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Annotation

Confusions Concerning Sleep Disorders and the Epilepsies in Children and Adolescents

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There are interesting parallels between sleep disorders and the epilepsies. Firstly, both terms refer to a group of conditions differing in cause, clinical manifestation, natural history and impact on the lives of patients and their relatives. Secondly, the investigation and management of both sets of conditions require a combined physical and psychological approach. Thirdly, neither field is usually covered systematically (if at all) in medical education, although some initiative has been taken in the USA for the introduction of these areas into the curriculum of medical schools (Medical School Curriculum Task Force, 1988). This relative neglect no doubt explains the fourth parallel – that in both sleep disorders and the epilepsies misdiagnosis or inadequate diagnosis appears to be common.

This account is concerned principally with these diagnostic problems, of which there are different varieties. The types of diagnostic error and the conditions in which diagnostic confusion may well occur are set out below:

(a) sleep disorders confused with each other:
- nightmares
- night terrors and other 'arousal disorders'
- rapid eye movement (REM) sleep behaviour disorder

(b) sleep disorders misdiagnosed as epilepsy:
- arousal disorders
- REM sleep behaviour disorder
- head-banging and related phenomena
- nocturnal enuresis
- obstructive sleep apnoea
- automatic behaviour disorder
- idiopathic central nervous system (CNS) hypersomnia with sleep drunkenness

(c) epilepsy misdiagnosed as sleep disorder:
- complex partial seizures of temporal lobe origin
- complex partial seizures of frontal lobe origin
- nocturnal/hypnogenic paroxysmal dystonia
- episodic nocturnal wanderings
- non-convulsive status epilepticus

It is suggested that if the salient features of these conditions are known they will usually be recognised, provided that a detailed account is obtained of each patient's episodes of disturbed behaviour, the timing of the episodes, and the circumstances in which they occur. In some cases, video and physiological monitoring will be needed. Although emphasis is placed on children and adolescents, many of the conditions and issues are relevant to adult psychiatry. Firstly, some comments on present classification schemes are required.

Classification of sleep disorders

Textbooks of paediatrics and child psychiatry often understate the range of sleep disorders. There may simply be brief accounts of night wakening, nightmares, sleep walking and night terrors. The true diversity of sleep disorders is demonstrated in the classification of sleep and arousal disorders compiled by the Association of Sleep Disorders Centers (1979) which however needs some modification to include those sleep disorders and types of sleep behaviour seen specifically in infants and children (Ferber, 1985). At any age, four main categories of sleep problems can be identified: disorders of initiating and maintaining sleep (insomnias), disorders of excessive daytime sleepiness (hypersomnias), disorders of the sleep–wake cycle, and episodic types of behaviour made worse by sleep or occurring exclusively during sleep (parasomnias). Most confusions concerning sleep disorders and epilepsies seem to consist of difficulty in distinguishing between nocturnal epileptic attacks and other parasomnias.

Perhaps especially in children, there is a need to distinguish between sleep disorders and sleep behaviour. The former term implies a problem to the child or parents, whereas a sleep behaviour may or may not be a problem; it may simply reflect a certain phase of development. For example, it is usual for infants to wake repeatedly during the night (Anders, 1979). Such wakenings are only a problem if the child habitually cannot settle back to sleep and cries or disturbs the parents in other ways. Other types of sleep behaviour which are not necessarily problematic include teeth grinding, head-banging or rocking, and snoring, although each of these can be a pointer to a serious condition.

Classification of epileptic seizures and syndromes

A scheme for classifying epileptic attacks (seizures) has been promoted internationally for the last 20 years (Gastaut, 1970; Commission on Classification and Terminology of the International League Against Epilepsy, 1981). Whatever the misgivings expressed by traditionalists, the scheme has the undoubted advantage of encouraging careful clinical observation of seizures in place of simplistic categorisation in terms such as '*grand mal*' and '*petit mal*'. It has become clear that what used to be called 'temporal lobe attacks', for example, are in fact a miscellaneous group of seizures and that the term is of limited clinical value.

