

carried out by an extremely skilled and experienced practitioner. As he points out, supportive psychotherapy is most often carried out by the multidisciplinary team rather than specialist psychotherapists, and often there is no supervision or training available. An inexperienced therapist might be able to practice the distributive psychotherapy referred to by Holmes, that is, to spend a part of the session questioning and history-taking, and the rest in a more passive, listening and understanding mode. However, judging the niceties of when to draw attention to transference issues, or at what point to challenge instead of contain the patient, are no matters for a novice therapist, especially when faced with the type of patient thought to require supportive rather than dynamic therapy—in other words, the more damaged and vulnerable patient.

I suspect that Dr Holmes' own training and skills are not shared by the majority of those medical and non-medical practitioners of supportive psychotherapy who might well come to grief if they attempt to blend supportive and dynamic features in the way he suggests.

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### **Ethnic Density and Schizophrenia**

SIR: It is doubtful if 'ethnic density', as determined simply by population size within an area (especially one as large as a Regional Health Authority), can have any useful meaning. This measure used by Cochrane & Bal (*Journal*, September 1988, 153, 363–366) may be taken less as a determinant of 'density' than as denoting the presence or absence of a particular community within the area. The daytime density of psychiatrists in Belgrave Square is high compared with Eaton Square, but this tells us little about the individual motivations and psychology of these particular individuals, or their patterns of settlement within the Square.

Minority groups are hardly free to 'diffuse' throughout an area for reasons purely associated with individual psychopathology, as contrasted with external constraints associated with a racially biased provision of housing and employment. The actual pattern of distribution within a smaller local area may be a more significant indicator of those demographic factors possibly associated with psychopathology, such as 'social drift', group cohesion, and community support, let alone nosocomial factors which we now suspect account for much of the differential 'pathology' between ethnic groups.

To take the case of Nottingham: the 'foreign-born' community has settled predominantly in the inner-city areas where it also has the highest rate of schizophrenia, arguing against the ethnic density hypothesis of Faris & Dunham (Giggs, 1986). While higher rates of schizophrenia for each group taken separately are found among European and South Asian migrants in the outer-city council estates than in 'inner-city, low status' areas, the highest rates among West Indians occur when they live in the 'inner-city, high status' areas. For each of the three groups, however, the highest rates of settlement are in the inner city. Thus the ethnic density hypothesis seems to fit Europeans and South Asian migrants, but not West Indians.

It is in the specific pattern of settlement within a particular area, with all its political, economic, and psychological constraints, that explanations of differential pathology and access to services can be found. While I am sure this was not the intention of the authors, the bald 'ethnic density' argument by itself comes perilously close to ethological and thus biological explanations (e.g. Esser & Deutsch, 1977).

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### **Treatment of Suicidal Manic Depression**

SIR: I read with great interest the paper by Schou & Weeke (*Journal*, September 1988, 153, 324–327). In their guidelines for improving suicide prevention in manic depressive illness they state that "continuation or prophylactic treatment with antidepressants should presumably be carried out with full therapeutic dosage". They base this argument mainly on their finding that 13 patients who were treated prophylactically with sub-therapeutic doses of antidepressants committed suicide. However, 10 patients who received full therapeutic doses of antidepressants also committed suicide. In Modestin's (1985) study too, 21% of the patients had received full therapeutic dosage, compared with 26% who were

on smaller doses of antidepressants, at the time of suicide. Although Myers & Neal (1976) found that only 5 out of 44 patients had been given antidepressants in optimal dosage at the time of the suicide, their optimal dose was based on the "manufacturers recommended full dose". Often this is less than the therapeutic full dose employed by clinicians, and therefore it is not possible to draw any definite conclusions from their results.

From these findings it is difficult to justify the authors' above recommendation. There is an urgent need for further long-term studies in unipolar depression to establish the adequate prophylactic dosage of antidepressants. Until such time as these are carried out, it seems reasonable to use 'sub-therapeutic doses' for prophylaxis, just as we do with lithium in bipolar disorder and neuroleptics (depot medication) in schizophrenia.

Secondly, as the authors mention, it has been shown that the full prophylactic effect of lithium may not be achieved until after 6–12 months of treatment. If this is the case, how could one justify its use and continuation/prophylactic treatment of first choice in unipolar patients, since most patients are at risk of relapse in the first 6–12 months after recovery? For this reason I am inclined to agree with the recommendation made by Coppen *et al* (1978) that lithium prophylaxis should be used only in those unipolar patients who have had three or more attacks of depression.

As pointed out by Myers & Neal (1978), "the major difficulty in preventing suicide in psychiatric patients is to know when, in what may be a long illness, the danger periods occur". For example, in the above study the majority of suicides (28 patients) occurred during unfinished treatment with ECT or antidepressants. This suggests that the chances of suicide are greater when patients begin to recover from a severe depressive illness, and extra vigilance and care is necessary during this period. This is obviously of great importance when ECT is given to out-patients.

Again, most of the relapses are known to occur within the first 6 months of recovery after an illness. Hence careful, regular follow-up in a psychiatric clinic by an experienced psychiatrist, who knows the patient well, is as important as prophylactic drug treatment.

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#### Chronic Depression

SIR: The article by Scott (*Journal*, September 1988, **153**, 287–297) provides an excellent review of chronic depression. However, I do feel that she fails to emphasise the major role that alcohol abuse is likely to play in both chronic and resistant depression. Alcohol abuse is extremely common, and a recent study (Glass & Jackson, 1988) found that in two psychiatric hospitals over a 12-year period a steady 9–11% of the total number of diagnoses was accounted for by alcoholism, with 30–40% of alcoholics receiving an additional diagnosis. Most doctors, including psychiatrists, are also extremely poor at detecting alcohol problems among their patients.

Heavy alcohol use and affective illness often coexist (Dorus *et al*, 1987), and it is common clinical experience that depression rarely resolves with treatment while the patient continues to drink.

Recent work has demonstrated that one possible mechanism for this effect is the ability of alcohol to prevent the  $\beta$ -adrenergic receptor down-regulation that occurs with chronic antidepressant treatment (Linnoila *et al*, 1988). As the regular amount of alcohol intake in a clinical population to produce this effect is not known, great care should be taken in establishing the drinking habits of any patient with a seemingly chronic or resistant depression.

Scott mentions that some studies have demonstrated particularly low serotonergic function in chronic depression compared with acute depression, and recent work (Badawy *et al*, 1988) indicates that alcohol may be potent in decreasing serotonergic activity. Interestingly, current research also suggests that the new, highly selective serotonin reuptake blockers may have a special role to play in chronic and resistant depression.

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