The Dexamethasone Suppression Test in Depressed Kuwaiti Patients

SIR: We read with interest the multicentre WHO collaborative study of the response to the dexamethasone suppression test (DST) (*Journal*, April 1987, **150**, 459–462). The only Arab centre involved in this study was in Casablanca, Morocco (12 patients). In Kuwait, we recently carried out a similar study on 19 Kuwaiti depressed in-patients (11 males and 8 females; mean age = 34.3 ± 2.2 years) and 23 healthy controls.

Our study supported the view that post-dexamethasone plasma cortisol concentration is higher among depressed patients than among controls. The mean post-dexamethasone plasma cortisol concentration was 95.5 ng/ml, which is higher than the Casablanca results (37.4 ng/ml) and shows that variations are present even between the two Arab centres.

The percentage of abnormal responses was 32%, which is higher than at the Casablanca centre (25%). The range of variation in the WHO study was from 15% to 71%. DST response was normal in the controls.

The absolute value for serum cortisol decrease between 8.00 h and 16.00 h could not be used to predict and separate patients who were suppressors from those who were non-suppressors.

There was no statistically significant difference between suppressors and non-suppressors in the Hamilton Rating Scale for Depression (HRSD) scores before and after treatment. No differences were detected in total HRSD scores between males and females, either before or after treatment.

No significant correlation was elicited between initial serum cortisol levels and HRSD scores before or after treatment.

Comparing two types of antidepressants (amitriptyline and mianserin), it was found that the former was a more effective treatment in both suppressors and non-suppressors, while the latter was clearly ineffective as a treatment in suppressors. It seems that the question of possible involvement of factors such as geographical location and climate has to be considered when interpreting differences between centres, especially those with similar ethnic features.

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Reference

HAMILTON, M. (1967) Development of a rating scale for primary depressive illness. *British Journal of Clinical and Social Psychology*, **6**, 278–296.

Chronic Schizophrenia and Long-term Hospitalisation

SIR: In Dr Abrahamson's first letter about 'institutionalisation' (*Journal*, September 1986, **149**, 382), he appeared to argue that negative symptoms in schizophrenia were immutable. This premise seemed to be based only on the fact that the relationship between negative symptoms and length of stay often found in cross-sectional studies (he quoted a study by George Brown and myself in this context) could be explained by an accumulation of more severe cases in hospital as the less severe were discharged. In my reply (Journal, January 1987, **150**, 129–133), I pointed out that we had specifically argued against any cause and effect relationship and, instead, had provided evidence, in a comparative longitudinal study, that 'poverty of the social environment' did seem to increase 'clinical poverty', at least in a proportion of cases. More specifically designed experiments could not disprove the hypothesis.

In his second letter (Journal, November 1987, **151**, 708), Dr Abrahamson said that he had intended to agree with our point, and I am grateful for the assurance. However, what he gave with one hand he took away with the other, since he went on to juxtapose two quotations (100 pages apart) that he thought demonstrated that we had, after all, claimed that length of stay in hospital caused deterioration. What we in fact suggested was that social conditions in the three hospitals were responsible, acting through a biological vulnerability, for part of the deterioration *or lack of deterioration* found in their respective residents. The final sentence quoted by Dr Abrahamson illustrates the point. One fifth of the patients who remained in hospital during 8 years of the study (excluding those discharged) actually improved with time. If social conditions change with time, so will the negative symptoms of some patients. Length of stay, as such, is not a key factor.

That my interpretation of Dr Abrahamson's first letter was close to the mark is also suggested by his new statement that attitudes to discharge from hospital are entirely explained in schizophrenic patients by the severity of their disorder. We found that moderately impaired patients were more likely to have favourable attitudes than the severely impaired, but that controlling for severity, for example of blunting of affect or poverty of speech, did not remove the relationship to length of stay. Many people with

schizophrenia can react in the same way as normal people in similar circumstances. Apathy is not always a symptom of schizophrenia.