Attempts have also been made to solve the more difficult problem of classifying the epilepsies or epileptic syndromes. An account by the Commission on Classification and Terminology of the International League Against Epilepsy (1989) demonstrates the need for accurate investigation of the clinical, electrographic (EEG), aetiological, developmental and other features of the individual case. This degree of assessment permits accurate classification in the vast majority of children (Viani *et al*, 1988). The detailed evaluation required for accurate classification of a patient's seizures and syndrome is not merely an academic exercise: it is of fundamental importance in deciding what investigations are required, the choice of treatment, and in estimating prognosis. Benign centrotemporal (Rolandic) epilepsy of childhood (Blom & Heijbel, 1982) illustrates this point. This is a very common form of childhood epilepsy, probably often grouped previously with other seizure disorders under the general term of 'temporal lobe epilepsy', but now known to be unlike most other partial epilepsies in very important ways. If its characteristic clinical and EEG features are identified, the fear of a structural brain abnormality can be removed and all those concerned can be reassured that spontaneous remission is almost certain by puberty. Myoclonic seizures are another case in point, forming part of quite different syndromes. They may be part of a benign and transient disorder, or a feature of progressive neurodegenerative disease.

Sleep disorders confused with each other

There is a tendency to confuse different types of acutely disturbed behaviour in sleep and to call any such disturbance a 'nightmare'. Sometimes textbooks expressly equate nightmares with night terrors or one is said to be a variety of the other. In fact, fundamental differences exist between nightmares and night terrors (and other so-called 'arousal disorders') and in recent times accounts of yet other types of sleep disorder characterised by dramatic behaviour have been published.

Nightmares are frightening dreams occurring in REM sleep during the later part of the night when that stage of sleep is most abundant. In a nightmare the child will wake and give an account of a frightening dream experience. The term 'arousal disorder' (Broughton, 1968) covers a spectrum of behaviour types which have in common that they are sudden partial wakenings towards the end of a period of deep (stages 3 and 4) non-REM sleep, at the point of transition from this stage to a lighter level of sleep. As deep non-REM sleep occurs mainly in the first part of sleep, primarily in the first sleep cycle, these disorders happen within the first three hours or so after the child falls asleep. During the episode the child remains deeply asleep, and therefore inaccessible, but also exhibits behaviour more reminiscent of the awake state. Afterwards there is little or no recall of the events.

There is a spectrum of clinical accompaniments to these arousals, from mild changes to very dramatic behaviour, and the nature of the episode varies somewhat with the age of the child. There may be no more than turning over or perhaps brief moaning; or motor activity and vocalisations may be more marked, with crying and thrashing about in the young child or, at an older age, sitting up, staring, shuffling about in bed, with incoherent muttering or more formed sentences ('sleep talking') and apparent confusion. Alternatively, the child may get out of bed and walk about in a semi-purposeful way ('sleep walking') and indulge in conversations. There may be signs of agitation sometimes amounting to a state of terror, with crying out, intense autonomic overactivity (much greater than in nightmares) and avoidance behaviour ('night or sleep terrors'). Perhaps older children and adolescents in particular may rush about in a terrified state or even dash out of the house. Guilleminault & Silvestri (1982) provide some striking examples of these types of disturbed behaviour in adolescents (and adults) during non-epileptic parasomnias.

Therefore there are important differences between nightmares and arousal disorders regarding timing, clinical features (including the child's awareness and recall) and basic physiology (Kales *et al*, 1987). The form of management required is also different in these two types of condition. In general it is appropriate and possible to comfort a child who has woken from a nightmare. Attempts to wake a child in a night terror or agitated sleep-walking episode may cause him/her confusion and distress; calm

reassurance when the acute episode is over and making the environment safe to prevent accidental injury are more useful measures.

