

logic because of the confounding variable of active or inactive treatment.

Perhaps I have overlooked some strength in the methodology chosen; I would be glad to be corrected if this is the case.

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Reference

HALSTROM, C., CROUCH, G. & SHINE, P. (1988) The treatment of tranquilliser dependence by propranolol. *Post Graduate Medical Journal*, **64** (suppl.), 40–44.

ECT in neuroleptic malignant syndrome

SIR: In his otherwise impressive update on neuroleptic malignant syndrome (NMS), Kellam (*Journal*, August 1990, **157**, 169–173) seems to have included some factual inaccuracies while discussing the treatment of continuing or recurring psychosis after the successful treatment of NMS. Dr Kellam quotes Lazarus (1986) to the effect that electroconvulsive therapy (ECT) is safe after the syndrome has subsided. Lazarus, in his case report, suggested that ECT may offer a safe and rapidly effective intervention in cases of NMS unresponsive to supportive medical therapy.

Similarly, Hermesh *et al* (1987) recommended ECT for the treatment of NMS episodes rather than for continuing or recurring psychosis after the successful treatment of NMS. In my rejoinder to Hermesh *et al* (1987), I did not argue about the safety of further neuroleptic treatment for psychotic illness as Kellam seems to imply. I suggested that the drug treatment (i.e. bromocriptine or dantrolene) should be tried for the management of NMS before resorting to ECT (Adityanjee, 1987). I did mention having used ECT for the management of recurring psychosis after NMS had subsided (Adityanjee, 1987; Adityanjee & Chawla, 1989). Similar practice was adopted by Aizenburg *et al* (1985) who used ECT for the management of psychotic illness after NMS had resolved successfully with supportive treatment only.

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References

ADITYANJEE, P. DAS. (1987) Role of electroconvulsive therapy in neuroleptic malignant syndrome. *Acta Psychiatrica Scandinavica*, **76**, 603–604.

— & CHAWLA, H. M. (1989) Neuroleptic malignant syndrome and psychotic illness. *British Journal of Psychiatry*, **155**, 852–854.

AIZENBERG, D., SHALEV, A. & MUNITZ, H. (1985) The aftercare of the patient with the neuroleptic malignant syndrome. *British Journal of Psychiatry*, **146**, 317–318.

HERMESH, H., AIZENBERG, D. & WEIZMAN, A. (1987) A successful electroconvulsive treatment of neuroleptic malignant syndrome. *Acta Psychiatrica Scandinavica*, **75**, 237–239.

LAZARUS, A. (1986) Treatment of neuroleptic malignant syndrome with electroconvulsive therapy. *Journal of Nervous and Mental Disorders*, **174**, 47–49.

Was Hitler a Christian?

SIR: There are a number of dubious assumptions and implications in Samuel's (*Journal*, July 1990, **157**, 151) argument that Hitler, Mussolini, Stalin, Franco and Hoess were all Christians. Firstly, there is the assumption that anyone who professes to be a Christian is one by definition. Arguably, such a profession does fit the dictionary definition given by Philip Timms, although I would feel that in this context the use of the word 'profess' should more meaningfully be taken to include observance and practice of, rather than simply confession of, faith. Secondly, there is an implication that the failure of the Roman Catholic Church to proscribe *Mein Kampf* amounts to an acceptance of its contents not only by the Catholic Church (which is itself highly debatable) but also by the wider Christian Church as a whole.

The most serious implication of Dr Samuel's argument, which is both unjustified and offensive, is that there is some causal connection between the professed Christian faith of the individuals named and the acts of atrocity and inhumanity that they perpetrated during their lives. The lives that they led, as recorded in history, clearly betray the insincerity of any profession of Christian faith that they made. The teachings of Christ made clear that it was not those who 'professed' righteousness, but those who were truly repentant, who found favour in God's sight.

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This correspondence is now concluded.

Anorexia nervosa and OCD

SIR: I read with interest the paper by Holden (*Journal*, July 1990, **157**, 1–5) on the evidence for a

relationship between anorexia nervosa (AN) and obsessive-compulsive disorder (OCD). However, I find his analysis too selective and would like to suggest several missing points.

