Dying for palliative care

General practitioners have a crucial role

EDITOR,—The anonymous diary of a surgeon's death arouses compassion but also suggests that the general practitioner, who has a crucial role in palliative care, played a small part.[1] The general practitioner made a referral to hospital in August 1993 but seems to have had no part in the first two admissions, the decision to go to South Africa, or the subsequent hospital based treatment in March 1994 after the diagnosis of leukaemia. Serious difficulties in communication and considerable distress are described, but all we are told is that, three days after discharge from hospital, on 12 July, "Our GP called: asked for the third time how Jeffrey was coping psychologically. At no time did he offer any help to make Jeffrey comfortable."

There is no reference to a general practitioner during the difficult days after the final discharge from hospital on 22 July until 17 August, two days before death, when "Dr H, who was standing in for K," visited. Then, "shortly after Jeffrey's death our GP called to say he was sorry at the way things had gone." How did all this come about?

Doctors may not get the best care and sometimes contribute to this; consultant surgeons may be especially at risk. General practitioners may not wish to intrude, particularly when a patient has a condition, such as leukaemia, that requires modern specialised knowledge. If this was not a normal family relationship there may (but in an ideal world should not) have been awkwardness on both sides. The hospital specialists may know little about palliative care at home. Jeffrey "says if it were not for me he would be in a hospice" but "he was allowed to die in terrible discomfort with only me to look after him. No help was offered."

A breakdown in communication explains a lot. Unfortunately, this situation is still reported in terminal care and in the long term care of elderly and disabled people. There remains a big gap in medical education. The General Medical Council has recognised this, and medical schools are changing their curriculum. Much remains to be covered in continuing medical education. Help is available from CONtinuing Care At Home (CONCAH, 54 Glasshouse Lane, Countess Wear, Exeter EX2 7BU), a national multiprofessional association founded in 1992 to promote a high standard of service to people with disability living at home and their domestic carers. It will help to arrange workshops for doctors, nurses, therapists, carers, and others who may wish to learn how to work together more effectively.

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1 Dying for palliative care. *BMJ* 1994;**309**:1696-9. (24-31 December.)

Anger is a means of coping with illness

EDITOR,—The decision to afford someone three full pages—or, indeed, any space at all—to record their anger over their partner's terminal illness requires explanation.[1] All of us who do clinical work know that anger is a means of coping with the distress of illness, either one's own illness or that of a loved one. The fact that Jeffrey's partner should keep a minute record of what were perceived to be the gaucheries of the doctors and nurses is understandable; I hope it helped. The messages conveyed are, however, confusing (and this also I accept). At the start of the course of difficult treatment the author expressed anger that the doctors were attempting to keep the patient alive. But that is our job unless the patient refuses the treatment, after careful explanation of the chances of its success and any unpleasant effects. The author also expressed anger over the withholding of a "treatment" (a sedative) that would, it was explained, have depressed respiration to the extent of hastening death; our code of practice forbids euthanasia.

Anger is expressed because Jeffrey was seen by a consultant and not the director of the clinic. The (friendly) gesture of the consultant who came to sit on Jeffrey's bed is dismissed as rudeness; the day patient sister's offer of bereavement counselling is rejected as "too little, too late." Finally, the general practitioner is lambasted for explaining the system of living wills, which I perceive to be a humane arrangement and not "inhumanity."

Jeffrey seems to have received good hospital care and accommodation; he had a single room (with a door). This privileged accommodation, which few NHS patients receive, was probably due to his status as a consultant surgeon. But it provided his partner with another cause for scornful derision: "they marched in without knocking" (presumably, had they knocked on the door this would have been turned to, "they expected a man in his state to call out 'enter' ").

The introduction of teaching on communication skills for medical students is a welcome innovation. One thing that teachers will have to impart concerning the management of anger is the difficult task of controlling angry riposte. It is a hard task, and we all learn through reflection on our mistakes.

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1 Dying for palliative care. *BMJ* 1994;**309**:1696-9. (24-31 December.)