In recent years another type of sometimes dramatic behaviour disorder during sleep has been described. This is the 'REM sleep behaviour disorder' (Mahowald & Schenck, 1989), in which the person is able to act out his/her dreams. This is possible because of a disturbance of REM sleep mechanisms, whereby skeletal muscular tone is preserved during REM sleep instead of being reduced as is normally the case. If the content of the dream is violent patients may injure themselves or others, perhaps seriously. Clonazepam is said to be an effective treatment. At present it seems that this particular sleep disorder is likely to be encountered in children only very rarely. Most reports have been concerned with middle-aged or elderly males, but there have been some reports (Barros-Ferreira *et al*, 1975; Schenck *et al*, 1986; Herman *et al*, 1989) suggesting the same condition in children between the ages of two years (this child was initially thought to have epilepsy) and ten years. These children usually show signs of a brain-stem lesion, presumably affecting the brain-stem nuclei which control REM sleep. Many of the adult patients in whom this disorder has been described had previously been diagnosed as having nightmares (Mahowald & Schenck, 1989).

Sleep disorders misdiagnosed as epilepsy

There seems to have been a tendency, perhaps on the part of psychiatrists in particular, to consider epilepsy a likely explanation of apparently unprovoked episodes of violent or otherwise antisocial behaviour. Several reviews (e.g. Treiman & Delgado-Escueta, 1983) have demonstrated that this type of behaviour in the daytime is very unlikely to be epileptic in origin. The same appears to be true of such behaviour during the night. Other conditions, especially arousal disorders, are a more likely explanation and must be distinguished from epilepsy in spite of sometimes sharing such features as abrupt onset, confusion, amnesia and injury.

There have been various reports suggesting that violence, including homicidal behaviour, can occur as part of a sleep disorder and, in some, consideration of the underlying cause has mistakenly emphasised the possibility of epilepsy. Oswald & Evans (1985) described three cases, including that of a 14-year-old boy, where serious violence had been perpetrated on a sleeping partner or relative during what was eventually considered to be sleep walking. These authors reviewed other reports of apparently similar cases and referred to the outmoded notion that what are now called arousal disorders are a form of epilepsy. Scott (1988) described another such case in which the diagnosis of epilepsy was mistakenly entertained repeatedly and the patient advised not to pursue his occupation as an ambulance driver. As pointed out by Morehouse (1989), the REM sleep behaviour disorder should also be considered as an alternative diagnosis in cases exhibiting violence during sleep. In a series of 11 carefully investigated cases of violence in sleep, the youngest being 14 years old, seven were diagnosed as arousal disorder and the rest as single cases of REM sleep behaviour disorder, sleep apnoea, idiopathic CNS hypersomnia, and alcohol-related sleep disorder. Epilepsy did not feature at all (Broughton, personal communication).

Behaviour during sleep does not have to be dramatic for epilepsy to be considered. In the epilepsy and sleep disorders clinics at the Park Hospital for Children in Oxford, lesser forms of arousal disorder and even head rolling have been encountered in which epilepsy had been diagnosed or considered likely.

Case 1

JL, a boy with the Pierre Robin syndrome, was eight years old when he began to have night-time episodes lasting between 10 seconds and 10 minutes of shouting, incoherent speech, vigorous random leg movements and a frightened facial expression. These episodes occurred two to three times a month. Less frequently he would walk downstairs from his bedroom and carry out repetitive acts, such as walking around the table, in an unresponsive state. At the age of 11, at a time when his behaviour was generally disturbed, he was said to have had episodes during the day of diminished responsiveness and facial grimacing lasting up to three hours. An EEG was reported as "consistent with multifocal cortical epilepsy" and antiepileptic drugs were introduced. JL's daytime episodes subsided over the next 12 months but he was referred to the Park Hospital epilepsy service at the age of 13 because of persistence of his night-time attacks. Overnight video, sleep studies and oximetry showed repeated nocturnal episodes arising from stage 4 non-REM sleep with no evidence of seizure discharge or sleep apnoea. Review of the original EEG showed that it contained no convincing abnormality. The night-time episodes were diagnosed as an arousal disorder. On further inquiry the earlier day-time episodes seemed likely to have been a behaviour problem. Antiepileptic drugs were withdrawn and the need for psychological help was emphasised. Since then JL's attacks have become progressively less frequent.

