Listening and talking to patients

SIR,—I was delighted to read the first of the articles (27 September, p 845) by Professor Charles Fletcher on the issue of listening and talking to patients. Medical students learn their medicine by following the example of their seniors. While every attention is paid to the acquisition of the facts about the physical condition, too often insufficient attention is paid to associated emotional problems. A recent workshop run in association with the Association of Medical Education in Europe debated these difficulties and ways of improving the training of medical students in the understanding of the emotional aspects of patients' conditions. The following transcript is instructive.

"I went to the surgery and the first thing that put me off was the fact that I came in at the door and he was standing with his back to me and made no indication as to whether I should come in and sit down or not, but he did eventually turn round and I sat down and he examined my knee. He wasn't very forthcoming with information." "Did he tell you what was wrong with you?" "No, he didn't, but I asked questions. I thought he would tell me what was wrong and I kept asking questions but I didn't feel at the end that I knew what was wrong. There were also other things that rather put me off—I am not normally lost for words and I probably did not ask questions that I should have done." "Did he tell you about the treatment?" "No—in fact, he just said, 'That's it.' I said, 'Can I carry on cycling?' and he said, 'Yes.' I wasn't even sure whether it was time to go or not. I got up, walked to the door, and said, 'Is there any treatment? Shall I put anything on it or have physiotherapy?' He said, 'No.'" "Did you feel he had made an adequate physical examination?" "Well, yes, I suppose so and since then I have realised that it has slowly got better and therefore there was no treatment; it was just that in myself I felt that the whole interview was very dissatisfying."

Clearly it is not enough for the doctor to be satisfied that a complaint of pain requires no active treatment.

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Admission of old people to psychiatric units

SIR,—Dr Ian Pullen's observations on admission rates of old people to psychiatric units (27 September, p 843) are extremely important. The policy of freezing psychiatric inpatient provision for people suffering from dementia at the level current in 1971, expressed as a ratio of beds to numbers at age 65 years and older, has drastically reduced the psychiatric accommodation available to demented patients, whose number has increased as the proportion of very old has increased.¹ Alternative forms of residential care, notably "part III" homes, have responded to this pressure by accepting or retaining greater numbers of very disabled people.² Yet this redistribution of roles has been achieved by (often fraught) negotiations at practitioner level, which can seek only to get the best possible out of what is a bad job.

While agreeing that more facilities must be provided, we would suggest that the residential and continuing care components should be considered separately from assessment and treatment requirements. Appropriate siting and organisation will allow more effective use of any increase in capacity, and further

development of isolated mental hospitals or geriatric hospitals would be wasteful.

At the present time this country is littered with innumerable undeclared scandals, some of which will come to public notice when chance clash of local personalities generates enough heat to titillate the media. It is essential that people receiving and giving services to mentally ill old people are protected by an adequate and responsible monitoring system that reflects provision inside and outside institutions, both hospital and non-hospital, and can mobilise an effective response when problems are identified. Such issues must not be obscured by justifying the neglect of elderly people on grounds of economic imperatives.

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¹ Jolley D. *Br Med J* 1977;i:1335-6.

² Wilkin D, Mashiah T, Jolley D. *Br Med J* 1978;iii:1274-6.

SIR,—Dr Ian Pullen's article (27 September, p 843) was significant in itself and opportune in drawing attention to the crisis—that is, the "time of danger or decision or both"—in the care of the elderly. Factors to be considered pertaining to the 1970 "fall" in the admission rate of elderly patients appear to be as follows:

(a) As observed, there was an initial effect of increased local authority (Part III) places during the past decade, together with various other day places. Now these are usually filled, their residents and patients progressively aging and necessary ambulance transport not always being available.

(b) There was an innovatory effect of various psychogeriatric combined endeavours in the community and hospitals, together with community psychiatric nurse and social worker help. These are very useful, but still leave a high proportion of patients requiring indefinite hospitalisation.

(c) The admission-blocking "stigma effect" (where present) faded because of hospital upgrading, admissions being not only accepted but entreated by often genuinely overwhelmed relatives and friends. Many of us are involved in this heartbreaking situation where extensive and varied efforts by conscientious and concerned GPs, hospitals, and other agencies often prove inadequate. Tolerance and endurance are at breaking point and dedicated "supporters" understandably insist that "something must be done," although we have no beds available.