- (a) Evidence suggestive of a positive relationship
 - (i) Abnormal brain serotonin has been reported in both AN and OCD. Thus tryptophan has been used to treat both conditions, while a recent open trial of fluoxetine, a serotonergic reuptake blocker, has shown favourable result in weight maintenance of AN patients (Weltzin *et al.*, 1990). This trial was based on the belief that AN is related to OCD, which responds to fluoxetine treatment. This weakens Holden's conclusion that there is currently little advantage in joining the two conditions "until linkage of the syndromes is shown to have meaningful benefits for management".
 - (ii) Maudsley Multiphasic Personality Inventory profiles of AN and OCD patients are remarkably similar, both showing abnormal elevations on the scales of depression, psychopathic deviate, psychasthenia and schizophrenia (Goodwin & Andersen, 1984; Carey *et al.*, 1986).
- (b) Evidence against a positive relationship
 - (i) Psychodynamically, AN is a defence against underlying deficits in the sense of self, identity and autonomy, which is believed to be due to a paucity of encouraging responses to child-initiated cues in early parent-child interactions. The characteristic defence mechanism of the AN patient is rigid denial. In contrast, OCD is usually construed as a regressive fixation at the anal-sadistic era of psychosexual development, which is characterised by the defences of undoing, isolation, displacement and reaction formation.
 - (ii) In terms of psychological treatment, behavioural therapy alone is generally ineffective in AN patients who typically defy treatment contracts and create power struggles, but is the most important form of therapy for OCD. If AN were an authentic "compulsion neurosis" as noted by the author, it should respond well to exposure and response prevention. Besides, dynamically oriented individual therapy is the cornerstone to the management of AN, but is often pessimistically contra-indicated in OCD.
 - (iii) Although both conditions are regarded as multifactorial in aetiology, current evidence is

generally moving more towards sociocultural hypotheses for AN, but neurobiological for OCD. This is hardly examined by the author.

Overall, there is suggestive neurochemical and psychometric evidence, debatable phenomenological resemblance, tenuous genetic and epidemiological data, and contradictory treatment results to support AN as an OCD. Theoretical arguments apart, anyone who has worked in psychotherapy with these two groups of patients will not dispute the striking dissimilarity in the therapist's reaction towards the resistant, unconcerned ("let me be") anorectic patient, and the distressed, eagerly help-seeking ("let me not be") obsessive-compulsive patient. For this reason alone, I believe they are disparate conditions!.

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References

- CAREY, R. J., BAER, L., JENIKE, M. A., *et al.* (1986) MMPI correlates of obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *47*, 371-372.
- GOODWIN, R. & ANDERSON, A. E. (1984) The MMPI in three groups of patients with significant weight loss. *Hillside Journal of Clinical Psychiatry*, *6*, 188-203.
- WELTZIN, T. E., KAYE, W. H., HSU, L. K. G. & SOBIEWICZ, T. (1990) Fluoxetine improves outcome in anorexia nervosa. Paper presented at the 143rd Annual Meeting of the American Psychiatric Association, 12-17 May, New York, USA.

Toad-lickers psychosis – a warning

SIR: Since we in England generally follow our American colleagues in experiencing novel epidemics of psychoactive substance abuse, the following warning may be timely. The skin glands of bufo toad species secrete the hallucinogen bufoterine, which although having only 0.1% of the hallucinogenic potency of lysergide, is active orally and produces the same effects. The Australian cane toad is popularly kept as a pet in the US, and licked by its owners for the resulting hallucinatory effects. The phenomena has caused so much concern that the state of South Carolina is considering legislation to ban ownership of cane toads. Bufoterine is a monoamine oxidase inhibitor, blocks uptake of noradrenaline and has complex effects on reflex activity in the spinal cord (Bowman & Rand, 1980). These active properties are responsible for the more unpleasant side-effects of toad-licking: sweating, distressing palpitations, vomiting and involuntary defaecation.