# Correspondence

EDITED BY LOUISE HOWARD

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## Risperidone treatment of amphetamine psychosis

**Sir:** Risperidone is an atypical antipsychotic drug indicated for psychoses in which both positive and negative symptoms are prominent. Misra & Kofoed (1997) have reported for the first time a case of methamphetamine-associated psychosis responding to risperidone. In that report risperidone was prescribed after the discontinuation of methamphetamine. We report another case of amphetamine psychosis responding to risperidone while still on amphetamine.

Mrs P., a 76-year-old married Caucasian woman, has been on dexamphetamine for narcolepsy since the age of 28. She remained well until September 1996 when she developed acute schizophrenia-like psychosis. She experienced auditory hallucinations – voices told her that her husband was having an affair, and she believed people were following her and commenting on her activities. She also believed an implant had been placed in her skull and voices were transmitted through a satellite. She attempted to remove the implant with a needle. There were no signs of an affective disorder. She required formal admission and responded well to sulpiride while still taking dexamphetamine.

She discontinued sulpiride after five months and again developed paranoid delusions and auditory hallucinations. She believed she would be burgled and killed, and stopped eating and drinking, believing her food was poisoned. She was admitted formally and treated with risperidone 3 mg and dexamphetamine 15 mg daily. Routine investigations were normal. She had recovered fully by discharge four weeks later. She was followed-up regularly and has remained well on risperidone and dexamphetamine for the past 18 months.

To our knowledge this is the first report of a successful outcome of risperidone treatment of amphetamine psychosis while on risperidone and dexamphetamine concomitantly. It has been a well-tolerated and effective treatment and may be useful for other narcolepsy sufferers.

**Misra, L. & Kofoed, L. (1997)** Risperidone treatment of methamphetamine psychosis (letter). *American Journal of Psychiatry*, **154**, 1170.

**A. Jha, H. Fourie** West Herts Community Health NHS Trust, Logandene, Ashley Close, Hemel Hempstead, Herts HP3 8BL

## Amiodarone-induced depression

**Sir:** Amiodarone hydrochloride is a class III antiarrhythmic agent used for the management of ventricular and supraventricular arrhythmias. The adverse effects of amiodarone are well documented and include cardiovascular effects (i.e. severe bradycardia and sinus arrest), thyroid malfunction, severe pulmonary toxicity (including pulmonary fibrosis and interstitial pneumonitis), liver toxicity, peripheral neuropathy, myopathy, ataxia and tremors (British Medical Association & Royal Pharmaceutical Society of Great Britain, 1998). However, amiodarone's effect on mental state does not appear to have been reported in the currently available literature, with the exception of an amiodarone-induced delirium that occurred 17 days after starting therapy in a 66-year-old man (Trohman, 1988).

We report this case in which amiodarone may have played a significant role in the timing, as well as in the clinical presentation, of a depressive episode.

A 65-year-old school dinner lady presented for admission in a severe retarded depressive state with obsessive–compulsive features. She appeared physically unwell and somewhat older than her age. The presenting clinical features included psychomotor retardation, fatigue, social withdrawal, and morbid preoccupation with health. The depressive symptoms had had a gradual onset, beginning nearly eight months before presentation. At that time she had suffered a myocardial infarction which was further complicated by ventricular tachycardia for which she was treated with amiodarone. At the time of presentation, she was already on a selective serotonin reuptake inhibitor, prescribed a few months earlier, but with little or no effect. Past psychiatric history revealed one major depressive episode that occurred postnatally and was treated with electroconvulsive therapy 37 years previously. She recovered fully and remained well until the onset of the presenting complaints. The patient has longstanding obsessive–compulsive disorder but with no real evidence of any previous neurotic or social decompensation at any time prior to the present episode.

The rather atypical presentation of her depressive episode in its mode of onset, course, duration, limited response to antidepressants and a predominant somatic component, favoured the possibility of an underlying aetiology other than just functional.

Thyroid function tests were within normal limits, with slightly elevated free thyroxine levels but normal levels of thyroid-stimulating hormone. There was no clinical evidence to suggest a hyperactive thyroid.

After consultation with the medical team, amiodarone was discontinued. Within one week and without any change in psychotropics, a rapid and dramatic improvement was observed in the mental state as well as her somatic symptoms. She became clinically asymptomatic except for her obsessional constitutional traits and longstanding obsessive–compulsive disorder.

The clinical improvement which coincided with the amiodarone withdrawal appears to suggest that amiodarone may be implicated, directly or indirectly, in triggering psychiatric symptoms in hitherto predisposed patients. In this case, we have no reason to assume that the patient's depression may have been caused by any amiodarone-induced thyroid disorder.

Although no firm conclusion can be made based on a single case, we recommend that amiodarone may be relevant in elderly psychiatric patients before assuming functional aetiology for a gradually developing depressive state.

**British Medical Association & Royal Pharmaceutical Society of Great Britain (1998)**

https://doi.org/10.1192/bjp.174.4.366b Published online by Cambridge University Press

*British National Formulary*. London & Oxford: BMA & The Pharmaceutical Press.