(d) No doubt realistically, our area figures confirm the most important contributory element—that is, people are living longer in the community, in Part III accommodation, and on our wards. Whatever the reason, the "through-put" (an unpleasant administrative expression) has been greatly diminished and admissions reduced and sufferers languish in the community or "block" acute beds.

(e) Important in some areas is the actual reduction in available hospital beds owing to financial restrictions and other causes. District general units attract staff away from and "demote" the status and attractiveness of well-run and spacious psychiatric hospitals "because there's little or no acute work and they're like old people's homes."

The later article on the ethical problems of feeding demented patients (27 September, p 847) properly focused on the interpersonal "care and caring-for" question, but simultaneously and pathetically reflects on the greater problem and dilemma described above. Where does "striving officiously" begin? Who is "responsible" if an old lady wanders, causes a road traffic accident, freezes to death, or assaults an enfeebled spouse? Truly "it is not enough to maintain the present

first admission rate; provision should be made for it to increase." Any suggestions about how to relieve this growing ethical and practical crisis would be welcome.

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Faking and the intent to deceive

SIR,—The myth that Gregor Mendel, founder of the science of genetics, in some dishonest way tampered with or falsified his results surfaces from time to time in less scholarly publications, but it is rather a shock to find it being given credence in your columns. Dr T J Hamblin (6 September, p 683) includes Mendel's name in a list of biologists who have faked their results "in order to establish a pet theory."

There is indeed an element of truth behind the myth. R A Fisher demonstrated long ago that Mendel's published results on garden peas were rather consistently too close to theoretical expectation.¹ Although the excess of closeness is slight in any one series, over the whole body of data the combined odds are formidable—about 14 000 to 1 against getting such a close fit with expectation. However, Fisher himself, along with those who have studied the matter since, was at pains to dissociate himself from any suggestion that Mendel himself had deliberately "cooked" or fudged the results. This would be contrary to all we know of Mendel's straightforward, honest, and modest character. The most telling point, perhaps, is that when a botanical bigwig of the time persuaded him to make crosses between species of hawkweed (*Hieracium*) Mendel obtained results wildly at odds with his theory. A person only slightly more devious would have found some excuse (blaming faulty cross-fertilisation technique?) to refrain from publication. Not Mendel: he published, while frankly admitting that the results were contrary to his theory. (We know now that apomictic reproduction is common in *Hieracium*, which explains the apparently anomalous results.)

There are several ways in which the "too good to be true" feature of Mendel's results could have arisen without the least dishonest intent on his part.^{2 3} One of the more likely ones depends on the fact that in the bulk of his work Mendel used characters diagnosable in the seeds. Taken at their face value, Mendel's figures indicate a remarkably low average yield of seeds per plant, which suggests that he did not generally score all the seeds on every plant, but stopped counting once he had ample to establish his point. In such a situation one will normally keep at least rough mental running totals; and it then becomes all too easy to decide, in all innocence, that "enough is enough" at a point when agreement with expectation is especially good. On this view, Mendel was guilty only of neglecting a rather subtle point of experimental design by not setting himself a fixed stopping point in advance: a commonplace precaution nowadays but unrecognised in his day.

It is in practice sometimes difficult to decide where honest error or simple carelessness ends and dishonesty begins. Dr Hamblin mentions one "Kammering"—I presume he means the zoologist Paul Kammerer. Certainly, the notorious midwife toad, whose "nuptial pad" proved to be due to the injection of Indian ink, was a fake; but it is by no means certain that Kammerer himself perpetrated it. So too with Kammerer's other controversial experiments. He was a slapdash experimenter and careless in the way he reported his results and one need not accept any of his claims to have demonstrated Lamarckian inheritance, still less swallow whole the partisan account of his work by Koestler,⁴ in order to recognise that he may have been essentially honest throughout.

If the essence of forgery is intent to deceive, Mendel was assuredly not guilty. As for

Kammerer, I think that "Not proven" is the fairest verdict.

