unfounded, as at no stage during our involvement with this lady did she demonstrate any affective symptoms whatsoever. Instead she demonstrated the typical features of anorexia nervosa, as defined by the DSM–III–R criteria, of severe weight loss, a typical distortion of body image, and a fear of becoming fat. Amenorrhoea was obviously not of relevance because of her advanced age. Although it is difficult to be certain of the exact circumstances surrounding her first episode fifty years beforehand, her account, confirmed by that of other family members, is strongly indicative of anorexia nervosa, and gives no cause to promote an alternative diagnosis.

Reports of eating disorders in the elderly are not restricted to that of Bernstein (1972), which O'Shea rightly questions. We have reviewed sixteen cases of anorexia nervosa or bulimia nervosa in the over-fifties reported in the recent literature (Cosford & Arnold, submitted), and found serious doubt regarding the diagnosis in only four. The remaining twelve demonstrated the typical psychopathology of an eating disorder, apart from the patient's age. Interestingly, half had arisen for the first time in later life, while the other half had initially arisen in the patient's youth, and they either remained persistently unwell for many years, or suffered a relapsing and remitting course, with remissions of up to fifty years in some cases.

It is our assertion that advanced age should not by itself rule out the diagnosis of anorexia nervosa, when the typical features are undoubtedly present, and there is no alternative major physical or psychiatric cause for the patient's symptoms.

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Buspirone-induced mania: possible interaction with disulfiram

SIR: McIvor & Sinanan (*Journal*, January 1991, **158**, 136–137) report the case of an alcoholic patient who developed mania when treated with buspirone 20 mg/day and disulfiram 400 mg/day. The authors attribute mania to buspirone treatment and suggest a possible mechanism consisting of facilitation of dopaminergic functions by this drug.

Although azapirones can have antidepressant effects (Robinson, 1989) and buspirone could cause mania by itself, we have reasons to believe that in this particular case the interaction between buspirone and disulfiram could cause mania. Drs McIvor & Sinanan point out that no significant interaction has been reported with buspirone and disulfiram. However, both drugs can interact in at least two sites: (a) the brain, and (b) the liver.

(a) The brain: disulfiram inhibits dopamine beta-hydroxylase resulting in decreased brain norepinephrine levels and increased dopamine levels (Ciraulo & Ciraulo, 1988). Through this mechanism disulfiram can enhance the facilitation of dopaminergic action by buspirone and contribute to the development of mania. In fact, the patient reported by Drs McIvor & Sinanan required high-dose neuroleptic medication to control his symptoms, which concurs with the presence in the synaptic cleft of high concentration of dopamine (as could be expected with this mechanism). Furthermore, it has been observed that alcoholics with low cerebrospinal fluid dopamine beta-hydroxylase activity are more likely to develop dysphoric symptoms (Ciraulo & Ciraulo, 1988). It could be argued that the patient took disulfiram only intermittently, which makes a possible buspirone–disulfiram interaction less likely. However, two factors must be considered: (a) disulfiram is eliminated within three to five days and its principal metabolite (diethyldithiocarbamate) also inhibits dopamine beta-hydroxylase; (b) the patient was taking 400 mg/day of disulfiram which is a high dose compared with the standard dose recommended to avoid adverse effects: 250 mg/day (Kaplan & Sadock, 1988).

(b) The liver: disulfiram is an inhibitor of the hepatic microsomal enzyme oxidase system (Ciraulo & Ciraulo, 1988). As buspirone is eliminated by oxidative metabolism (Gammans *et al*, 1986), disulfiram could contribute to the elevation of plasma levels of buspirone by delaying its elimination.

In summary, by either or both of these mechanisms, disulfiram could have contributed, with buspirone, to the development of mania. Therefore we think that a possible buspirone–disulfiram interaction must be considered when buspirone is prescribed as a non-addictive anxiolytic in alcoholic patients on disulfiram.

