removed the next morning. After I had recovered from the anaesthetic the house officer came to tell me that while I had been anaesthetised the senior registrar had in fact examined me internally. I was furious at this.

I am fortunate to have had enough support to recover from the hell of being abused. But even being able to articulate my needs was not sufficient to protect me from further abuse. How much worse it must be for those who haven't been as fortunate as I in recovering from such trauma.

I write because I wish to bring this to the attention of the medical profession. I have seen many doctors who, sadly, lack sensitivity in dealing with this issue. Since internal examinations are common it is important that doctors respect the needs of those who may require particularly sensitive handling. Maybe much could be learnt from my experience, which did go badly wrong.

1 Campling P. Working with adult survivors of child sexual abuse. *BMJ* 1992;**305**:1375-6. (5 December.)

EDITOR,—I was heartened by the sensitivity of Penelope Campling's editorial on working with adult survivors of child sexual abuse.[1] Even though child abuse—mainly the sexual variety—has been a more open subject in the past few years, I have not seen many articles of this nature in professional magazines.

Though not wishing to detract from Campling's writing, I wish to point out that many of the reactions, feelings, and transferences that she mentions also apply to those who were not sexually abused but "only" emotionally abused. The sexual act does violate the child, but the real damage is done to the psyche, wherein lies the trauma—the memory, the damage to self esteem. Similar damage is done by repeated emotional abuse, and I believe that the true damage done by this is only beginning to be seen. Repeated put downs, rejections, and criticism can produce the same feelings of confusion, fear of all authority figures, guilt, low self esteem, seeking for love, and other reactions listed by Campling.

Doctors who diagnose child sexual abuse from their observations of a patient may be wrong. Sexual abuse may not be the abuse that took place. Emotional abuse may be the underlying trauma (and this often comes with a fear of being physically harmed). As an adult survivor of both types of abuse and a professional dealing with others coming to terms with their abuse, I know. I am delighted to see people like Campling dealing with this subject with such caring.

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Bronchospasm during disulfiram-ethanol test reaction

EDITOR,—We would like to add some comments to the case report of E Zapata and A Orwin dealing with bronchoconstriction and hypertension experienced by a 45 year old man taking alcohol and disulfiram.[1]

We investigated the role of acetaldehyde in causing respiratory distress in subjects with impaired hepatic acetaldehyde dehydrogenase activity or receiving treatment with disulfiram by giving alcohol to guinea pigs.[2] Intravenous administration of acetaldehyde elicited a prompt rise in blood pressure and increase in bronchial resistance associated with augmentation of blood histamine concentration. Antihistaminic agents significantly antagonise acetaldehyde actions. Furthermore, we established that captopril, a well known angiotensin converting enzyme inhibitor, strikingly potentiates the effect of acetaldehyde. Thus, since the administration of NK2 tachykinin receptor antagonist reduced the response of the airways to acetaldehyde in animals pretreated with the angiotensin converting enzyme inhibitor, a contribution of neuropeptides to acetaldehyde activity was considered a critical point. Unexpectedly, the infusion of very low doses of acetaldehyde induced hyperresponsiveness of bronchial smooth muscle to substance P. Acetaldehyde is clearly able to interfere with complex neural and humoral mechanisms regulating the bronchomotor and cardiovascular functions.

These findings in guinea pigs may have implications for human respiratory pathology. Robuschi *et al* showed that aerosol challenge of asthmatic patients with acetaldehyde caused a consistent reduction in forced expiratory volume in one second.[3] Thus it is reasonable to presume that asthmatic patients, particularly those treated with angiotensin converting enzyme inhibitors, may face exacerbation of broncho-obstructive symptoms on taking alcohol and its conversion to acetaldehyde. Furthermore, in patients with a genetic disposition or pathological alterations characterised by a reduced aldehyde dehydrogenase activity[1 4 7] the repeated alcohol intake, through the mechanism here described, may result in bronchial hyperresponsiveness and haemodynamic alterations.

More information will put into correct perspective, in conjunction with the known pathogenetic factors (allergic, inflammatory, infectious), the contribution of alcohol intake to the multifactorial pathogenesis of bronchial asthma.

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Monitoring cyclosporin treatment

EDITOR,—D J M Reynolds and J K Aronson provide a clear approach to monitoring cyclosporin treatment after organ transplantation.[1] Although they acknowledge the use of this potent immunomodulator in other diseases, however, they fail to emphasise the differences in monitoring cyclosporin treatment in autoimmune diseases.

As a second line agent cyclosporin is a relatively new drug in rheumatoid arthritis. Generally, lower doses are used than in organ transplantation. Most rheumatologists agree that monitoring the treatment in rheumatoid disease does not routinely require measurement of blood cyclosporin concentrations[2 3]: patients' blood pressure, renal biochemical variables, creatinine clearance, and full blood count are monitored. Patients with rheumatoid disease are particularly susceptible to nephrotoxicity.[4] A rise in creatinine (<30% above the baseline value) is acceptable and, indeed, expected.[5] Cyclosporin concentrations are checked only in those patients who are receiving additional treatments that may have an appreciable interaction with the immunosuppressant.

Cyclosporin is becoming increasingly important in the management of a wide variety of diseases; monitoring of the drug will vary according to the underlying disorder.

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Treating hypertension in elderly patients

EDITOR,—I agree with Kevin O'Malley and Eoin O'Brien that "a cautious approach to implementing the results of recent landmark studies may be wise."[1] The authors declare that the consistency across a range of interventional studies in the past decade is impressive. On the contrary, the consistency is virtually non-existent, except that all interventions seemed to do some good, but not the same good, and all but one had no significant effect on overall mortality.

An Australian trial of treatment of mild hypertension in elderly people showed no reduction in fatal strokes but a 75% reduction in ischaemic cardiac deaths.[2] In the double blind component of the study by the European working party on high blood pressure in the elderly there was an apparently impressive but barely significant reduction in cardiac deaths (−47%, p=0·048).[3] The authors reported "a non-significant decrease in cerebrovascular death"—that is, no significant reduction in cerebrovascular death.

In contrast, Coope and Warrender reported a 50% reduction in total strokes, but the "incidence of myocardial infarction was unaffected by treatment."[4] At the end of the five year systolic hypertension in the elderly trial there was a reduction in total stroke incidence of 6 per 1000 patient years of treatment.[5] There was no significant reduction in deaths from myocardial infarction. In the Swedish trial in old patients with hypertension the reduction in overall mortality was largely due to a reduction in mortality from stroke.[6] There was no effect on fatal or non-fatal myocardial infarction. The recently published Medical Research Council trial showed a reduction in strokes but no significant reduction in coronary events.[7]

If treatment was applied to the generality of elderly people with hypertension these conflicting trial results suggest four possible outcomes. Firstly, cardiac death would be reduced but not death due to stroke. Secondly, death due to stroke would be reduced but not cardiac death. Thirdly, both cardiac death and death due to stroke would be reduced, but the near universal absence of a fall in reported overall mortality makes this unlikely. Fourthly, neither death due to stroke nor cardiac death would be reduced. On this basis I suggest that there remains a great deal of doubt that mild to moderate hypertension in elderly people should be treated.

In elderly people in industrialised societies the prevalence of arterial hypertension approaches or