Case 2

SO was said to have always been a light sleeper and to have taken a long time to get to sleep. As a young child she would chatter to herself until 1.00 a.m. or roll from side to side for long periods, sometimes banging her head on the pillow. This behaviour was still evident by the age of three and gradually worsened over the next few years. The rocking

was sometimes accompanied by growling and occurred repeatedly during the night as well as occasionally during the day, associated with hand flapping. SO was sleepy during the day and both her learning and her general behaviour were a problem, particularly at school. She had had a tonic–clonic seizure associated with fever at the age of two years, which was said to have been followed by a right Todd's paresis, and at about the same age she underwent bilateral reimplantation of her ureters because of congenital renal abnormalities. When she was seven, SO was referred for EEG to explore the possibility that her persistent night-time attacks were epileptic in nature. Combined video and cassette EEG recordings overnight showed that the rolling episodes occurred as SO woke repeatedly through the night. Each episode lasted about 10 seconds and occurred in quick succession for a total duration of between 10 and 30 minutes until she settled back to sleep again. No convincing seizure discharge was seen at any stage. Sleep structure was disrupted, with short overall sleep time. It was considered that SO was suffering from unusually persistent head rocking and banging associated with disturbed daytime behaviour and that her disrupted sleep was the cause of the daytime sleepiness.

There are other more basic sleep phenomena that can raise the possibility of nocturnal seizures. An obvious example is enuresis, which is common and not usually of epileptic origin. Nevertheless, this possibility needs to be considered if there is any suggestion of convulsions during the night or if there is no response to conventional treatment for enuresis. Enuresis is one of several possible manifestations of obstructive sleep apnoea in children, which tend to be more varied than in adults where loud snoring is the predominant sign. Other nocturnal symptoms in this condition that might be construed as evidence of seizure activity are difficulty in breathing, restless sleep and intense sweating (*Lancet*, 1989).

There are two other sleep disorders which, although apparently uncommon, have clinical features which could be confused with complex partial seizures of temporal lobe origin or non-convulsive status epilepticus. Alternatively, they may be misconstrued as a primarily psychiatric disorder. The first is the automatic behaviour syndrome (Guilleminault *et al*, 1975*a*). This is said to be characterised by a continuation of activities in an altered state of consciousness, sometimes with hallucinations, followed by complete amnesia. Each episode lasts a few seconds to several hours, during which time repetitive 'microsleeps' occur. During an episode the patient appears blank and out of touch with his/her surroundings. There may be drooping or fluttering of the eyelids and vocalisations of varying length and meaning. The attacks tend to occur in circumstances that predispose to drowsiness. Sense of time is lost and the patient's activities can be a source of embarrassment, perplexity or danger. This condition has been associated mainly with the narcolepsy syndrome but also with other causes of daytime sleepiness, including sleep apnoea. Reported series include children (Guilleminault *et al*, 1975*b*). Some of the ways in which automatic behaviour of this type is different from temporal lobe complex partial seizures (of usual duration) are absence of aura, wider range of duration, alerting effect of stimulation and lack of both affective change and post-ictal symptoms (Pedley, 1983). However, the distinction between the automatic behaviour syndrome and non-convulsive status epilepticus, whether absence status or complex partial status (Stores, 1986), may be difficult from the clinical features alone, but physiological recording during the episodes would demonstrate microsleeps in the former condition and seizure discharge in the latter.

The same type of distinction might need to be made between 'idiopathic CNS hypersomnia with sleep drunkenness' (Roth *et al*, 1972) and non-convulsive status epilepticus. The age of onset in idiopathic CNS hypersomnia is said to be from 15 years onwards. Salient features are great difficulty getting up in the morning, in spite of an apparently sound night's sleep, and inability to become fully alert for a long time after waking. Considerable difficulties arise at school, at work, or elsewhere, because of extreme tiredness, disorientation, incoordination and often automatic behaviour, as previously described. Unfortunately the prospects for improvement are poor.

