Managing sudden bereavement

EDITOR,—In their article on managing sudden bereavement in accident and emergency departments M W Cooke and colleagues seem to have overlooked the fact that staff themselves need support in these incidents.¹ While it is essential, as the authors emphasise, that accident and emergency staff receive adequate training in bereavement care to provide the necessary service for relatives in these circumstances, the staff also have their own needs. Casualty staff are subjected to many conflicting demands and pressures during their working day, not least of which is the management of sudden bereavement and its after-effects.

The BMA's board of science and education will shortly be releasing an educational package on dealing with the unexpected death of a patient. The video, entitled *A Stressful Shift*, consists of a 20 minute dramatised account of the work of a casualty house officer during a difficult 24 hour shift, which includes the death of a child. This powerful fictional account is followed by brief discussion among three doctors about ways of managing sudden bereavement, including breaking bad news to relatives and minimising avoidable stress for staff. The board of science and education hopes that this package, together with a report that it will be publishing soon, *Stress and the Medical Profession*, will assist members of the profession in caring for bereaved relatives and will prompt recognition of the needs of doctors and other staff in maintaining the balance of the humane professional—a term that should not be regarded as an oxymoron.

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1 Cooke MW, Cooke HM, Glucksman EE. Management of sudden bereavement in the accident and emergency department. *BMJ* 1992;304:1207-9. (9 May.)

EDITOR,—In their article M W Cooke and colleagues conclude that immediate care of relatives is good.¹ Informed by my own experience when my son died in an accident and emergency department, I gave thought to what would have been helpful both to me and the hospital staff at that time. Although these observations refer to the sudden death of a child, they could equally apply to the death of any relative.

I think the single most helpful thing staff in accident and emergency departments can do is to give back to the relatives the responsibility that the hospital assumed when the dead child was alive. The following simple format could be followed. On arrival at the hospital as much decision making as possible should be given to the parents. Up to this moment all decisions concerning the child have been made by the parents. This will therefore come naturally to them. The first decision should be when they see the child and the first question asked of them, before any other official procedures begin, should be, "Would you like to see her immediately?" followed by a second question, "Would you like to wash her?"

These questions are saying to the parents "She is still your child, you can still care for her, you can touch her." If the answer to these questions is yes, the child's condition can then be explained. The parents should be told that a nurse will help if

dressings, injuries, or drains need attention. The parents should also be allowed to change their minds. If the answer is no the parents should be asked if they would like to wait until the child is washed and reassured that if they change their minds while waiting they only have to say so and they can then see their child.

I would suggest that they should not be asked a question containing alternatives such as "Would you like to see her right away or would you like to wait until we have washed and dressed her?" If the question is asked in this form the parents will try to guess what might be the normal or correct answer. The bereaved parents are in a strange place with unknown etiquette, and almost unbelievably at such a time will be foundering about trying to discover the correct responses. Asking a simple question is much more likely to elicit the response they actually mean and need to give.

Professionals can help parents if they remain mindful that when all the parents' tenderest emotions are centred on the child, that at that very moment, their child is effectively taken into care. Up to this moment no decision has been taken about their child without their consent. In the event of sudden death their child is not only lost to them in life, she is lost to them in responsibility as the officials take over. The parents are doubly bereft.

Both the death and loss of responsibility are matters of fact. But I don't believe an immediate and abrasive ripping away promotes healing. I think we can find a much less traumatic and injurious way of facing the facts.

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1 Cooke MW, Cooke HM, Glucksman EE. Management of sudden bereavement in the accident and emergency department. *BMJ* 1992;304:1207-9. (9 May.)

Postviral fatigue syndrome

EDITOR,—A M O Bakheit and colleagues suggest that the buspirone challenge test may be useful in distinguishing between a "primary depressive illness" and the postviral fatigue syndrome.¹ It is difficult to assess the truth of this from the data presented in their paper. To know the usefulness of this test the numbers of controls and patients scored above or below an appropriately chosen cut off point of serum prolactin concentration needs to be known. Unfortunately, the authors present the prolactin concentrations as mean values. If the aim of the test is to exclude major depression then a cut off which produces few false negative results should be chosen. Were the high mean values in the postviral fatigue group due to one or two extreme outliers? It is noteworthy that the standard deviations in the patients were much higher in the controls at baseline assessment. Why was this?

