 consecutive cases examined with coagulation factors and fibrinolytic components, in which we found fibrin/fibrinogen degradation products (FDP) decreased levels of fibrinogen and plasminogen, while the platelets, P and P complex (factors II, VII, and X), and ATIII were normal. In some patients followed up for a long time during treatment FDP disappeared and the levels of fibrinogen and plasminogen rose. The trigger mechanism presumably consists in liberation of plasminogen activators from the uterus, which is known to be rich in such enzymes, combined with reduced influence of the placenta when partly separated.

For the apparently paradoxical non-thrombogenic effect there are probably several explanations. In a series of patients receiving tranexamic acid for two weeks in a dose of 4 g daily we examined histochemically the fibrinolytic activity in vein biopsy specimens before and on the last day of treatment. Tranexamic acid did not suppress the content of fibrinolytic activators in the vessel wall.

The perinatal mortality in abruptio placentae is about 35% and many children (fetuses) are dead on admission. Using tranexamic acid

routinely in cases of abruptio placentae perinatal mortality in recent years has been 8% in our department. We have not had any complications of haemorrhagic diathesis nor have there been any thrombotic complications.

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- ¹ Åstedt, B, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1972, **79**, 205.
- ² Åstedt, B, et al, *Acta Obstetrica et Gynecologica Scandinavica*, 1970, **49**, 171.
- ³ Edgington, T S, *Thrombosis et Diathesis Haemorrhagica*, 1975, **34**, 671.

Hairy-cell leukaemia

SIR,—It was a pleasure to see the current interest in hairy-cell leukaemia (HCL) reflected in your recent publication of a useful leading article (8 April, p 872) on this fascinating disease. However, our pleasure was tempered by the misinterpretation of our own work on the subject¹ and by the rather scant discussion of the considerable controversy surrounding the nature of the hairy cell (HC).

You state that we considered the HC to be "a unique cell type, possibly representing a leukaemic proliferation of monocytes." Although many authors have agreed that HCs are related to cells of the monocyte-histiocyte series,² we in fact stated that "these arguments have never been especially cogent" and that our description of a receptor for IgM on HCs, but not on monocytes, "further distinguishes hairy cells from monocytes." HCs possess a number of characteristic, if not unique, properties,³ and we believed that their possession of an IgM receptor further set them apart from both B cells and monocytes. However, this view is no longer tenable since the cells of chronic lymphocytic leukaemia (CLL), but not of a variety of other B-cell or monocytic proliferative disorders, have also been shown to bear a receptor for IgM.^{4,5}

Synthesis of surface membrane immunoglobulin (SmIg) is an essential feature of B cells. However, in the demonstration of SmIg stringent technical precautions are necessary to avoid the problem of extrinsic IgG binding by Fc receptors. When these precautions are observed only a very small population of normal B lymphocytes express IgG SmIg.⁶ In much of the early literature on HCL no account was taken of these technical considerations, and the many reports showing apparent SmIg of both light-chain types and mixed heavy-chain isotypes, including IgG, led to scepticism^{1,2} about whether HCs express genuine intrinsic SmIg, more especially since the cells express a strong Fc receptor.^{1,2} Recently, however, we and others,^{7,8} by using sensitive methods and observing stringent technical precautions, have shown that HCs synthesise monoclonal SmIg which may be IgG alone or of multiple heavy-chain isotypes, including D, M, G, and A. These results explain the previous controversy concerning the true nature of HC SmIg and also seem to place HCs late in any scheme of B-cell ontogeny.⁶

In conclusion, the present state of knowledge suggests that HCL represents a clonal proliferation of an unusual form of B lymphocyte. If there is a normal counterpart to the HC it has yet to be identified unequivocally, but it is presumably an IgM-receptor-bearing cell appearing late in B-cell development, possibly at the stage of activated memory cells

since these latter cells have also been shown to synthesise IgG SmIg. The different but characteristic clinical pictures associated with other B-cell neoplasms such as CLL and multiple myeloma, which may represent clonal proliferations of cells blocked at other stages of B-cell development, suggest that these observations on the nature of the HC may be relevant to the distinctive clinical picture of HCL.

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- ⁵ Pichler, W J, and Knapp, W, *Journal of Immunology*, 1977, **118**, 1010.
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- ⁷ Burns, G F, et al. Submitted for publication.
- ⁸ Rieber, E P, et al, in *Immunological Diagnosis of Leukemias and Lymphomas*, ed S Thierfelder, H Rodt, and E Thiel. Berlin, Springer, 1977.

* * Mr Burns and his colleagues are correct in pointing out that what they really said in their article was that leukaemic reticuloendotheliosis may be a leukaemic proliferation of a recently recognised population of circulating mononuclear cells which differ from cells of the monocyte/histiocyte series. Their letter is a valuable elaboration of the comments on the nature of the hairy cell, which was, of necessity, dealt with briefly in the leading article.—Ed, *BMJ*.

Duodenal erosions on cimetidine therapy

SIR,—A patient with multiple gastric and duodenal erosions developing while on cimetidine was reported by Dr J Webster and others (7 January, p 20). We would like to report a further case.

A 57-year-old man was admitted with haematemesis and melaena. He smoked 30 cigarettes a day and drank one bottle of sherry and a quarter of a bottle of whisky a day. He had taken Alka-Seltzer tablets (aspirin, anhydrous citric acid, and sodium bicarbonate) in the 24 hours prior to admission. There was no relevant past medical history. On examination he had five spider naevi, hepatomegaly, and leuconychia. Endoscopy revealed four gastric erosions on the greater curve of the stomach with evidence of recent bleeding. The haemoglobin concentration was 12.6 g/dl and the bleeding settled with conservative management, including cimetidine given initially intravenously and then orally. Liver biopsy showed micronodular cirrhosis with fatty change in keeping with his disturbed liver function tests. He received cimetidine (200 mg thrice daily and 400 mg at night) for a total of 60 days.

Five months later the patient complained of abdominal pain suggestive of a duodenal ulcer. Endoscopy revealed a duodenal ulcer 5 mm in diameter. Treatment was commenced with full doses of oral cimetidine, with symptomatic relief. Twenty-one days later he was readmitted with haematemesis and melaena. The haemoglobin concentration was 7.6 g/dl and liver function tests were normal. Endoscopy revealed a normal stomach and oesophagus with multiple duodenal erosions in the first and second parts of the duodenum, many of which appeared to have bled