ning of the history of man and because of their appearance, particularly their faces, some of them have had certain characters or personalities attributed to them. These resemblances may be quite false, as in the case of the bear, who is, in fact, not an amiable and comforting creature but quick-tempered and treacherous; but the image is irresistible and so it is with lots of animals.

A child can relinquish his own identity and go along with the animal, imagining what it is like to be doing what he is doing, eating what he is eating, belonging to that world. So when a rabbit, for example, is placed on the bed of a child in hospital and he watches its shallow breathing, its alae nasae, and its little sensitive movements he is fascinated, he loves it, he loses himself for a while in identifying with the rabbit, going on a sort of journey with him and so leaving his troubles behind.

Not all children in hospital have troubles that they want to escape from, but many are, or have been, under stress in various ways. Think of a child of 6 or 7 years recovering from abdominal surgery with intravenous drip and nasogastric suction drainage perhaps; watch his expression as a guinea-pig, rabbit, or dog (and they can be quite compatible together in the ward, by the way) comes to be his companion for a time. He is soothed, he relaxes, smiles, and feels better.

All who have pets as a permanent feature of the life of their children's wards will be grateful to Mr Cooper for the useful knowledge and guidance provided in his article and for making it respectable to have animals in the ward and so defending us from the attacks of hygienists.

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Shoplifting

SIR,—Your leading article (20 March, p 675) emphasises the problem of unintentional shoplifting (as suggested in my previous letter (28 February, p 523)) and indicates that the incidence probably varies, being greater in some areas than Dr S Bockner (20 March, p 710) finds. Perhaps this reflects the varying factors, including the number of individuals referred, the wisdom and selectivity of the officers of the court and the lawyers concerned, the type of catchment area (High Street rather than Oxford Street shoppers), and the availability of social and family psychiatric histories. Important also are individual clinical interests, attitudes, and judgments, taking into account the patient's presentation (preferably by inpatient observation), with appropriate social, psychological, probation, and nursing reports also.

Perhaps the numbers concerned imply occasionally but none the less significant rather than esoteric diagnoses, but no one would dispute Dr Bockner's sensible considered view that "greed does not justify dishonesty" and it would be naive and unpractical to be unaware of this majority and non-psychiatric problem. Indeed, before necessary "pruning" because of limitation of space, my previous letter opened by saying, "Regarding the ever-increasing 'business' of shoplifting, little need be said about offences committed for greed."

Concerning organic contributions to shoplifting, an experienced consultant surgeon has told me of cases in which bizarre behaviour and unwitting shoplifting have occurred in cases of severe hypoglycaemia due to insulinomas. He has asked if other readers could let him know of any similar cases in their experience, either through your correspondence columns or myself.

Your article wisely stresses both the preventive aspects and how the law has struggled with this problem for centuries. Significantly, Sir Roger Ormrod, FRCP, Lord Justice of Appeal,[1] has written that "courts have always to deal with individuals; and their need—sometimes it is almost a desperate need—is for information, knowledge and advice on an individual basis."

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[1] Ormrod, R F G, *British Journal of Psychiatry*, 1975, **127**, 193.

Osteomalacia and calcium deficiency

SIR,—In a reply to "Any Questions?" (24 January, p 203) it is stated that "an authoritative review of calcium requirements concluded 'there is no firm evidence that calcium deficiency exists in humans.'"

In the "authoritative review"[1] there is no reference to the work of Dr Marshall Day and myself of over 35 years ago.[2-4] In Palampur in the Kangra District of the Punjab we recorded that 25% of the children had rickets and 50% of the women had osteomalacia. Surprisingly, they had almost perfect teeth, with one of the lowest rates of dental caries and dental hypoplasia. The Punjab Public Health Department carried out an exhaustive nutritional inquiry and concluded that the alarmingly high incidence of rickets and osteomalacia was due to calcium and phosphorus deficiency. The diets contained very little calcium as little or no milk was available because, being a Hindu area, most cows were barren in a hill district with little and poor pastures. The children wore few or no clothes and there was no purdah, so that the skin of everyone had more than sufficient tropical sunlight for vitamin D synthesis.

These old observations are perhaps again important in considering the cause of the high frequency of osteomalacia in elderly people in Britain, many of whom have low intakes of vitamin D and also low intakes of calcium.

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[1] Walker, P P, *American Journal of Clinical Nutrition*, 1972, **25**, 518.
[2] Taylor, G F, and Day, C D M, *British Medical Journal*, 1939, **1**, 919.
[3] Taylor, G G, and Day, C D M, *British Medical Journal*, 1940, **2**, 221.
[4] Day, C D M, *British Dental Journal*, 1944, **76**, 155 and 143.

Geriatrics in the cottage hospital

SIR,—Professor J A Davis (20 March, p 713) asks what an enthusiastic general practitioner has to offer his long-stay geriatric patients. Luckily we have clinical assistantships covering 101 long-stay geriatric patients in two hospitals as well as a 50-bed "part three accommodation" home for the elderly within our practice.