At the end of his first letter, Dr Abrahamson said that neither a disease process nor a social model would be sufficient to explain 'institutionalisation'. The introductory chapter of *Institutionalism and Schizophrenia* (Wing & Brown, 1970) contains a set of theories, since further developed, concerning the interaction between host and environment.

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WING, J. K. & BROWN, G. (1970) *Institutionalism and Schizophrenia*. London: Cambridge University Press.

Incest and Anorexia Nervosa: What is the Link?

SIR: I, and I suspect many other mental health professionals, have been struck by the recent large increase in the number of women referred for assessment and management of the longer-term consequences of sexual abuse in childhood and adolescence.

While every woman's story is unique, one particularly common sequence of events is 'difficult childhood', sexual assault in adolescence, teenage anorexia nervosa, followed by difficulties in adult sexual adjustment, often with chronic eating disorders. Over the past three months I have seen six women who presented with this personal history.

A review of the standard psychiatric texts failed to clarify the role of sexual assault in the aetiology of female eating disorders. A literature search by Medline and Psych-Info revealed just two significant papers. Openheimer *et al* (1985) systematically enquired into the sexual histories of 78 women with DSM – III defined anorexia nervosa or bulimia nervosa. Fifty of the 78 subjects reported coersive sexual events, coital and non-coital, prior to the onset of the eating disorder. They concluded, "frequently the sexually molested subject has feelings of inferiority or disgust about her own femininity and sexuality. These may become entangled with concern about her body weight, shape and size . . . Do these events play a part in causing the disorders which they precede? At present we cannot be sure".

Sloane & Leichner (1986) presented five case histories of women presenting with eating disorders after significant sexual assaults. In reviewing the numerous psychodynamic theories of the causation of eating disorders, they observed, "once the factual nature of such material is seriously entertained, certain aspects of anorexia nervosa cease to be as perplexing and can be seen as a rather logical outcome of early sexual trauma". Sloane & Leichner also comment on the underlying themes of avoidance of sexual feelings, sex role conflicts, concern with sexual activity, and sexual appearance.

Both papers emphasised that the many subjects who are sexually abused do not develop 'apparent' eating disorders and many subjects with eating disorders have 'apparently' not been sexually abused. I have qualified both comments with 'apparent'; the uncertain epidemiological status of both conditions and the massive denial of sexual assault maintained by many victims cautions against dogmatic statements.

If sexual abuse and eating disorders are causally linked, what are the mediators of that link? Based on my recent case series I would offer one explanation. Many female anorexics present with obsessional symptoms. Very often these reflect an underlying insecure, under-confidant and unassertive personality who copes by emotional over-control, the anankastic personality trait.

All the patients I have seen describe 'unhappy childhood' with either (a) dominant, controlling, and destructive mothers and distant or absent fathers who capitulate to the dominant spouse, or (b) dominant but emotionally distant and physically punitive fathers with distant or indulgent mothers. From this early environment the child develops a poor self-image and learns to suppress all emotional responses, particularly negative ones, to "keep the peace at all costs" by taking all responsibility for any actions against or by it.

When the adolescent is sexually abused, often in a coersive way by a male family member, the intensely painful emotions are handled by the girl as she has learnt to handle all emotions, by introjection and acceptance of unreasonable guilt compounded by fear, anger at herself and the perpetrator, and despair at her inability to share these painful emotions with anyone else. As Sloane & Leichner observe, certain aspects of anorexia nervosa (guilt, anger, autonomous control, and avoidance of secondary sexual characteristics) become logically understandable as a way of denying the unbearable and guilt-ridden sexual past.

Subsequently, often coincidental with moving away from home to college, nursing or marriage, the burden of keeping silent is lifted and the victim is able to experiment and explore other aspects of relationships with men. Many slowly begin to trust and relax to share intimate feelings. If this trust is abused, then