Epilepsy misdiagnosed as sleep disorder

The opposite form of misdiagnosis is failure to recognise nocturnal epileptic attacks as epilepsy at all. This is particularly liable to happen in the case of non-convulsive seizures, which form a high proportion of seizure types.

Interictal focal epileptic discharges and partial seizures are provoked by both REM and non-REM sleep to the extent that some patients have exclusively nocturnal attacks. Seizures of a complex partial type are those most likely to be confused with non-epileptic parasomnias, especially arousal disorders, because their clinical features, especially automatic behaviour, overlap. Most complex partial seizures arise from a temporal source, therefore it is this form of seizure that numerically is most likely to be mistaken for a non-epileptic parasomnia. However, perhaps as many as 30% of complex partial seizures have an extra-temporal origin and of these possibly a third arise from a seizure source in the frontal area, mainly orbito-frontal or mesial-frontal (Williamson & Spencer, 1986).

Complex partial seizures of frontal origin have only relatively recently been described (especially in

children), with the result that they are generally unknown to clinicians. Mainly because of their sometimes bizarre nature, including complicated automatisms and vocalisations, those occurring in the daytime are often construed as 'hysterical' or as evidence of some other form of psychiatric disorder. This appears to be true for both adults and children. When attacks of this sort occur in children at night (which many do), they may well be diagnosed as 'nightmares' (Stores & Zaiwalla, 1989). This mistake is difficult to justify, not only because of the nature of the automatisms in these attacks and the lack of any evidence of dreaming, but also because of their very frequent occurrence (perhaps many each night) and their abrupt onset and termination, sometimes after only a matter of several seconds. Conventional EEG recordings can be of limited value in this form of epilepsy, although there is some suggestion that ictal recordings which emphasise frontal and prefrontal electrode placements may provide useful evidence of a frontal seizure source (Stores & Zaiwalla, 1989).

Case 3

From the age of three years CJ had episodes of inappropriate behaviour during the day which were initially diagnosed as hysteria. The diagnosis was changed to epilepsy when she started to report abdominal sensations at the start of some of these episodes. Antiepileptic drug treatment was associated with a reduction of the daytime episodes but at the age of nine years CJ began to have frequent night-time attacks consisting of thrashing movements of her limbs, grunting, sitting up and rocking from side to side for up to about 30 seconds. When no definite EEG changes were recorded during two of these night-time episodes the diagnosis was revised again to 'pseudoseizures' and psychiatric treatment was provided, including the use of amitriptyline. The attacks increased in frequency. Reassessment at the Park Hospital included overnight video and cassette EEG monitoring during many of these attacks which occurred throughout the night. The episodes were brief and mainly consisted of complex motor automatism including repeated flexion–extension or scissor movements of the legs. At the onset of these attacks a left pre-frontal sharp-wave discharge was seen repeatedly before more widespread EEG changes occurred. The attacks were diagnosed as complex partial seizures of left frontal origin. No evidence of a structural abnormality was found.

Crowell & Anders (1985) described a ten-year-old child diagnosed as having 'hypnogenic paroxysmal dystonia' which they felt should be included in the differential diagnosis of epileptic attacks and pseudoseizures. The originators of this term (Lugaresi *et al*, 1986) have more recently preferred the term 'nocturnal paroxysmal dystonia' and have documented two forms: short and long-lasting. Attacks in the shorter form have many features in common with complex partial seizures of frontal origin and may well be further examples of that form of epilepsy (Tinuper *et al*, 1990); the nature of the longer episodes remains obscure.

