British Journal of Psychiatry (1986), **149**, 520–525

Correspondence

Dominance Hierarchies in Psychotherapy Groups

Sir: The article by Kennedy and McKenzie (*Journal*, June 1986, **148**, 625–631) is fundamentally flawed. The underlying assumption of the authors is that social behaviour has biological determinants, and that the social behaviour of the highest organism phylogenetically will include the social behaviour of the lower primates, such as chimpanzees. In particular, the authors say there is a "natural" tendency to form hierarchies and furthermore, these hierarchies have a regulatory function, resolving the tension between the development of cohesion and conflict. The central error is to ignore the importance of culture and variation in human social organisation. It is not true that humans are "naturally" hierarchial. There is much research by social anthropologists describing in detail how many societies of people who live by hunting and gathering are, remarkably, non-hierarchial (e.g., Lee & DeVore, 1968). Amongst some hunters and gatherers there is a leader whose position is attributable as much to personality factors as his skill in hunting; however there are no ways to impose his authority, and his position is very tenuous. When conflicts arise the hunting band is likely to divide.

Many anthropologists (e.g., Sahlins, 1977) consider direct extrapolation from ethological studies to human behaviour to be an abuse of the method. One reason why this occurs is that aspects of social organisation, the culture, of the writers are seen in the animal society organisation.

This is made relevant in considering the subject of hierarchies in psychotherapy groups: these groups share the culture of the society of which they are a part. When the authors see hierarchy in psychotherapy groups they will consider it to be "natural". They are, in fact, seeing the way the group reproduces the outside social structure.

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ECT as a Contributor to the Production of Delusional Misidentification

Sir: I read with interest the conclusion of Dr Hay's paper (*Journal*, June 1986, **148**, 667–669).

I have seen a 58 year old woman who presented with a three week history of delusional misrepresentation which began after she had an anaesthetic. She had become constipated with the onset of a depressive illness two months previously, and believed that she had cancer. She sought a surgical opinion. A sigmoidoscopy was performed under anaesthetic which excluded bowel pathology, but confirmed constipation. She had suffered no previous psychiatric illness. Both the patient and her husband agreed that the delusional misidentification began after the sigmoidoscopy. This suggests that an anaesthetic agent rather than electrical stimulation may be the cause of such symptoms.

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Lithium Augmentation of Tricyclics

Sir: Pai *et al* (*Journal*, June 1986, **148**, 736–738) describe a further series of patients where it is claimed that lithium augments the effects of another antidepressant. Nearly all the other studies making this claim, including those cited in the report, seem to be describing the same phenomenon. Namely, that some severely depressed patients who have completely failed to respond to a first-line antidepressant dramatically respond within anything from a few days to a couple of weeks to the addition of lithium. The simplest and most economical explanation of nearly all of the cases reported is that lithium has had a major acute antidepressant effect. Although most of the authors consider this possibility, it is usually dismissed and some complicated explanation for either summation or potentiation is invoked to explain the observation.

All of the controlled studies of the acute antidepressant effects of lithium, subsequent to the negative result of Stokes *et al* (1971) have shown lithium to have a major acute antidepressant effect, substantially better than placebo and at least, if not better, than an antidepressant such as imipramine.

Two controlled trials conducted by myself and colleagues in Dundee (Worral *et al*, 1979) are representative and discussed the issues at that time. One of those trials, in which tryptophan was compared with lithium and tryptophan in combination, is regularly quoted in the subsequent combined studies and used as evidence for lithium having an augmenting effect on other antidepressants. We specifically stated that the design of that trial could not possibly prove that. The simplest explanation in that trial was that lithium was acting alone and, in truth, tryptophan was only used because we did not obtain ethical permission for a placebo comparison.

What seems to have happened is that influential figures twenty years ago stated that lithium did not have important acute antidepressant effects and, with minor modifications, that statement has been authoritatively repeated in nearly all reviews since. What the combined studies have shown is that when a first-line treatment has proved ineffective and the clinician has then tried lithium, he has not believed his own eyes when he has seen a major acute antidepressant response, and some form of potentiation or augmentation of the first drug by lithium is instead suggested.

Why expose patients simultaneously to two drugs without first trying each separately, especially if, as is likely, continuation treatment is going to be needed? It may be that in a few patients lithium does need to be used along with another antidepressant, or a neuroleptic, and it would be surprising if on occasion two antidepressants from a different class did not have more effect than one. A controlled trial to prove that would be feasible, but before going to that trouble an open-minded reappraisal of the effects of lithium alone might make such a trial less necessary.

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At Risk Drinking

Sir:I was delighted to read the article by Dr King (*Journal*, May 1986, **148**, 533–540). In this and other articles Dr King has used the CAGE Questionnaire. Unfortunately, he has repeated a failure of other authors to attribute the questionnaire to the correct source. The CAGE questions were developed by me and my colleagues at the University of North Carolina in the late 1960s. The CAGE questions have been used in many different studies by now, and recently (Ewing, 1984) I published a paper in the *Journal of the American Medical Association* describing their origins, clinical use and efficacy. That reference might be the best one for Dr King and others to use in future.

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Anhedonic Depression

Sir: Young *et al* (*Journal*, March 1986, **148**, 257–267) have suggested the possible existence of two subtypes of endogenous depression—anhedonic subtype and vegetative sub-type. Earlier, Hibbert *et al* (1984) reported that symptoms of reduced interest and pleasure and of low mood represent features of the state of depression which are related to each other, but are not much related to the more 'biological' symptoms of reduced sleep and appetite.

In a study of 34 RDC major depressives, I classified the patients as anhedonic and non-anhedonic based on high or low scores on the sub-scale anhedonia-asociality of the Scale for the Assessment of Negative symptoms (SANS) (Andreasen, 1981). The anhedonic sub-type had a significantly ($P < 0.001$) longer duration of illness than the non-anhedonic sub-type. Almost 20% of anhedonic depressives had a duration of illness longer than nine months with treatment. The response to tricyclic antidepressants was unsatisfactory in the anhedonic depressives and the need for adjuvant therapy with electroconvulsive shocks and antipsychotics was significantly ($P < 0.01$) more often required for them. Anhedonic depressives also required a much longer duration of treatment than the non-anhedonic depressives (Chaturvedi & Sarmukaddam, 1986). Interestingly, anhedonic depressives had significantly higher total scores on SANS than non-anhedonic depressives.

These findings help further in identifying an anhedonic sub-type of depression. However, in my study, no demographic differences were observed