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1 Bakheit AMO, Behan PO, Dinan TG, Gray CE, O'Keane V. Possible upregulation of hypothalamic 5-hydroxytryptamine receptors in patients with postviral fatigue syndrome. *BMJ* 1992;304:1010-2. (18 April.)

EDITOR,—A M O Bakheit and colleagues report an enhanced prolactin response to buspirone in patients with postviral fatigue syndrome and suggest this may be due to upregulation of hypothalamic 5-hydroxytryptamine receptors.¹ They fail to mention the considerable evidence indicating that the drug is a moderately potent dopamine antagonist, a pharmacological action which suggests an alternative explanation for their data.^{2,4}

They administered a single 60 mg dose of buspirone—in excess of the daily maximum of 45 mg recommended by the *British National Formulary*—so antidopaminergic effects may well have been significant in their studies. The fact that the prolactin releasing effect of buspirone can be blocked by the drug metergoline does not prove 5-hydroxytryptamine receptor specificity. Indeed, metergoline is used commonly as an alternative dopamine agonist to bromocriptine in managing hyperprolactinaemia. Thus enhanced prolactin release after buspirone in postviral fatigue syndrome may reflect, at least in part, inhibition of increased hypothalamic dopaminergic tone on the anterior pituitary. It would be interesting to study the same groups of patients using a specific D2 dopamine antagonist (such as domperidone) to see whether this is the case.

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- 1 Bakheit AMO, Behan PO, Dinan TG, Gray CE, O'Keane V. Possible upregulation of hypothalamic 5-hydroxytryptamine receptors in patients with postviral fatigue syndrome. *BMJ* 1992;304:1010-2. (18 April.)
- 2 Riblet LA, Taylor DP, Eison MS, Stanton HC. Pharmacology and neurochemistry of buspirone. *J Clin Psychiatry* 1982;43:11-6.
- 3 Meltzer HY, Flemming R, Robertson A. The effect of buspirone on prolactin and growth hormone secretion in man. *Arch Gen Psychiatry* 1983;40:1099-102.
- 4 Meltzer HY, Lee HS, Nash JF. Effect of buspirone on prolactin secretion is not mediated by 5-HT-1a receptor stimulation. *Arch Gen Psychiatry* 1992;49:163.

EDITOR,—A M O Bakheit and colleagues report enhanced release of prolactin after administration of buspirone in patients with the postviral fatigue syndrome compared with controls and patients with depression.¹ Their report would have been improved if they had described more fully the differences between the two groups of patients. As they point out, depression can be difficult to distinguish clinically from the postviral fatigue syndrome.

The authors used the criteria of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* to define depressive illness,² but to qualify for this diagnosis a patient need complain of only four symptoms such as fatigue, hypersomnia, retardation, and loss of concentration in addition to depressed mood. The criteria they used for the postviral fatigue syndrome included depression, fatigue, reversed sleep pattern, and constipation alternating with diarrhoea. It would be surprising if there was not a substantial overlap between the two groups and if one of the main factors affecting diagnosis was not whether the patient presented first to a psychiatrist or a neurologist.

This diagnostic overlap makes it all the more remarkable that a significant difference in response to buspirone was found between the groups, and we would like to know exactly which symptoms or pathological findings account for this difference.

The authors state that all the patients with the postviral fatigue syndrome had characteristic mitochondrial changes, and some had RNA enteroviral sequences, but as the necessary investigations were not applied to the depressed patients or controls it is not possible to tell whether these findings did in fact distinguish the group with the syndrome.

Another issue that concerns us is that the authors fail to refer to the considerable body of work suggesting that release of prolactin mediated by 5-hydroxytryptamine is suppressed in depressed patients^{1,4} and that this response is enhanced by antidepressant treatments, including electroconvulsive therapy.⁵ There is a non-significant trend in their study for the depressed patients to have a smaller rise in prolactin secretion than controls, with the patients with the postviral fatigue syndrome having a significantly higher response than that of both the other groups. The finding that the prolactin response seems to be changed in opposite directions in the postviral fatigue syndrome and depression is of particular interest but makes it all the more frustrating that the clinical features of the two groups are not described more fully. Differences in neuroendocrine responses raise the possibility of subdividing poorly defined disease entities in a way that may better reflect underlying neuroendocrine disease, but to capitalise on this we need to know more about the correlations between these responses and individual symptoms.