However, our cottage hospital population usually has an average age over 65 years, and here we can offer an efficient and often homely atmosphere for our aged sick. What's more, we can usually offer immediate admission to our acute beds by maintaining a high turnover. This is often difficult to arrange with a district general hospital or "acute" ward of a geriatric hospital, especially at the weekend for a mildly disturbed, sick old person. The terminally ill receive the most caring attention, often from auxiliary nurses who know the patient personally, and bereaved relatives are often dealing with their own GP, to whom they can turn for help themselves.

This does not alter my premise (Personal View, 28 February, p 519) that a 22-bed acute hospital offering casualty, diagnostic, and cold surgery beds should not be allowed to become filled with *long-stay* old people.

Children we do not admit, for an ill child must be under the care of a specialist, as I think Professor Davis will agree.

ANDREW CROWTHER

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Domiciliary oxygen in chronic bronchitis

SIR,—Your leading article on this subject (28 February, p 484) rather gave the impression that the capital cost of the oxygen concentrator was beyond the reach of most of us. In fact, this unit costs less than the figure which you quote for supplying cylinder oxygen for one year and, what is more, since the running cost when obtaining the same volume of oxygen from the concentrator is only about £100 per annum it is possible to save about £300 in the first year and more than £1000 per annum thereafter.

The work by Stark and Bishop which led to the report in which oxygen concentrators were stated to be noisy[1] was done on the first concentrators ever to be produced. As a result of this report the manufacturers redesigned the unit. Considerable development has taken place in the last few years. The concentrator has been silenced and can now be used in the bedroom without disturbing sleep, while efficient suppressors have been fitted to avoid any interference with television or electronic equipment. Concentrators are now by far the cheapest and best method of obtaining low-pressure domiciliary oxygen.

There is another feature of the concentrator worthy of serious attention: it can be fitted with a time switch to turn it off after a period of use, allowing the patient to stay asleep.

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[1] Stark, R D, and Bishop, J M, *British Medical Journal*, 1973, **2**, 105.

Computer-held medical records

SIR,—It is indeed true that changing the structure of records changes the behaviour of physicians (Dr C J Bulpitt and others, 20 March, p 677). Preliminary work in general practice shows that the introduction of a system of problem-orientated medical records markedly affects the way general practitioners manage patients.

We hope to show that merely introducing a hypertensive flow-sheet improves the initial

work-up before initiating treatment, defines the treatment goals, and improves control. The use of a problem-orientated summary card in addition seems to achieve most of the objectives attempted in the computer-held record system. For research perhaps manual handling of notes is still necessary, but recall of information is much simplified, as compared with unstructured notes.

The problem-orientated record system has the advantage that it is very inexpensive and can be used by both general practices and hospitals which have no access to computers.

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SIR,—In the discussion of their work on computer-held records of hypertensive patients Dr C J Bulpitt and his colleagues (20 March, p 677) compare their computer system with the clerical retrieval from standard notes. However, many of the advantages they have found using the computer could be achieved using a simpler approach—for example, punched feature cards.[1] We have used punched feature cards in a general practice of 10 000 patients for three years[2] and, should it prove necessary, they can be used as an input to a computer.[3]

The false logic of "a computer can solve our problems, therefore we should use a computer to solve our problems" appears to be all too common. Perhaps more attention should be paid to simple, less expensive, and more flexible information handling systems, of which punched feature cards are an outstanding example.

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[1] Harden, R McG, *et al*, *British Journal of Hospital Medicine*, 1975, **13**, 195.
[2] Harden, K A, Harden, R McG, and Reekie, D, *British Medical Journal*, 1974, **2**, 162.
[3] Harden, K A, and Harden, R McG, *Journal of the Royal College of General Practitioners*, 1975, **25**, 143.

Recurrent haematuria

SIR,—The paper by Dr J Michael and his colleagues from St Thomas's Hospital on unexplained recurrent haematuria (20 March, p 686) comes as a welcome addition to the literature on this important subject. However, one or two points deserve comment.

I have never questioned the *value* of renal biopsy in the syndrome of recurrent or persistent haematuria, only its justification and practicability as a routine in such patients. Unexplained haematuria remains a large group in most disease indices, being second in magnitude (25 cases) in Burkholder's group of haematuria cases[1] and making up 5-10% of all patients with haematuria in the Manchester review.[2] Leaving aside economic and temporal factors it seems unlikely, should this incidence be reflected nation-wide, that facilities exist for the efficient performance of renal biopsy and the accurate interpretation of the histological and immunofluorescent findings in every patient falling into this group. The pursuit of a diagnosis, especially if it prevents needless over-investigation, is to be applauded; it was Burkholder's contention—rightly so, I feel—that *invasive* investigations with a recognised morbidity which do not lead to any significant change in patient treatment might be open to criticism.

Furthermore, the average age in the St Thomas's group was 25 years—an age group in which renal biopsy might be expected to yield a high incidence of abnormalities reflecting the high incidence of so-called focal nephritis in young adults with haematuria. The average age in the Manchester review was 50 years, in which group the search for incipient malignant disease is a more significant problem and in which renal biopsy might not play such a useful role.

