intensity". The third patient was not helped by the treatment. The length of the follow up period was only five months. These results surely do not warrant such a dogmatic and optimistic conclusion. This needs to be said as we are being told on the one hand that pimozide is a therapeutically effective drug for such patients, and now that pimozide plus behaviour therapy or behaviour therapy alone can work the miracle. A larger number of patients, stringently defined inclusion criteria, controlled studies and a much longer follow up period are required before any such claim can be made.

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DIAGNOSTIC USE OF SLEEP DEPRIVATION

DEAR SIR,

It is generally agreed that the improvement in mood that follows Sleep Deprivation (SD) in depressed patients is usually short-lived (Post *et al*, 1976). For this reason, the therapeutic use of SD has been questioned (Knowles *et al*, in press).

There is, however, one use of SD which has received no mention in the literature, that is, as an aid to diagnosis. In our experience, SD can be decisive in clarifying the sometimes very difficult differential diagnosis between depression and dementia. We have studied a series of such cases referred, because the diagnosis was obscure, to the Treatment Evaluation Unit, Kingston Psychiatric Hospital. In some cases, one 40-hour period of SD resulted in a complete reversal of mood and a dramatic return to normal intellectual function. The duration of this reversal, though it may be brief, is usually long enough to allow psychometric testing to be done to determine whether there is intellectual deterioration. When faced with such a diagnostic question, it is also clinically useful to have a measure of the degree of recovery that is attainable, thus setting a goal for subsequent treatment. Knowing that the syndrome can be reversed has allowed us to undertake treatment with a more precise indication and with greater confidence in the results than would have been possible otherwise.

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ADOPTION RESEARCH IN SCHIZOPHRENIA

DEAR SIR,

I was involved recently in the adoption of the baby of a sixteen-year-old schizophrenic, whose own mother had also suffered from schizophrenia. The social workers explained that the adopting parents were entitled to the background information about the baby's origins, and I agreed to meet them. Questions were asked about the heritability of schizophrenia, and even what signs to look out for in adolescence in the unfortunate event that the daughter should develop the illness. I realized that the child entered its family trailing a background of schizophrenia, and would be watched closely all its life to see if the hereditary taint would show itself in abnormality.

What is the bearing of this on the adoption research from Oregon and Denmark (see Gottesman, 1978), which I had thought represented cast-iron evidence for schizophrenia being to a substantial extent truly inherited by genetic mechanisms? If there was some transmission of background information to the adopting parents, as can occur in this country, did it invalidate the aim of the research to separate genetic and environmental influences on the children studied?

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Reference

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EARLY AGE OF ONSET OF PSYCHIATRIC DISORDER AND THE PROPORTION OF ILL RELATIVES

DEAR SIR,

Family studies of psychiatric disorders have consistently revealed higher rates of morbidity in the relatives of patients with an earlier onset of disorder. From a review of 18 studies, we recently noted that this relationship appeared to be nonspecific and held for affective disorders in general, bipolar and unipolar subtypes, alcoholism, and possibly schizophrenia. In an almost repetitive fashion, several authors concluded that a stronger or independent genetic component was involved in the etiology of early onset disorders,

whereas late onset disorders were variously attributed to exogenous, organic, other genetic, or as yet undetermined mechanisms.

We looked at this relationship in large samples of patients drawn from various drug research programs: the Early Clinical Drug Evaluation Units (ECDEU) databank and the National Institute of Mental Health (NIMH) collaborative studies of drug therapy. Relatives evaluated as part of these programs were diagnosed according to similar diagnostic criteria and consistent applications of the family history method. In spite of lack of age corrections for risk periods (especially for affective disorders), and although only the proportions of patients with ill first-degree relatives (positive family history) could be tabulated, this statistic produced results similar to findings from studies reporting morbidity risk estimates as shown in Figs 1–3, diagnostic nonspecificity was clearly evident.