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1 Bakheit AMO, Behan PO, Dinan TG, Gray CE, O'Keane V. Possible upregulation of hypothalamic 5-hydroxytryptamine receptors in patients with postviral fatigue syndrome. *BMJ* 1992;**304**:1010-2. (18 April.)
2 American Psychiatric Association. *Diagnostic and statistical manual of mental disorders, third edition, revised*. Washington, DC: APA, 1987.
3 Mitchell P, Smythe G. Hormonal responses to fenfluramine in depressed and control subjects. *J Affective Disord* 1990;**19**: 43-51.
4 Anderson IM, Ware CJ, de Roza Davis JM, Cowan PJ. Decreased 5HT-mediated prolactin release in major depression. *Br J Psychiatry* 1992;**160**:372-8.
5 Shapira B, Lerer B, Kindler S, Lichtenberg P, Gropp C, Cooper T, et al. Enhanced serotonergic responsivity following electroconvulsive therapy in patients with major depression. *Br J Psychiatry* 1992;**160**:223-9.

AUTHOR'S REPLY.—In response to Simon Hatcher, all three groups in our study had baseline prolactin concentrations that did not differ significantly from each other. The high mean value in patients with the postviral fatigue syndrome did not represent odd high cases: this is a finding seen in several studies. The reason for suggested high baseline prolactin concentrations is unknown.

As J S Bevan will know, buspirone has more than one action. Some data suggest that it acts as a dopamine antagonist.¹ Clearly, further experiments, which are under way, are necessary for a complete understanding of its mechanisms.

David Curtis and Timothy Bullock raise many issues. We emphasise that 20-30% of our series of patients with the postviral fatigue syndrome have no depression whatsoever and complain predominantly of fatigue and myalgia. When depression occurs it differs clinically from severe endogenous depression in that the patients do not experience guilt or delusions, are well able to enjoy themselves, and have no loss of interest and, usually, normal libido. Though they may have depressed mood, more usually their severe emotional lability is to the fore and is often the conspicuous symptom.

For research purposes we select patients whose illness occurs after a well defined viral illness and in whom the myalgia is so severe that occasionally they have been referred for cardiologists if the pain resembles that of a myocardial infarct. This highlights muscle involvement. We did not carry out

muscle biopsies on depressed patients because we believe that this would not be ethical. We are well aware of the decreased release of prolactin mediated by 5-hydroxytryptamine in major depression.² The many studies quoted in this paper confirm the decreased response and tend to support our conclusion. Depression and fatigue occur quite commonly in disorders such as idiopathic cyclic oedema, multiple sclerosis, and Parkinson's disease, but these disorders are not primarily depressive illness. The depression in the postviral fatigue syndrome is only one of its symptoms.

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1 McMillen BA, Matthews RT, Sanghera MK, Shepherd PD, German DC. Dopamine receptor antagonism by the novel anti-anxiety drug buspirone. *J Neurosci* 1983;**3**:733-8.
2 Anderson IM, Ware CJ, Davis JMDR, Cowan PJ. Decreased 5HT-mediated prolactin release in major depression. *Br J Psychiatry* 1992;**100**:372-8.

EDITOR.—A M O Bakheit and colleagues recently reported¹ a possible upregulation of hypothalamic 5-hydroxytryptamine receptors in patients with the postviral fatigue syndrome, giving some evidence for hypothalamic functional abnormalities in these patients, which are different from others with depression. There is a growing body of evidence which claims that this clinical condition is organic and cannot be simply perceived as a somatisation disorder in patients with predisposition to psychiatric disease.²

We reviewed and quantitatively analysed with Ceretec and single photon emission tomography the brain perfusion of 14 patients fulfilling the Oxford criteria for diagnosis of myalgic encephalomyelitis.³ They had all had disease for more than six months (more than half the time) manifested with generalised malaise and myalgia, as well as significant physical and intellectual disability. None had any medical condition known to produce fatigue or had recently or in the past had psychiatric disease.

