Matters arising

delayed-onset dystonia following perinatal anoxia probably has been underestimated in the past. Thus, 8% (5 of 40) of our patients with torsion dystonia as the only clinical abnormality, had a positive history of complicated labour, but only one patient had suffered severe asphyxia. The practical problem one faces in the other four patients is to decide whether they actually have delayed-onset dystonia or if expression of their dystonia was facilitated by perinatal anoxia. In this relatively small number of patients we have noticed the coexistence of dystonia with other abnormal movements in the form of postural tremor (three patients) ballism (two patients) and reflex or action myoclonus (three cases). The possible diagnostic importance of these findings will only be judged adequately after extensive clinical-epidemiological data about the incidence of other movement disorders in primary torsion dystonia is collected. To our knowledge such information is not yet available. For the time being in the absence of a definitive marker for torsion dystonia, the role of anoxia as an aetiological or predisposing factor in patients with pure dystonia and normal laboratory tests will be difficult to assess.

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References


We are grateful to Obeso and his colleagues for bringing to our attention two probable cases of delayed-onset dystonia due to perinatal anoxia. We would certainly agree that there has been a number of clear descriptions of this phenomenon prior to our report of 1980. We would also agree that the incidence of this occurrence is probably underestimated; since our original report we have seen numerous additional cases.

We strongly concur with Obeso et al that this form of dystonia can be clinically indistinguishable from idiopathic (primary) torsion dystonia. Thus, like Marsden and Harrison1 we believe that a diagnosis of idiopathic torsion dystonia cannot be made in the presence of a history of an abnormal birth. In the absence of markers for the primary dystonias, diagnosis can be difficult where there is some mild or questionable birth complication or development delay. Given the clinical similarity between delayed-onset and primary dystonia, it was our policy in a recent study of the clinical course of autosomal dominant torsion dystonia among non-Jews to exclude from consideration any individual within a pedigree who had a history of frank birth injury.2 Such an exclusion criterion is needed to insure a pure sample of primary dystonics, even within a family group.

Not only will the discovery of markers for the primary dystonias assist differentiation between these two conditions, but also the development of measures of the degree of birth asphyxia. In addition, it is possible that magnetic resonance imaging may prove useful by allowing visualisation of striatal pathology, which would be anticipated in dystonia due to perinatal asphyxia.

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References


Book reviews


Everyone involved in the care of patients with epilepsy is aware of the striking association between epilepsy and psychiatric disorder, which is most common in patients with complex partial epilepsies.

Hughlings Jackson believed that "epilepsy was the cause of insanity in 6% of the insane". Would that things were so black and white! This book, whilst short, explores the shades of grey in some depth. It is a multi-author volume. The Editor contributes both an introductory review and a conclusion. The meat in the sandwich consists of a number of chapters, some of which represent review articles, others of which contain some original investigative data. The topics include interesting chapters on the kindling model of epilepsy which explores both the neurochemical basis, and animal behavioural changes which occur during the process. Subsequent chapters review the roles of antiepileptic drugs in psychopathology, and the associations between depression and psychosis and epilepsy. The practical problems of how to treat psychiatric disorder in patients with epilepsy are considered and perhaps the most useful chapter documents the difficulty of defining the extent to which antidepressant drugs can cause seizures. Finally the possibility that anticonvulsant drugs may have psychotropic properties is explored. A considerable amount has been written concerning the relationship between epilepsy and psychiatric disorder over recent years. This book is a useful summary of the field, but readers should not expect any clear answers to the many questions and problems they face in everyday clinical practice. One area that one was sorry to see receive little attention was the question of whether criminal, violent, or generally antisocial behaviour may occur as a sub-clinical ictal phenomenon. Clinical neurologists rarely accept this but will be well aware that many of their psychiatric colleagues seem to hold a diametrically opposed view as judged by the number of referrals of such patients who have some form of EEG abnormality!

When one accepts that no specific psychiatric disorder can be associated with epilepsy, and that biological factors, drugs