are now hundreds of clinicians (nurse therapists, psychiatrists, psychologists) doing exposure therapy for OCD throughout the UK. UK purchasers of health care thus have a widely available option of choosing exposure therapy for OCD which is usually acceptable, inexpensive and more effective than is medication. The 30% or so of OCD sufferers who have concomitant depressed mood need both exposure and antidepressants.

Similar sidelining in the *BJP* occurs when Tallis (1995) writes "Behaviour therapy remains the most effective and thoroughly evaluated psychological treatment of OCD" in a section actually devoted to "Cognition and cognitive therapy" and omits discussion of inexpensive yet effective self-exposure therapy. Readers and patients interested in such valuable self-exposure therapy may find it helpful to read Lee Baer's excellent book *Getting Control* (1991).

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Clozapine-induced hypersalivation and the alpha2 adrenoceptor

SIR: Hypersalivation can be a troublesome side-effect of clozapine, limiting its usefulness in the management of some cases of schizophrenia (Fitton & Heel, 1990), but the pharmacological basis of this remains obscure. As well as its action at several dopamine and serotonin receptor subtypes, clozapine can block muscarinic acetylcholine receptors and the alpha2 adrenoceptor (Reynolds & Czudek, 1995), which have opposing effects on the control of salivation. While muscarinic blockade leads to diminished salivary secretion, alpha2 antagonists can increase salivation (Berlan *et al*, 1992), suggesting that this latter action may underly clozapine-induced hypersalivation. To test this hypothesis, we administered the alpha2 agonist lofexidine to one patient in whom the side-effect was particularly distressing.

The 54-year-old man had suffered from chronic schizophrenia since 1959. In 1993 he was commenced on clozapine; the dose of clozapine was increased to 900 mg per day and there were improvements in his social interactions, communication and personal hygiene. Unfortunately he had severe hypersalivation which did not respond to thioridazine, procyclidine, or reduction in dose of clozapine to 600 mg daily. With the addition of lofexidine 0.2 mg b.d., there was a significant improvement in the hypersalivation, with nursing staff observing that the previously persistent dripping of saliva onto the patient's clothes quickly ceased. Because of the risks involved lofexidine was not continued for more than one month and it was subsequently necessary to discontinue the clozapine.

Lofexidine is an alpha2 agonist which is licensed in the UK only for the short-term treatment of opiate withdrawal symptoms. It could not be used for long-term treatment without running the risks of depression and exacerbation of psychosis, which limited the usefulness of the similar agent clonidine in mania (Hardy *et al*, 1986), obsessive–compulsive disorder and Tourette's syndrome (Ashanuddin, 1982). However, awareness of the pharmacological basis of the hypersalivation may permit development of strategies to combat this limiting side-effect of a major antipsychotic.

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Suspected congenital sertraline dependence

SIR: We report the suspected occurrence of neonatal withdrawal symptoms from maternal use of sertraline throughout pregnancy, which as far as we are aware has not been previously reported. Withdrawal syndromes for sertraline, fluvoxamine and paroxetine have been reported to occur in adults (Szabadi, 1992; Louie *et al*, 1994; Pyke, 1995).

The mother, aged 32 years, had been commenced on sertraline 150 mg daily, increasing to 200 mg within 2 weeks, for a depressive illness. Within 3 months she became pregnant and remained on sertraline 200 mg throughout her pregnancy. She also took lithium and thioridazine for the first 6 weeks of her pregnancy only, unaware at this time of the pregnancy. The pregnancy proceeded without complication. After a normal full term delivery of a healthy boy she continued with sertraline until 3 weeks postpartum, when this was stopped abruptly. She had breastfed since delivery.

The baby, previously feeding and developing well, after one day developed symptoms of agitation, restlessness, poor feeding, constant crying, insomnia and an enhanced startle reaction. These symptoms were intense for approximately a further 48 hours and then began to subside over the next few days. The mother remained well with no adverse symptoms after stopping sertraline.

Although withdrawal symptoms may have been expected from shortly after birth, it is possible that breast milk concentrations were sufficient to prevent the symptoms noted after complete cessation. The half-life of sertraline of around 26 hours may account for the onset of symptoms in the infant about one day later. Unfortunately, we were unable to measure breast milk sertraline concentration. In addition, the manufacturing company, Invicta Pharmaceuticals, have no data regarding breast milk concentrations or the transplacental transfer of sertraline, and no studies have examined these factors. In view of the popularity of the SSRIs we would call for the manufacturing companies to investigate these important parameters.

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Behavioural toxicities of antidepressants

SIR: We were surprised to read the editorial by O'Hanlon & Freeman in April's *BJP* (1995). It is essentially a précis of a paper published elsewhere by the same writers, with substantially the same content, references and, indeed, verbatim quotations Authors of contributions to the *BJP* are required to avow that "their (article's) substance has not been published or submitted for publication elsewhere". Perhaps this rule should apply also to editorials.

Readers of the full article, published as a 'review' in the *Journal of Drug Development and Clinical Research* (1995) will also be aware that the work was sponsored by the manufacturer of an antidepressant that has been found to be behaviourally toxic. This information was not supplied in the *BJP* editorial. This is puzzling, as one of the authors has castigated contributors for submitting articles without declaring the contributions of potentially interested parties (Freeman, 1993).

In the future, if editorials are written as the result of commercial sponsorship, then this information should be available to readers. They can then judge for themselves whether there is a potential conflict of interest.

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— & O'HANLON, J. F. (1995) Acute and subacute effects of antidepressants on performance. *Journal of Drug Development and Clinical Research*, **7**, 7–20.

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AUTHORS' REPLY: The purpose of any editorial is to express the authors' opinions, within a limited space, concerning an issue of important scientific interest. It is entirely different from that of a lengthy review of research, even if the conclusions of that paper also reflect the same opinions. Our major review on antidepressants was read by the Editor, but he invited the editorial instead, on grounds of space. This clearly did not preclude the later submission of the full study to another journal.

Of course, neither our editorial, nor any other in the *British Journal of Psychiatry*, was written with any sort of "sponsorship". To suspect the same implies either naiveté or a conspiratorial outlook on life.

We find it regrettable that Kerr *et al* refer to "an antidepressant that has been found to be behaviourally toxic". Whether the drug they refer to (dothiepin) is behaviourally toxic was not crucial to our conclusions. Our major point in both the review