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Clinical irrelevance of HAD factor structure

SIR: Moorey *et al* (*Journal*, February 1991, **158**, 255–259) happily reported the two distinct, stable, consistent and reliable factors on factor analysis of the Hospital Anxiety and Depression Scale (HAD) which corresponded to the questionnaire's anxiety and depression subscales. Clinical experience of using the HAD, does not however, support this bi-dimensional solution. In our trial with HAD in patients suffering from clinical anxiety and depression we found that the mean anxiety subscale scores were much higher in the depressed patients – higher than the anxiety score of the anxiety disorder patients and also higher than the depression subscale score of the depressive disorder patients! Thus the two subscales hardly discriminate between clinical anxiety and depression, irrespective of the statistical derivations. In fact, the authors themselves noted substantially more anxiety than depression but they have not reported the anxiety and depression subscale scores separately in their depressed and anxiety patients. It seems that the total HAD scores are more meaningful clinically than the subscale scores. At the moment HAD is a widely used instrument and it would be very useful if prospective authors gave subscale scores and correlated these with the clinical observations.

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A possible antiaggressive effect of cyproterone acetate

SIR: Some clinical studies suggest that testosterone may influence certain kinds of aggressive behaviour in human males (e.g. Olweus *et al*, 1980).

We would like to report the case of a young psychotic patient whose aggressive and impulsive behaviour has been dramatically improved by a competitive inhibitor of the androgen binding site – cyproterone acetate (CPA) – whereas a LHRH analogue treatment (reducing the plasma testosterone level to castration values) failed to maintain the antiaggressive effect of CPA.

Case report. A 30-year-old man with a psychiatric diagnosis of infantile psychosis was studied. Since the age of ten, he had shown aggressive and impulsive behaviour treated with varying combinations of neuroleptic drugs, propanolol, benzodiazepines or carbamazepine, depending on the patient's level of aggression.

During the month which preceded this study, his aggressive behaviour gradually increased, resulting in verbal and physical aggression against either objects or staff members, leading to occasional injuries. The patient's last treatment which included levomepromazine (300 mg/d), carbamazepine (600 mg/d) and diazepam (200 mg/d), was absolutely ineffective. Instances of physical aggression occurred almost every day and sometimes twice a day, and required immediate sedative injection and isolation. At the time of this observation, the level of aggression assessed on the OAS Scale (Yodofsky *et al*, 1986) reached 18.

Before starting the CPA treatment, the plasma testosterone level was within the normal range (5.2 ng/ml) as were the other plasma hormone levels (LH, prolactin, FSH, Δ4 androstene-dione).

In a first step, the CPA treatment was added to the former treatment at a daily dosage of 50 mg and slowly increased up to 200 mg daily. His aggressive behaviour gradually decreased over a period of one month, making it possible to reduce the neuroleptic treatment down to 150 mg/day and to withdraw the carbamazepine and diazepine prescriptions. The level of aggression became 0 on the OAS. No significant change was observed in the plasma testosterone level.

This improvement was maintained for a period of four months. Then we decided to test if a reduction in the testosterone gonadal secretion (without affecting the androgen receptor level) would produce the same clinical effect. A monthly injection of a long-lasting LHRH analogue treatment was started and the CPA treatment was withdrawn after the second injection (gosereline acetate 3.6 mg monthly).

Whereas the testosterone level dropped to castration values (the 0.25 ng/ml remaining originated from the adrenal), the aggression score returned to its initial value fifteen days after the interruption of CPA treatment. CPA had to be reintroduced in order to control the patient's aggression.

The beneficial effect of CPA treatment on aggressive behaviour could be explained either by its well known antiandrogenic effects or by its progestational properties leading to antioestrogenic effects. LHRH analogue treatment is devoid of progestational effects. With regard to the lack of clinical efficacy of LHRH analogue treatment, the antioestrogenic hypothesis seems more plausible.

Some preclinical studies suggest that, in fact, oestrogens could be more involved than androgens in