When compared with a group of 24 non-depressed age and sex matched controls (normal volunteers) there was significant reduction of the perfusion to several areas of the brain cortex but particularly the brain stem (table). This original

Brain stem perfusion (mean (SD)) normalised to cerebellum and to visual cortex in 14 patients with myalgic encephalomyelitis and 24 controls

Normalisation area	Patients with myalgic encephalomyelitis	Controls
Cerebellum	0.74 (0.04)*	0.80 (0.04)
Visual cortex	0.75 (0.05)*	0.84 (0.05)

p<0.001, Student's *t* test for independent samples.

finding confirms that single photon emission tomography with Ceretec is an important tool for investigating patients with myalgic encephalomyelitis, including the chronic fatigue syndrome and the postviral fatigue syndrome. This is the first observation of an organic component of this disease, with significant deficits in brain perfusion, particularly in the hypothalamus and pons.

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1 Bakheit AMO, Behan PO, Dinan TG, Gray CE, O'Keane V. Possible upregulation of hypothalamic 5-hydroxytryptamine receptors in patients with postviral fatigue syndrome. *BMJ* 1992;**304**:1010-2. (18 April.)

2 Cluff E. Medical aspects of delayed convalescence. *Rev Infect Dis* 1991;**13**:138-40.
3 Sharpe MC, Archard LC, Banatvala JE, Borysiewicz LK, Clare AW, David A, et al. A report—chronic fatigue syndrome: guidelines for research. *J R Soc Med* 1991;**84**:118-21.

Managing tuberculosis and HIV infection

EDITOR.—The British Thoracic Society's guidelines on managing tuberculosis and HIV infection are incomplete and contain some points that clinicians who look after people infected with HIV with or without tuberculosis would take issue with.¹ Data that I and a colleague obtained in a retrospective study of nine patients with HIV infection (five English with BCG scars, three Africans and one Indian who had had BCG vaccination but did not have a scar) showed that previous BCG vaccination does not protect people with HIV infection from developing tuberculosis.² This should be another strong reason for not vaccinating such people, whatever their immune status. Normal chest radiographs in such people presenting with fever either with or without cough are well reported,^{2,3} and induction of sputum with hypertonic saline in patients unable to produce sputum is an important aid to diagnosis apart from invasive procedures such as bronchoscopy with bronchoalveolar lavage and biopsy.

Clinicians should also bear in mind that ethambutol should be included in the initial treatment regimen if central nervous system disease or disseminated disease is present or resistance to isoniazid is suspected.⁴ Lifelong chemoprophylaxis with isoniazid after completion of treatment is controversial and, on the basis of current evidence, should be judged on each patient's clinical circumstances. As tuberculosis can be one of the earliest infections to occur in the course of HIV infection,⁵ such people should not be universally subjected to chemoprophylaxis with isoniazid throughout the time that they are still immunocompetent. Regular clinical monitoring may be more appropriate until more data are available as the guidelines acknowledge that reactions to antituberculous drugs are commoner in HIV positive people and desensitisation with steroid cover may be considered in difficult cases.⁶

Tuberculosis in people with HIV infection is an important public health issue in the United Kingdom as it is one of the few diseases that can be infectious to both HIV negative and HIV positive contacts and has a good outcome if treated appropriately.

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1 Subcommittee of the Joint Tuberculosis Committee of the British Thoracic Society. Guidelines on the management of tuberculosis and HIV infection in the United Kingdom. *BMJ* 1992;**304**:1231-3. (9 May.)
2 Ong ELC, Mandal BK. Tuberculosis in patients infected with the human immunodeficiency virus. *Q J Med* 1991;**291**:613-7.
3 Pedro-Botet J, Gutierrez J, Miralles R, Coll J, Rubies-Prat J. Pulmonary tuberculosis in HIV-infected patients with normal chest radiographs. *AIDS* 1992;**6**:91-3.
4 American Thoracic Society. Mycobacterioses and the acquired immunodeficiency syndrome. *Am Rev Respir Dis* 1987;**136**: 492-6.
5 Barnes PF, Bloch AB, Davidson PT, Snider DE. Tuberculosis in patients with human immunodeficiency virus infection. *N Engl J Med* 1991;**324**:1644-50.
6 Ong ELC, Mandal BK. Multiple drug reactions in a patient with AIDS. *Lancet* 1989;ii:976-7.

EDITOR.—Effective surveillance of tuberculosis is required to monitor the effectiveness of the recent guidelines on the management of tuberculosis and HIV infection¹ and reassure those responsible for controlling infection that in the United Kingdom