mental state examination and, where indicated, a physical examination or selective physical examination, and special investigations. This applies as much to people with Down's syndrome as it does to the general population.

We hope our papers help to establish that people with Down's syndrome experience increased rates of certain psychiatric disorders, and that the pattern of adaptive behaviour in these individuals changes with age. There is, of course, a distinction between descriptive psychopathology by which a psychiatric diagnosis is made, and aetiological factors which contribute to the described illness. Having established different rates of psychiatric disorders, the next stage is to determine the relevant aetiological factors. The view of Drs Prasher & Krishnan, that sensory impairment and medical illness account for the altered rates of psychiatric disorders, is speculative. We look forward with great anticipation to the publication of results from the Birmingham Down's syndrome study to support their hypothesis.

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Combination treatment of depression

SIR: The question of how to treat depressed patients who have failed to respond to a first antidepressant agent is of great clinical importance. Seth *et al* (*Journal*, October 1992, **161**, 562–565) describe the successful treatment of such patients by the addition of a selective serotonin reuptake inhibitor (SSRI) to the tricyclic antidepressant; a potentially more straightforward manoeuvre than lithium augmentation or changing to a monoamine oxidase inhibitor. Points by subsequent correspondents are well made (Cowen & Power, *Journal*, February, 1993, **162**, 266–267) but, given the potential hazards of combining drugs, may fail to address one critical issue – would *changing* to an SSRI be equally as effective as adding one? One influential study (Nolen *et al*, 1988) found no benefit in changing to fluvoxamine for patients who were tricyclic antidepressant non-responders, and is frequently cited to dismiss the strategy. However, a number of other authors have found that a clinically significant proportion of tricyclic non-responders do respond to a subsequent SSRI given alone (Lingjaerde *et al*, 1983; Delgado *et al*, 1988; Beasley *et al*, 1990; White *et al*, 1990). Admittedly these have been open studies but I would argue that it is premature to dismiss changing to another non-monoamine oxidase inhibitor antidepressant in favour of combination drug treatment on current evidence. Further research is required to determine the best 'second-step' treatment for patients failing to respond to an adequate trial of a tricyclic antidepressant drug.

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Dysfunctional attitudes and Beck's cognitive theory of depression

SIR: I would like to comment on Brittlebank *et al*'s article (*Journal*, January 1993, **162**, 118–121) on autobiographical memory in depression. They compared a measure of autobiographical memory to the Dysfunctional Attitudes Scale (DAS; Weissman, 1979) with respect to: (a) whether scores on each measure fell as depression scores decreased (i.e., whether the variables behaved as state or trait markers); and (b) whether scores on each measure predicted depression at three- and seven-month follow-up. The findings concerning autobiographical memory are interesting

and noteworthy, and the authors adequately discuss their implications.

Perhaps because their focus was on autobiographical memory, the authors do not emphasise the implications of the DAS results, which I believe are also important. The implications are twofold. Firstly, with respect to the state v. trait marker issue, the DAS results are consistent with Beck *et al*'s (1979) cognitive theory of depression, in that dysfunctional attitudes did not significantly change as depression remitted (i.e., behaved as a trait marker would). This is particularly important in light of the controversy over whether cognitive styles are indeed trait-like depressogenic vulnerability factors (see for example, Coyne & Gotlib, 1983).

Secondly, although it is not unreasonable to compare the autobiographical memory measure to the DAS in predicting depression at follow-up, it should be noted that Beck *et al*'s (1979) theory predicts an association between dysfunctional attitudes and future depression in the presence, but not the absence, of negative life stress (see Haaga & Beck, 1992 for a recent treatment of this issue). Put differently, the theory contains a diathesis-stress component, and both the diathesis (dysfunctional attitudes) *and* the stress (negative life stress) are necessary to initiate the sequence towards depression. Because the Brittlebank *et al* (1993) study did not assess negative life stress, the study cannot speak for the validity of this aspect of the theory, neither with respect to predicting future depression, nor treatment response.

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Blood-letting in bulimia nervosa

SIR: In their recent article, Drs Parkin & Eagles (*Journal*, February, 1993, **162**, 246–248) wrote that "there does not appear to be any reference in the literature to deliberate blood-letting (...) in association with eating disorders". It is indeed a rare association and, in my own clinical work with eating disordered patients over the past 15 years, I have seen it only once in a nurse with chronic anorexia nervosa. Nevertheless, I have found a few references in the literature. In a comment on "Blood-letting as purging behaviour" Cosman 1986 briefly presents the case of a 26-year-old woman. In the French literature, deliberate blood-letting has been described by Jean Bernard (1969) under the term "syndrome of Lasthénie de Ferjol", named after the heroine in a 19th century French novel by Barbey d'Aurévilly. A relationship between this syndrome and anorexia nervosa has been suggested by the Flemish psychiatrist Myriam Van Moffaert (1976). Finally, the similarities have been discussed by Loloum *et al* (1985).

These French authors present the case of a 26-year-old woman with anorexia nervosa (fasting alternating with binge-eating episodes) who, during her brilliant nursing studies, started blood-letting herself several times a week.

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Tourette's syndrome and the amygdaloid complex

I was interested to read the thought-provoking commentary by Handley *et al* (*Journal*, March 1993, **162**, 421) on my article (Jadresic; *Journal*, October 1992, **161**, 532–534) which advocates a key role for the amygdaloid complex (AC) in Tourette's syndrome (TS).

One of their contentions is that the cadence, pitch, volume, etc., in the coprolalia of TS differs from that of purely emotionally generated obscenity and this would therefore militate against the involvement of the amygdala in TS. The verbal as opposed to the prosodic aggressive content of vocalisations raises interesting theoretical links with models of laterality in brain function. This model includes the well known association of the dominant hemisphere with verbal aspects of language and the more controversial association of non-dominant hemisphere involvement in prosodic aspects of language