**Trohman, R. D. (1988)** Amiodarone induced delirium. *Annals of Internal Medicine*, **108**, 68–69.

**A. Ambrose, E. Salib** Hollins Park Hospital, Hollins Park, Hollins Lane, Winwick, Warrington WA2 8WA

## Psychological treatments for hypochondriasis

**Sir:** Clark *et al*'s (1998) loose use of terms is misleading. Their "cognitive" therapy was in fact cognitive–behavioural therapy (CBT) with (p. 219) "A mixture of cognitive and behavioural techniques" including "behavioural experiments" by imaginal exposure ("inducing symptoms by deliberate body focusing or dwelling on fearful thoughts"), live exposure ("increasing engagement in activities that were avoided because of illness beliefs (for example, exercise)"), and "response prevention for repeated bodily checking and prevention of reassurance seeking . . . others who were normally involved in the provision of repeated reassurance were included in the response prevention programme and were given instructions in how to deal correctly with any further requests for reassurance". Homework included exposure and response prevention (ERP).

In contrast, "behavioural stress management" included only weak exposure *without* mention of 'cognitive' therapy's strong behavioural components of: ERP in the first few sessions; behavioural experiments and response prevention by patients and others to deal with checking and reassurance seeking; and exposure homework. It did include anti-exposure reassurance ("remind patients that previous physical investigations had proved negative and their doctor was convinced they did not have a serious illness"). The procedure is best termed stress management with a small behavioural component late in therapy.

The design's having more behavioural (ERP) experiments in the cognitive (80%) than in the behavioural therapy (0%) sessions shows in Table 1. The Table does not mention exposure homework, but the description (see above) suggests this too was advised more in the cognitive than the behavioural sessions. Because the authors' cognitive therapy was also more behavioural (had more ERP) than their behavioural treatment, their design cannot support the claim that cognitive therapy was a specific treatment, unlike behavioural stress management. They compared CBT (cognitive restructuring plus ERP) on the one hand with stress management including limited exposure and additional methods on the other. The early superiority of their CBT (which was not sustained) could be explained by its greater use of ERP than the stress management protocol which introduced exposure later in treatment.

It is possible that cognitive therapy alone, without behavioural experiments and ERP, may have produced similar improvement, but the study has no such contrast group. What was specific about a form of cognitive therapy that included strong behavioural methods in a design which had no treatment group that omitted both cognitive and behavioural components?

Clark *et al*'s design is out of date, as controlled studies have found in several anxiety disorders, including hypochondriasis, that exposure alone and cognitive therapy alone were each therapeutic in their own right. In depressive disorders too, purely behavioural (without cognitive) methods were just as helpful. None of these controlled studies is cited.

Clark *et al*'s preoccupation with cognitive effects leads them to ignore recent findings that neither cognitive nor behavioural therapy is crucial for improvement. Sufficient yes, necessary no. One or the other can do the trick, and each may be an unwitting way of using other effective ingredient(s) that are as yet unidentified. Future studies are more likely to advance knowledge if they separate cognitive from behavioural components and test whether they work by similar or different mechanisms or in ways that are neither cognitive nor behavioural.

As an aside, on Fig. 1's measure none of the follow-up differences between the two treatments was significant.

**Clark, D. M., Salkovskis, P. M., Hackman, A., *et al* (1998)** Two psychological treatments for hypochondriasis. A randomised controlled trial. *British Journal of Psychiatry*, **173**, 218–225.

**I. Marks** Institute of Psychiatry, De Crespigny Park, London SE5 8AF

**Authors' reply:** Our controlled trial demonstrated that two new treatments, developed by our group, produce substantial improvements in hypochondriasis. Professor Marks quibbles with the labels chosen for the treatments and our use of the term "specific treatment effect". Personally, we are more concerned with effectiveness than with labels. However, our terminology was not inappropriate.

The term 'cognitive therapy' was introduced over 30 years ago and from the start denoted a cognitive theory-based treatment involving verbal disputation *and* behavioural procedures, both of which had the *explicit* aim of changing patients' dysfunctional beliefs (see Beck, 1970). Our cognitive therapy for hypochondriasis has these characteristics. Some people prefer the term cognitive–behavioural therapy (CBT). We chose cognitive therapy not because we think our behavioural procedures are unimportant: quite the contrary. Instead, it was because the term CBT is used in a variety of different, and potentially confusing, senses. For some people it equals cognitive therapy as defined above. For others, such as Marks, it includes a mixture of procedures that are each given with different rationales, viz. an anxiety habituation rationale for exposure and a belief change rationale for verbal disputation.

The term 'specific treatment effect' also has a long-standing meaning, which we adhered to. At least since Gelder *et al*'s seminal paper (1973) on specific and non-specific effects in psychotherapy, the term has been used to denote a demonstration that the effects of a therapy cannot be accounted for simply by a series of specified procedures that would be present in any well-conducted psychological treatment, irrespective of orientation. Our cognitive therapy programme clearly passed this test as it was superior to behavioural stress management on 7 out of 10 hypochondriasis measures at post-treatment, despite behavioural stress management involving the same repeated assessments, being administered by the same therapists for the same amount of time, involving systematic out-of-session homework, and being rated as equally credible by patients. This demonstration of specificity seems rather more convincing than Marks' own claims for specificity in his recent trial of treatments for post-traumatic stress disorder (PTSD) (Marks *et al*, 1998). In that trial, exposure was only superior to the control treatment (relaxation) on three out of nine primary PTSD measures and there was no evidence that patients

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