The implications of such findings are several: (1) higher genetic loading predisposes to earlier onset, (2) genetic subtypes with different ages of onset exist, (3) rearing by a mentally ill parent leads to earlier onset, and (4) social and intrafamilial stress factors experienced in common by relatives increases the likelihood of earlier onset and possibly onset per se. The fact that these possibilities are confounded and not mutually exclusive suggests to us that a better understanding of age of onset might be achieved if the status of this important variable were raised to that of

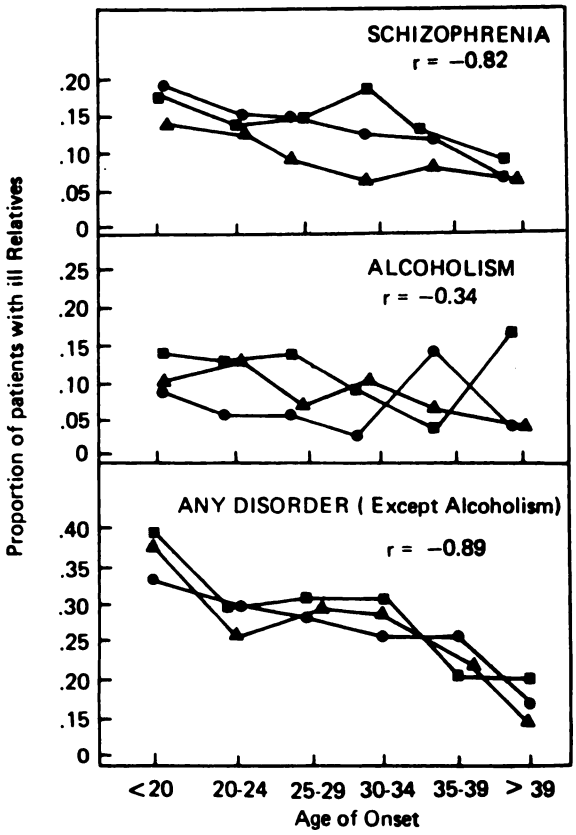

FIG 1.—Proportion of patients with ill first-degree relatives by age of onset for various schizophrenic subgroups.*

* Subgroup codes:
ECDEU Acutes (N = 1,224) ■——■
ECDEU Chronics (N = 1,241) ●——●
NIMH Acutes (N = 469) ▲——▲ (Parent data only)

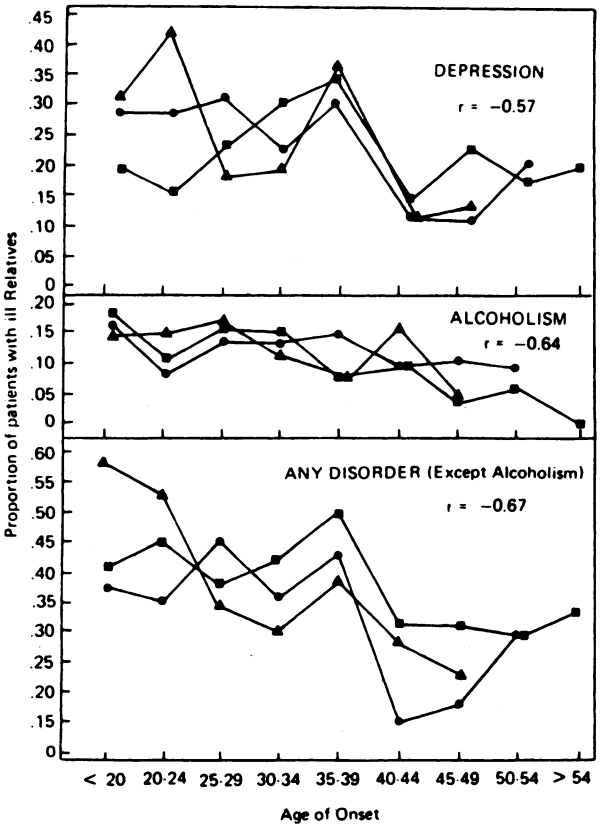

FIG 2.—Proportion of patients with ill first-degree relatives by age of onset for various ECDEU affective subgroups.*

* Subgroup codes:
Major affectives (N = 384) ■——■
Neurotic depressives (N = 511) ●——●
Anxiety neurotics (N = 285) ▲——▲