Medical care continues to be important for health

EDITOR,—John P Bunker's estimate of the part medical care has played in improving life expectancy[1] is a valuable correction to McKeown's much quoted negation of 20 years ago. One of McKeown's main arguments concerned tuberculosis, and this features large in the Canadian report quoted by Ken Judge.[2] The introduction to that report reproduces McKeown's graph of mortality from respiratory tuberculosis in England and Wales, showing that the rate standardised for age fell steeply from 4000 per million in 1840 to less than half that figure in 1880, when the tubercle bacillus was first identified, and then more slowly to the time when streptomycin first became available. Even with chemotherapy the fall in such a graph seems only slightly more rapid.

The slight decline in mortality after the identification of the bacillus could have been due to fewer fatal diagnoses, though Ziehl-Neelsen staining of sputum was not readily available everywhere until 30 years later and culture not at all. The chest services provided by local authorities, including beds in sanatoriums, developed fully after the first world war. They made diagnosis more accurate and provided both treatment and preventive services. Yet tuberculous primary infection, usually without overt disease, was so common in English urban populations that around 40% of 13 year olds were found to be tuberculin positive in the BCG studies carried out by the Medical Research Council after the second world war. By 1970 that proportion was less than 10%. This was no less important an indication of successful health care than the reduction in deaths by 95% in the same period.

Almost 60 years ago I worked in a hospital for infectious diseases with 400 beds. The first sulphonamide drugs had just become available, and their effect was dramatic in reducing mortality from secondary infections in measles and whooping cough as well as in puerperal sepsis. Soon sulphapyridine and other drugs of the sulphanilamide group had greatly changed the prospect in conditions as diverse as lobar pneumonia, meningococcal meningitis, and gonorrhoea. Yet in the hospital surveys of 1943-4 we found some 25 000 beds in hospitals for infectious diseases and, as surveyors, thought that there should be more. Health care, including immunisation, has reduced that need by nine tenths.

Bunker gives us a valuable reassessment of what health care has done. The publication of figures on mortality in hospitals will help in the development of local studies of outcome, provided they are not used as crude indicators of efficiency in themselves. Speculation about improvements in health that might be produced by social change is important and was well developed in the Black report, but the need for care during the long period that any such improvement will take remains.

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1 Bunker JP. Medicine's core values. *BMJ* 1994;**309**:1657. (17 December.)
2 Judge K. Beyond health care. *BMJ* 1994;**309**:1454-5. (3 December.)

Diagnosis and management of chronic sinusitis

Do not rely on computed tomography

EDITOR,—Certain aspects of the diagnosis of sinusitis were neither emphasised in Kathryn L Evans's review nor mentioned in the summary points.[1] The most important advance in the assessment of chronic sinusitis is rigid, rod lens, nasal endoscopy. Evans only briefly mentions endoscopy and does not address its pivotal role in the diagnosis of sinusitis and related nasal disease. A rigid endoscope allows direct inspection of key areas of the nasal cavity, in particular the region of the sinus ostia in the middle meatus. At present, endoscopic abnormalities are the most robust evidence of underlying sinus disease, and thus nasal endoscopy offers a more specific diagnosis than any form of imaging. Evans seems to emphasise the role of radiology—both plain radiography and computed tomography—in diagnosis at the expense of endoscopy.

Computed tomography should not be relied on for diagnosing chronic sinusitis, as alone it greatly overdiagnoses the condition. Its predictive accuracy (the percentage of positive results that are true positives) is abysmal because up to 40% of asymptomatic people have some sinus opacification.[2,3] Computed tomography should usually be reserved as a preoperative investigation for patients already diagnosed as having chronic sinusitis by endoscopy. In this role it is unsurpassed in providing a "roadmap" for the surgeon, which includes the extent of disease, anatomical variants, and potential surgical hazards.

We recommend that the following steps should be incorporated into the management of patients with symptoms of chronic sinusitis. Firstly, all patients should receive prolonged medical treatment and should be referred for an otolaryngological opinion if symptoms continue. Secondly, rigid endoscopy is mandatory to assess patients with persistent or severe symptoms. Finally, computed tomography should be performed only if sinus surgery is planned or in cases in which endoscopically occult sphenoethmoid disease is suspected.