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¹ Fisher RA. *Ann Sci* 1936;1:115-37.

² Sturtevant AH. *A history of genetics*. New York: Harper and Row, 1966:13-6.

³ Wright S. In: Stern C, Sherwood S, eds. *The origin of genetics*. San Francisco: Freeman, 1966:173-5.

⁴ Koestler A. *The case of the midwife toad*. London: Hutchinson, 1971.

Postneonatal mortality and child abuse

SIR,—Jacqueline Roberts and others (12 July, p 102) demonstrated a threefold-raised post-neonatal death rate among siblings of abused infants. This finding fits in with our own observations in Cardiff infants.

We have been studying the feasibility of the use of scoring systems to assess groups of infants at high risk of non-accidental injury and sudden infant death syndrome, based on the data of the Cardiff births survey. Eight factors were found predictive of both these outcomes: low maternal age, low social class, residence in the poorer wards of the city, maternal smoking, lack of or late onset of antenatal care, lack of employment of the mother during pregnancy, admission to the special care baby unit, and artificial feeding. Three further factors were related to abuse: mother unmarried, low maternal weight, and low birth weight (which in our own data was, surprisingly, only weakly related to cot death). In addition, cot death was more common in male infants and twins and in deliveries occurring from August to January; when pregnancy had been complicated by antepartum haemorrhage, rupture of membranes 24 hours before onset of labour, venereal disease, or thromboembolism; after spontaneous deliveries and those in which the second stage of labour was short; when an episiotomy had not been judged necessary; when the mother had had two or more previous deliveries; and when the partner was unemployed. Many of the factors predictive of both abuse and cot death are likely to remain unchanged for the women concerned from one delivery to the next; most of the additional factors favouring cot death are specific to the index pregnancy. In view of this, the Oxford findings are not surprising.

We have quantified the predictive power of a scoring system in terms of the estimated probability that when an abnormal and a normal baby are chosen independently at random the abnormal one will have received the more adverse score (1-3). $P=1.0$ denotes perfect prediction while in the absence of discriminatory power p takes the values 0.5. The abuse predictor can predict abuse with a power of $p=0.858$ and cot death with a power of $p=0.768$. The cot death score predicts cot death and abuse with $p=0.818$ and 0.773 respectively. Thus the two predictors do not select quite the same infants as being at high risk, but they are in quite close agreement. On epidemiological and social grounds, much the same group of infants is at high risk of both non-accidental injury and cot death.

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Recurrent abdominal pain in a patient on haemodialysis

SIR,—I was interested to read the report by Dr Martin Feinroth and others (27 September, p 838). A patient attending this hospital suffered from dialysis-induced abdominal pain which I believe was a further example of the syndrome they describe.

A 54-year-old woman had prior to her recent death been attending this hospital thrice weekly for the past 10 years for maintenance haemodialysis, chronic renal failure having been diagnosed in 1968. Her condition was complicated by hypertension, gross cardiomegaly, angina pectoris, and episodes of congestive heart failure. Her medication included digoxin; latterly the dosage required was 0.0625 mg twice daily (a typical random serum digoxin being reported as 1.5 nmol/l (1.17 ng/ml)). An attempted renal transplant in 1975 had been a failure. She unfortunately died in June. For the past few years she had complained of severe central abdominal pain about 10 or 15 minutes after dialysis was commenced. She always maintained that this pain was quite distinct from the angina she suffered on exertion and numerous electrocardiograms recorded during these episodes showed no evidence of new myocardial damage. Once dialysis was finished she became pain free until the next dialysis. Abdominal x-ray examination was reported on several occasions as showing "no evidence of intestinal obstruction." Her gall bladder was removed in 1977 following several attacks of typical cholecystitis.

We had always suspected that this patient's recurrent abdominal pain was due to intestinal ischaemia precipitated by (a) fluid removal during dialysis and (b) relative hypotension, her blood pressure typically falling from her normal 160/110 mm Hg to 100/60 mm Hg during dialysis. We considered the patient's general condition to be so poor that treatment was confined to analgesics. The most satisfactory method of treatment we found was the self-administration of 50% nitrous oxide and 50% oxygen from an Entonox apparatus.