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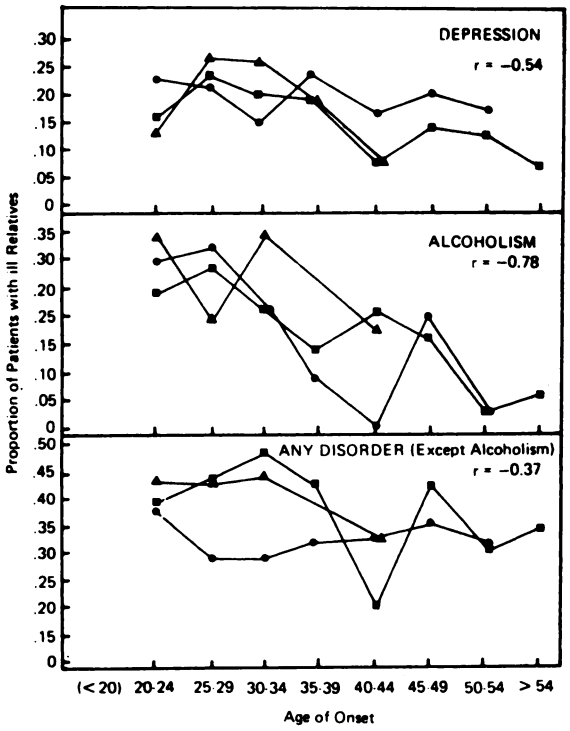

FIG 3.—Proportion of patients with ill first-degree relatives by age of onset for various NIMH affective subgroups.*
* Subgroup codes:
Major affectives (N = 289) ■——■
Neurotic depressives (N = 281) ●——●
Depressed schizophrenics (N = 96) ▲——▲.

an independent variable in future research so that the full range of possible determinants (genetic and non-genetic) could be simultaneously assessed. The diagnostic nonspecificity with which earlier onset appears associated with increased morbidity in relatives may be pointing to the involvement of general, nonspecific factors that are shared in common by all psychiatric disorders irrespective of their genetic independence. Such research might also provide a basis for estimating the extent to which gene-environment interactions play a role in these disorders.

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FURTHER EVIDENCE OF DOMINANT HEMISPHERE DYSFUNCTION IN CHRONIC SCHIZOPHRENIA

DEAR SIR,

The recent literature on hemispheric malfunctioning in schizophrenia seems to suggest different pathophysiological mechanisms: a specific dysfunction (Gruzelier and Venables, 1974), a non-specific overactivation of the dominant hemisphere (Gur, 1978), or a functional disconnection between the two hemispheres (Beaumont and Dimond, 1973). We are currently carrying out a neuropsychological study on a group of right-handed chronic undifferentiated schizophrenic inpatients using two simple tests which have been found useful for the evaluation of specific hemispheric affections: the Short Aphasia Screening Test (SAST), an easily administered test which measures the dysfunctions in language and visuo-spatial systems, and locates the lesions in the left, right or both the hemispheres (Heimburger and Reitan, 1961), and the Quality Extinction Test (QET), which quantifies the percentage of unilateral extinctions during bilateral simultaneous tactile stimulation of the two palms of the hands (Schwartz, 1977).

The results, reported in the Table, for 30 schizophrenics and 30 normals matched for age, sex and handedness, confirm a previous report of a dysfunction in the dominant hemisphere in schizophrenics using the SAST (Taylor, 1979). The results for the QET, here used for the first time in psychiatric patients, show a higher left side extinction in schizophrenics compared with the controls: similar data are

TABLE

Distribution (% of the total number of responses) of the errors for SAST and of unilateral tactile extinction for QET in schizophrenics and normals (n = 30)

	SAST		
	Dominant side	Non-dominant side	No errors
Schizophrenics	63	17	20
Normals	10	10	80
Chi-square = 22.85 (d.f. 2) P < .01			
	QET		
	Dominant extinction	Non-dominant extinction	No extinction
Schizophrenics	97	0	3
Normals	44	12	44
Chi-square = 20.35 (d.f. 2) P < .01			

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