This said, however, the results from St Thomas's certainly complement those from other centres investigating the younger groups of recurrent haematuria and, with those of Higgins and Aber,[3] seem to indicate the diagnosis, in the young male patient with this complaint, of mesangial IgA nephropathy, with its apparent independence from the haematuria/loin pain syndrome, of possible renovascular origin, occurring more commonly in the female. This may well represent a significant step forward in the clarification of this important disease complex and its management.

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[1] Burkholder, G V, *et al*, *Journal of the American Medical Association*, 1969, **210**, 1729.
[2] O'Reilly, P H, *Postgraduate Medical Journal*, 1974, **50**, 746.
[3] Higgins, P M, and Aber, G M, *British Journal of Urology*, 1974, **46**, 601.

Propranolol and the nephrotic syndrome

SIR,—In a recent report (12 April 1975, p 68) my colleagues and I described a patient who had developed the nephrotic syndrome in association with practolol therapy. In the same patient propranolol was substituted for practolol in a dosage of 40 mg daily for 10 months and 160 mg daily for another six months. Vague circumscribed psoriasiform skin lesions on the arms and a proteinuria of 2 g/24 h were then detected. No urinary protein had been detected on routine examination during the previous months. The propranolol was stopped, but four weeks later the patient developed a severe normotensive nephrotic syndrome with a proteinuria of 21 g/24 h, urinary casts, no excess of urinary red or white cells, a creatinine clearance of 70 ml/min, and a serum albumin concentration of 26 g/l. Tests for antinuclear factor in the serum were negative.

Treatment with prednisolone, initially 60 mg daily, was instituted six weeks after stopping the propranolol therapy because of persistent severe proteinuria. After three weeks the urine contained only a trace of protein and the serum albumin level was 35 g/l. Two weeks later the urinary protein was quantitatively normal and the creatinine clearance was 82 ml/min. The rash had also resolved.

It was assumed that the propranolol had induced a minimal-change glomerulonephritis, as the practolol had done. This diagnosis was not confirmed by a second renal biopsy, although it was supported by the excellent and swift improvement with prednisolone. Although practolol and propranolol are different chemically, they both act as beta-blockers and, in this man's case, they both appear to be capable of inducing a nephrotic syndrome.

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Bottle-feeding and tummy-ache in infants

SIR,—Newborn babies commonly have tummy-ache. The infant cries incessantly, will not be comforted, flexes his legs, and continually seeks the breast, although his sucking is frequently interrupted by spasms of distress. The syndrome is conventionally ascribed (without evidence) to "wind," and since it is maximal at around one week of age—shortly after discharge from hospital—tends not to come to the notice of the doctor. The mother and her advisers frequently interpret the signs as resulting from under-feeding and hunger, and this is a major factor in the customary decision to abandon breast-feeding.[1]

Virtually all hospital-born babies receive feeds of cow's milk for the first 1-3 nights of life, even if they are subsequently to be breast-fed. Cow's milk is antigenic and evokes an antibody response in normal bottle-fed neonates.[2] A local immune response in the gut would be associated with hyperplasia of the mesenteric lymph nodes and Peyer's patches, with inflammation and hyperperistalsis leading perhaps, in exceptionally severe cases, to intussusception. The timing of the syndrome is just right for primary immune response.

Total avoidance of cow's milk in neonatal life has been advocated[3] as a means of preventing atopy. I suggest that it would also avoid "wind" and greatly increase the likelihood of successful breast-feeding.

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[1] Davies, D P, and Thomas, C, *Lancet*, 1976, **1**, 420.
[2] Truelove, S C, and Jewell, D P, in *Clinical Aspects of Immunology*, ed P G H Gell, R R A Coombs, and P J Lachmann. Oxford, Blackwell, 1975.
[3] Soothill, J F, *et al*, British Society for Allergy and Clinical Immunology, Oxford meeting, 13 December 1975.

Amoebic meningoencephalitis in Britain

SIR,—Dr J L Griffin (17 January, p 153) has suggested that in our case of amoebic meningoencephalitis[1] an acanthamoeba (which was not isolated) must have been the primary pathogen, the *Naegleria gruberi* which was isolated from the cerebrospinal fluid (CSF)[2] being a secondary invader.

We agree that in the CSF the amoebae which were seen resembled acanthamoebae more than naegleriae, although naegleriae can move slowly and produce spiky pseudopods.[3] Dr Griffin suggests that culture for acanthamoebae from the CSF should have been done immediately. In fact, although not mentioned in our papers, this was done in this laboratory but no amoebae were isolated. Immediately after collection the first specimen of CSF from case 1 in which amoebae were seen (two days after admission) was inoculated on to klebsiella-agar plates and into HeLa cell cultures, both of which had previously been found suitable in this laboratory for the growth of acanthamoebae. The surface of the agar was of a dryness known to be suitable for the growth of acanthamoebae, although this degree of dryness was found subsequently to be unsuitable for the growth of naegleriae. The day after collection this CSF