

EDITORIAL

Where have all the catatonics gone?¹

It has become commonplace for textbooks of psychiatry (Slater & Roth, 1969; Lehmann, 1975) to note the apparent decline in the incidence of 'catatonic schizophrenia'. To a great extent the view is due to the impressions of senior clinicians who in their practising lifetime have seen catatonic signs virtually disappear, but there is also documentary support (Achte, 1961; Morrison, 1974).

It is generally agreed that the concept of catatonia was introduced by Kahlbaum (1874) and was included, along with hebephrenia and paranoia, within the category of dementia praecox by Kraepelin (1913). For nearly a half century subsequently there was no serious dispute as to the standing of the entity as a valid schizophrenic type. From about the 1930s, however, it was noted that the incidence and prevalence of the condition was on the decline (Morrison, 1974) and it has virtually disappeared from the modern psychiatric unit. The decline has been attributed to varying causes. Early detection and management of the condition, a liberal and humane hospital atmosphere, and increased and more profitable occupation of the patients were some of the reasons adduced (Achte, 1961; Slater & Roth, 1969; Curran *et al.* 1976). The fact that the fall in incidence preceded modern pharmacological treatment by a generation was taken to mean that social management had effectively dealt with large numbers of patients with a serious medical condition. Another view, principally due to Hare (1974), noted the generally lower 'virulence' of all schizophrenias and suggested that the phenomenon might be due to temporal factors unrelated to any question of modern management.

What is the evidence for the notion that 'catatonic schizophrenia' ever existed? It is still a belief among clinicians – partly due one suspects to the unavailability of his work in English translation for a hundred years – that Kahlbaum (1874, 1973) described cases which were so similar to Kraepelin's in other respects that the latter had no hesitation in including them in the group that came to be called the schizophrenias. This clearly is not so. Looking back on Kahlbaum's cases, it is evident that a number are of neurological, possibly post-encephalitic, origin (C. D. Marsden, personal communication). Even more interestingly, perhaps, a substantial number among the remaining case-histories seem to depict affective disorder. 'Mania', 'melancholia', 'cyclical illness', 'full recovery' are words and phrases used in the description. This was not a solitary report. For the next 100 years (Abrams & Taylor, 1976) the presence of catatonic features amid affective symptomatology was a not uncommon, though unheeded, finding.

That catatonic phenomena occasionally have an organic basis has never been in dispute, and the number of physical causes is now impressive (Gelenberg, 1976). Modern teaching emphasizes the underlying organic basis and the approach to the present-day stuporose patient, for instance, is essentially neurological.

The commonly held view of catatonia – synonymous with catatonic schizophrenia – included both phases of the condition: retardation and excitement. No distinction was drawn between disorders which had one and not the other state and those in which both states were present. In fact, two recent manuals of classification (WHO, 1978; APA, 1980) continue in the same vein. There is evidence now that states of excitement and stupor are quite distinct, not only in obvious presentation but also in course and prognosis (Morrison, 1973). Furthermore, it has been repeatedly noted that individual catatonic symptoms have little or no value in themselves, whereas 'catatonic schizophrenia' has received vastly discrepant prognoses (Wortis, 1975). This would be entirely in keeping with the view held in neurological circles that the course and prognosis of neurological conditions which manifest catatonic features depend wholly on the underlying disease process.

The evidence seems to suggest that the conditions which were studied and described as catatonia

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included conditions which ran a basically schizophrenic course with co-existing catatonic features, and others which were manic-depressive illnesses with catatonic signs and organic states demonstrating these phenomena. In other words, the 'catatonia' in functional illness was fortuitous, or 'added on'. If, as nearly everyone seems to agree, the incidence of these signs has diminished greatly, an explanation must be sought quite separately, one imagines, from that to be found for the alleged decline of, say, the 'virulence' of schizophrenia.

It is not unlikely that these catatonic features were the result of epidemic and endemic infective diseases, quite possibly viral. The viral basis of some catatonic states is acknowledged (Gelenberg, 1976). A possible reason for a decline in the symptomatology of catatonia, then, may be found in the decline of some infective disorder. This is not unique in psychiatric history. There was an impressive fall in tuberculous 'super infection' in psychiatric hospitals at about the same time (Slater & Roth, 1969), and it is of some interest that the predisposition of catatonic patients to contract tuberculosis was noted by Kahlbaum. Nevertheless, if there were any temptation to establish a category of tuberculous schizophrenia, it seems to have been successfully resisted.

It could, of course, be argued that the decline of tuberculosis in psychiatric hospitals coincided with its decline in the general population following the introduction of specific therapy and that no comparable change has occurred in viral infections. But this view would fail to take into account the position of one of the agents which would have helped us with our enquiries – the virus which was probably responsible for epidemic encephalitis lethargica. The decline of post-encephalitic Parkinsonism is now attributed to the disappearance or attenuation of this agent (Walton, 1977).

Evidence based on genetic findings (for example, Gottesman & Shields, 1972) is occasionally presented in support of a catatonic subtype and the fact that all four of the Genain quadruplets (Rosenthal, 1963) had catatonic episodes is cited. The reasoning is suspect. A genetic predisposition to affliction by environmental agents is not in question and a familial incidence of disorder is no more support for the genetic basis to – and, hence, the specificity of – catatonia than it ever was for, say, tuberculosis or the plague.

The hypothesis, then, is that schizophrenia, like other conditions, could co-exist with catatonic signs. There seems tacit recent approval of this notion in the International Pilot Study of Schizophrenia report (WHO, 1973) which states in conclusion: 'Catatonic movements were rated . . . but very few of them [patients] were given a diagnosis of catatonic schizophrenia. Conversely, most of the patients placed in this diagnostic subgroup did not have catatonic movements.'

The varying prognoses mentioned before are understandable if there were a heterogeneous collection of disorders masquerading under the label of 'catatonic schizophrenia'; the oft-reported good response of the catatonic type to electroconvulsive therapy would then begin to make sense. Perhaps many of these catatonic patients belonged, like many schizo-affectives, to the affective camp (Tsuang, 1979).

It seems, therefore, to be important to consider the question of diminishing catatonic symptomatology separately from any consideration of changed prognosis of the underlying states. One is even more cautious in attributing change in type or incidence of symptomatology to treatment or management. Perhaps the more salubrious hospital atmosphere reduces the susceptibility to catatoniogenic infection. There is also the important consideration that what are often taught as cardinal, even pathognomonic, features of conditions like schizophrenia – namely, speech disorder, hallucination, alienation of thought, etc. – could be nothing more than epiphenomena (Hunter, 1973) which might disappear within a generation and raise questions about the alleged change of course in the underlying disease condition when there might have been none. It also makes one understand the confused nature of the results of investigations into psychiatric disorder: experimental rigour is constrained by diagnostic concepts. It also persuades one to re-focus attention on the longitudinal course of psychiatric illness which is once again becoming an important concern, born this time out of research (Crow, 1980). Most of all, it cautions one not to reify prematurely disease concepts and symptomatology.

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I am very grateful to Professor C. D. Marsden for a valuable discussion of the concept.

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