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1 Evans KL. Diagnosis and management of sinusitis. *BMJ* 1994; **309**:1415-22. (26 November.)
2 Havas TE, Motbey JA, Gullane PL. Prevalence of incidental abnormalities on computed tomographic scans of the paranasal sinuses. *Arch Otolaryngol Head Neck Surg* 1988;**114**:856-9.
3 Bolger WE, Butzin CA, Parsons DS. Paranasal sinus bony anatomic variations and mucosal abnormalities: CT analysis for endoscopic surgery. *Laryngoscope* 1991;**101**:56-64.

Author's reply

EDITOR,—I believe that the points raised by Alexander C Maclennan and Gerald W McGarry were addressed in my review. In fact, we both reference the same paper for the same reason.[1] When an overview of a broad subject is presented to the medical profession as a whole it is possible to imply an inappropriate emphasis on one investigation. I did not, however, imply that computed tomography should be relied on for diagnosing chronic sinusitis; indeed, I stated that it should be requested only "after failure of maximal medical treatment, if a complication arises, or if malignancy is suspected."

I agree that rigid nasal endoscopy is important in diagnosing chronic sinusitis, but it cannot stand alone as a diagnostic investigation as it also has limitations as already stated—for example, the inability to identify concha bullosa or Haller's cells. Not all patients are able to tolerate the middle meatus being inspected with a rigid endoscope, and if they are the maxillary sinus ostia are rarely visualised in the depths of the ethmoid infundibulum, and any view of the frontal sinus ostium will depend on the superior insertion of the uncinate process.

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1 Havas TE, Motbey JA, Gullane PJ. Prevalence of incidental abnormalities on computed tomographic scans of the paranasal sinuses. *Arch Otolaryngol Head Neck Surg* 1988;**114**:856-9.

Vitamin A supplementation in preschool children with acute diarrhoea

EDITOR,—Nita Bhandari and colleagues report the results of a trial of vitamin A in the prevention of respiratory and diarrhoeal morbidity in a population of children likely to be clinically or subclinically deficient in vitamin A.[1] We do not believe that the conclusions are justified by the results described, and the public health implications contained in the box are misleading.

The first two points in the box are not contentious and are based on previous research. The third point, referring to the authors' study, states that in children aged 1-5 vitamin A supplementation reduces the prevalence of diarrhoea associated with fever by 36%. This finding applies only to those aged 23 months and over, thus excluding some children aged 1 year, and was based on subgroup analysis of the data. The P value for this observation was only 0·05, and given that 20 subgroup analyses were carried out, we do not believe that this can be considered to be significant. The fourth point in the box is that children given supplements suffered less measles. This finding was not significant for the whole study population, and the significant reduction in the younger age group was countered by an opposite (and not similar as stated in the text) trend in the older age group. The main findings of this study are not emphasised in the box—that is, there was no difference in the incidence or prevalence of diarrhoea or respiratory infections between the treatment and control groups.

In the final paragraph the authors recommend that vitamin A supplementation should be provided to children aged 6 months to 5 years in settings in which vitamin A deficiency is a public health problem. Given that this conclusion could not be drawn from the results of this study and depends on the results of previous trials, an important ethical question arises. The study was carried out on a subgroup of children, many of whom were likely to have subclinical vitamin A deficiency; given the conclusion derived from literature that predated this study, it might be considered unethical to withhold vitamin A in these circumstances. Because vitamin A may be harmful it might be argued that a trial is justified, but a different trial design would have to be used to have sufficient power to detect adverse effects.

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1 Bhandari N, Bhan MK, Sazawal S. Impact of massive dose of vitamin A given to preschool children with acute diarrhoea on subsequent respiratory and diarrhoeal morbidity. *BMJ* 1994;**309**:1404-7. (26 November.)

Authors' reply

EDITOR,—Several of the issues raised by Paul Pharoah and colleagues are pertinent. The reduction in the prevalence of diarrhoea with fever and the incidence of measles was observed only in subgroup analysis and must be considered to be only suggestive. As there was a significant interaction of age on the effect of vitamin A and diarrhoeal morbidity, in this instance analysis within age subgroups was justified and, in a way, more meaningful than the overall analysis.