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"Benign" monoclonal IgE gammopathy

SIR,—H Ludwig and W Vormittag (23 August, p 539) presented the case history of a rather remarkable patient who has apparently been identified as suffering from a benign monoclonal IgE gammopathy. It would appear from the data presented in this paper that it is very likely that this patient's IgE gammopathy is a benign one. My particular interest in the paper arises from some general remarks and claims made in the introduction to the paper.

The authors state that "benign" monoclonal proteins can be found in about 1% of the population at 25 years of age and that with increasing age the incidence of paraprotein rises to more than 2.5% in individuals over 70. This statement is not attributed to anyone, but it would appear likely that they are quoting the results published by Axelsson *et al* in 1966.¹ I would like to point out that there are other studies which do not show such a high incidence. Hobbs in 1967² found an incidence of such paraproteins in 0.8% of 7200 hospitalised patients and my own group,³ in a population study in New Zealand, found an overall incidence of 0.5% paraproteinaemia in a population study of adults over the age of 21 years. We also found that the incidence of paraproteinaemia seemed to increase with age and by the eighth decade the percentage had risen to 1.5%.

The other contentious point in the introduction

is the statement that "benign paraproteinaemia of the IgC, IgA, IgM class or of light chain type is relatively common and only rarely followed by multiple myeloma or Waldenström's macroglobulinaemia." This comment is not attributed and is presumably the opinion of the authors, and I would dispute it. In the population study previously alluded to, my co-workers and I found that we had detected 11 cases of paraproteinaemia in a population of 2670 people. Nine of these 11 people were studied clinically, the other two having died between the population study and the clinical study. One of these was aged 74 years and died of a gastric ulcer and the second was 89 years and died of myocardial infarction. Necropsy had not been carried out on these patients. A third patient died while under clinical study as a result of an accident and her necropsy, although inadequately done for our purpose, did not demonstrate any haematological disorder. Two of the remaining patients were shown to have myeloma and two Waldenström's macroglobulinaemia, and another was shown to have lymphosarcoma and acute myelomonocytic leukaemia. The remaining three patients from this population study remain well 13 years after the initial discovery of their paraprotein. Their gammopathies remain, and in each case there is some suspicion of a lymphoproliferative malignancy. Thus, in five out of the nine evaluable patients in this study a haematological malignancy has developed. This greater than 50% incidence could not be regarded as an only rarely occurring phenomenon.

I appreciate that Drs Ludwig and Vormittag have not defined their use of the term "benign" monoclonal paraproteins, which makes it rather difficult to refute entirely their proposition that benign paraproteinaemia is seldom important. Most workers involved in the evaluation of such subjects do recognise that the stable non-progressing peak which is unassociated with immunosuppression of the other immunoglobulins or other biochemical abnormalities may be labelled benign. On the other hand, I would like to submit that such patients should have serious consideration given to exclusion of haematological malignancies. In my own experience, a considerable proportion of such cases do have an associated immunocytoma. After the initial studies the matter may need to be reconsidered at a later date.

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¹ Axelsson U, Bachmann R, Hallen J. *Acta Med Scand* 1966;179:235-47.

² Hobbs JR. *Br Med J* 1967;iii:699-704.

³ Carrell RW, Colls BM, Murray JT. *Aust NZ J Med* 1971;iv:398-401.

Protein synthesis and breakdown after vaccination

SIR,—Dr Kirstine Adam and Professor I Oswald (20 September, p 809) have criticised our methods for measuring protein turnover in the whole body (26 July, p 263) because we suggest that protein synthesis exceeds protein breakdown during feeding. They say that direct measurements of incorporation of isotope into protein of a number of individual tissues show the opposite—that protein synthesis is highest while food intake is least. They also point out that in order to calculate the rate of protein synthesis it is necessary to know not only the rate of incorporation of isotope into protein but also the specific radioactivity of the free amino-acid at the site of protein synthesis. It is extremely unfortunate, therefore, that not one of the studies quoted by Dr Adam and Professor Oswald involved measurements of the specific