Brit. J. Psychiat. (1982), **141**, 313–328

Correspondence

***Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader, and check the accuracy of all* references in Journal style.**

THE PRESENT STATUS OF FIRST-RANK SYMPTOMS

DEAR SIR,

Steven Hirsch (*Journal*, April 1982, **140**, 421) has admirably collected the literature reporting symptoms of depression occurring in the course of a schizophrenic illness. He concludes that depression is not *caused* by neuroleptic medication of schizophrenics. He dates the observations back to 1967 (Helmchen and Hippius, 1967) but here he is wrong. May I draw attention to the paper a decade before, by Segal (1956) (the paper was delivered the year before at a conference). This of course pre-dates any serious or prolonged use of chlorpromazine.

In that article, Segal—"As treatment progresses . . . the [schizophrenic] patient comes more and more frequently to experience, for a short time, depressive anxieties". In addition she also observes—"For the schizophrenic . . . this situation [i.e. depression] is intolerable, and therefore the steps that the patient has taken towards sanity have to be reversed . . . The saner part of the [patient] is lost". And her paper points out that it is usually the doctor who ends up despairing and depressed.

This is a more complex interaction between depression and schizophrenia than the "discovered check" model that Hirsch suggests. Segal's model is a developmental to-and-fro, trying to show how the schizophrenic makes efforts to take a developmental step forward, which depresses him, so he sheds it.

Segal's paper is historical evidence supporting Hirsch's view that depression is not a drug side-effect, because the paper predates neuroleptics. It also points to the accuracy of observations made by psychoanalysts (also their explanations); and that these can be useful to psychiatrists. Hirsch makes a plea to relinquish 'ideal' categories of disease for a more empirical approach to diagnosis. Symptom clusters must not be confused with diseases. That too might be learned from psycho-analysts.

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PSYCHOPATHOLOGY IN EPILEPSY

DEAR SIR,

I read with interest the excellent report by Kogeorgos, Fonagy and Scott (*Journal*, March 1982, **140**, 236–43) which dealt with some aspects of the controversial relationship between epilepsy and psychopathology. I would like to offer a few comments concerning one aspect of their study.

Kogeorgos *et al* report that they failed to replicate our previous findings of an interaction between seizure type and the age at onset of epilepsy (Hermann, Schwartz, Karnes and Bahdat, 1980), i.e. increased psychopathology in individuals with an adolescent onset of temporal lobe epilepsy (TLE) but no effect of age at onset in individuals with primary generalized epilepsy. I take exception to their contention for the following reasons: (1) We found the age at onset effect *only* in patients with a duration of epilepsy of from 1 to 6 years. As reported, we found no such effect for individuals with a duration of epilepsy for 7 or more years. As the mean duration of disorder of the Kogeorgos *et al* sample is 15.6 years, the age at onset effect is not to be expected and indeed was not found. Therefore, their findings might actually be considered to be in agreement with ours: (2) Even *if* all their subjects had epilepsy for 6 years or less, attempts at replication would probably be hindered by two additional factors: (a) Our focal epilepsy group consisted *only* of individuals with TLE while the Kogeorgos *et al* focal sample apparently included some patients with focal epilepsy of non-temporal origin; (b) Our dependent measures were the clinical scales of the Minnesota Multiphasic Personality Inventory (MMPI) and the age at onset effect was not found on all the scales and was, in particular, *absent* from the so-called neurotic scales of the

MMPI (Hypochondriasis, Depression, Hysteria). Our age at onset effects were found on the Psychopathic Deviate scale and three of the so-called MMPI psychotic scales. As Kogeorgos *et al* used a measure which seems to be particularly sensitive to neurotic symptomatology (Crown-Crisp Experiential Index), one might reasonably expect to find little similarity between our results.

We speculated that there was a complex interplay between seizure type, age at onset, and duration of disorder in the predisposition to specific types of psychopathology. Further work will be needed to support or refute that particular contention.

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BENZODIAZEPINES AND EFFECTIVENESS OF ECT

DEAR SIR,

We note the remark in Dr Elia's letter (*Journal*, March 1982, **140**, 322) concerning the Northwick Park ECT trial that "Johnstone *et al*, write that improvement scores were similar in patients with and without diazepines". Since no such statement appears in our paper it seems that the puzzlement Dr D'Elia has experienced arises more from the discrepancy between the findings of the trial and his own expectations than from an obscurity in our account. In fact the statement quoted earlier in Dr D'Elia's letter that "improvement scores were similar in patients with and without diazepam" is correct. Eighteen of the 62 patients who finished the course (8 on real ECT and 10 on simulated) received diazepam either as 5 mg thrice daily or as diazepam 10 mg in occasional doses to relieve distress. All the patients in the trial received a benzodiazepine hypnotic (nitrazepam) and this is clearly stated.

If Dr D'Elia wishes to reject the conclusions we have drawn from our study on the grounds that hypnotic/sedative medication was not discontinued for the period of convulsive therapy he will find few studies of ECT which he will be able to accept. Many authors are vague on this issue. We have tabulated information on concomitant medication from a number of studies (Table).

TABLE

Concomitant drug therapy (sedative, anxiolytic or antidepressant drugs given in addition to trial therapy) administered in trials of ECT

Information not given:	
Miller *et al* (1953)	
Brill *et al* (1959)	
Wittenborn *et al* (1962)	
Wilson *et al* (1963)	
McDonald *et al* (1966)	
Smith *et al* (1967)	
Cronin *et al* (1970)	
Information incomplete:	
Harris and Robin (1960)	—sodium amytal 3-6 gr when sedation was required
Robin and Harris (1962)	—groups shown retrospectively to be comparable for 'additional treatment'
Fahy *et al* (1963)	—barbiturate hypnotics were given for severe insomnia
Halliday *et al* (1968)	—some patients given antidepressants of unstated dose and others not; no statement on other drugs; barbiturate sedation as required
Freeman *et al* (1978)	—some patients given antidepressants of unstanted type and dosage and others not; no comment on hypnotics or other sedatives
Lambourn and Gill (1978)	—psychotropic drugs, except benzodiazepine hypnotics, were withdrawn
Taylor and Fleminger (1980)	—attempt made to withdraw additional medication apart from benzodiazepine hypnotics
Detailed information provided:	
Cronholm and Ottosson (1960)	—insulin (16 cases); phenobarbitone 25 mg+0.16 g opium tincture three times per day (31 cases); promethazine (5 cases); meprobamate (3 cases); chlorpromazine (3 cases); amylobarbitone (1 case); no concomitant drugs (28 cases)
Herrington *et al* (1974)	—an unsuccessful attempt was made to avoid hypnotic or sedative medication. A benzodiazepine hypnotic was finally given in 4 cases and diazepam in doses up to 30 mg/day in 11 cases
West (1981)	—amitriptyline 50 mg at night given to all patients

Thus the view that hypnotic/sedative medication has in some way obscured the efficacy of ECT in the

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