Several recent publications have reported the impact of vitamin A supplementation on morbidity. The findings ranged from no reduction in children given supplementation in Haiti[1] through a decrease in visits by a doctor because of diarrhoea in Ghana[2] to an overall reduction in the incidence of diarrhoea by 20% in Brazil.[3] In the last study in Brazil the benefit of supplementation was greater for severe episodes of diarrhoea. When diarrhoea was defined as the passage of three or more liquid stools a day the overall reduction in the prevalence of diarrhoea was 8%; the reduction increased to 20% when a more stringent definition of six or more liquid stools a day was used. The variable results reported may be related to the differences in severity of vitamin A deficiency across studies and the different methods of analysis.

When these studies are viewed together vitamin A supplementation seems to reduce the incidence of measles and to affect the severity of diarrhoea favourably but to have no impact on pneumonia. This is consistent with findings on cause specific mortality after vitamin A supplementation in the few studies providing such data.[4]

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1 Stansfield SK, Louis MP, Lerebours G, Augustin A. Vitamin A supplementation and increased prevalence of childhood diarrhoea and acute respiratory infections. *Lancet* 1993;**342**: 578-82.
2 Ghana VAST Study Team. Vitamin A supplementation in northern Ghana: effect on clinic attendances, hospital admissions, and child mortality. *Lancet* 1993;**342**:7-12.
3 Barreto ML, Santos LMP, Assis AMO, Araújo MPN, Farenzena GG, Santos PAB, *et al.* Effect of vitamin A supplementation on diarrhoea and acute lower-respiratory-tract infections in young children in Brazil. *Lancet* 1994;**344**:228-31.
4 Beaton GH, Martorell R, L'Abbé KA, Edmonston B, McCabe G, Ross AC, *et al. Effectiveness of vitamin A supplementation in the control of young child morbidity and mortality in developing countries. 1992. Report submitted to CIDA Faculty of Medicine, University of Toronto.* Toronto: University of Toronto, 1992.

Carrier testing in children for cystic fibrosis

EDITOR,—Your recent article debating screening for carriers of cystic fibrosis raises important issues.[1] One aspect, however, that has received relatively little attention is whether it is justified to test for carrier status in siblings of patients with cystic fibrosis during childhood. A recent report from the Clinical Genetics Society (UK) was not in favour of testing children for recessive disorders where this is of purely reproductive significance to that child in the future.[2]

However, we found that 90% (103/114) of parents of patients with cystic fibrosis wanted to know the carrier status of their other children; furthermore, 91% (104/114) believed that they had a fundamental right to know.[3] In contrast, we found that most clinical geneticists would try to dissuade the parents or postpone testing until the child was older; 41% (11/27) thought that the parents had no rights to all to know their children's carrier status. Out of 78 doctors working in child health for their views, we found that 76% (59/78) would want to know and 69% (54/78) thought that it was their right to know.

Clearly there is a divergence of opinion, and a consensus probably cannot be reached. As with many difficult ethical issues in clinical paediatrics, the problem of who should make these sorts of decisions remains. Should it be the experts (clinical geneticists), the clinicians who regularly see the families (paediatricians and general practitioners), or the families themselves once properly informed? While it may be prudent to wait until the siblings are old enough to express an opinion, problems may arise when parents of younger children are insistent as carrier testing is becoming more readily available. Commercial pressures will eventually make such testing available both in private laboratories and from local chemists, and the worry is that results will not be accompanied by adequate genetic counselling.

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1 Raeburn JA, Marteau T. Screening for carriers of cystic fibrosis. *BMJ* 1994;**309**:1428-30. (26 November.)
2 Clinical Genetics Society (UK). The genetic testing of children. Report of a working party of the Clinical Genetics Society (UK). *J Med Genet* 1994;**31**:785-97.
3 Balfour-Lynn IM, Madge S, Dinwiddie R. Testing carrier status in siblings of patients with cystic fibrosis. *Arch Dis Child* (in press).

Management of childhood nephrotic syndrome

EDITOR,—P D Mason and C D Pusey's review article on diagnosing and treating glomerulonephritis does not refer, in the paragraph on minimal change nephropathy in children, to the recently published consensus statement of the British Association for Paediatric Nephrology on the management of steroid responsive nephrotic syndrome.[1,2] In particular, Mason and Pusey's recommendations on treatment differ from those of paediatric nephrologists.

It is inaccurate to state that the use of alkylating