Another condition characterised by disturbed behaviour during the night and possibly epileptic in nature is 'episodic nocturnal wanderings' as described originally by Pedley & Guilleminault (1977) and more recently by Maselli *et al* (1988). Onset is usually in the late teens. Episodes consist of frequent stereotyped attacks of screaming, walking about and complex automatisms, including violent behaviour, during sleep. These episodes are more complicated, violent and longer lasting (up to several minutes) than frontal complex partial seizures or nocturnal paroxysmal dystonia. Their variable time of occurrence during the night does not particularly suggest an arousal disorder from deep non-REM sleep. Some of those with this condition were said to have 'epileptiform' activity in their inter-attack EEGs, but seizure discharge was not recorded at the time of their attacks. A good response to antiepileptic drugs (mainly carbamazepine) was usually obtained. Whether this condition is distinct from the epileptic and non-epileptic disorders discussed earlier remains uncertain. Oswald (1989) comes down firmly on the side of it being night terrors with sleep walking.

Finally, as suggested earlier, the various types of non-convulsive status epilepticus can be confused with sleep disorder. The clinical manifestations of this form of epilepsy are varied. Textbook descriptions emphasise the more dramatic forms in which the patient (adult or child) is stuporose or 'pseudo-demented', with clinical features somewhat similar to the long daytime episodes of sleep disorder described previously, sometimes including ataxia. But this obvious impairment of performance and behaviour is only one end of a spectrum. At the other extreme the clinical accompaniments of even gross continuous EEG abnormalities can be very subtle (Stores, 1986). Towards this less obvious extreme a salient feature can be that of a mild degree of sleepiness as illustrated by some of a series of children with non-convulsive status epilepticus seen at the Park Hospital for Children.

Case 4

From the age of seven JH began to fall asleep repeatedly during the day in spite of apparently sleeping well at night. At various times every day he went to sleep and was confused when he eventually awoke about three to four hours later. On other occasions he would simply yawn, stretch and complain of feeling tired. JH had a past history of a febrile convulsion at six months of age and infrequent tonic–clonic seizures since he was 18 months old.

Medication at the onset of his daytime sleepiness was carbamazepine and sodium valproate in modest dosage. Investigation at the Park Hospital, including cassette monitoring and combined EEG/video recordings, showed only occasional brief bursts of generalised polyspike and wave activity when JH was alert, but continuous generalised spike wave discharge during his tired and sleepy periods. These were diagnosed as episodes of generalised non-convulsive status epilepticus. Withdrawal of carbamazepine and an increase in the dosage of sodium valproate abolished the episodes completely.

Coexistence of epilepsy and sleep disorder

Night-time episodes of disturbed behaviour need not be exclusively either sleep disorder or epilepsy. Epilepsy is common – its prevalence rate in children has been estimated to be at least 1% (Rose *et al*, 1973). Sleep disorders are much more common than epilepsy (Simonds & Parraga, 1982). Epilepsy and sleep disorders therefore may well coexist in some children. As children with epilepsy have high rates of emotional disturbance (Stores, 1981), sleep problems associated with emotional upset are likely to be over-represented in this group. In fact, information on sleep problems in children with epilepsy is very limited, but the findings of Zaiwalla (1989) suggested a generally higher rate of sleep problems in such children compared with controls, and Tassinari *et al* (1972) have described night terrors in children with epilepsy.

Clinical assessment and physiological monitoring in sleep disorders

It is generally agreed that the basis of accurate diagnosis of episodic disturbances of behaviour is careful description of the subjective and objective nature of the attacks (including the precise sequence of events), the circumstances in which they occur, as well as their timing. The range of possible diagnoses, and the characteristic features of each condition, must be known.

However, detailed descriptions can be difficult to achieve, certainly in the case of night-time attacks in children. Encouraging parents to describe their observation in as systematic and as detailed a fashion as possible (perhaps with the aid of a diary) can improve the quality of reporting, and further information from siblings can be very helpful, especially if they share a bedroom with the child in question. However, parents or even siblings are unlikely to witness the onset of attacks (of particular importance in the case of seizures) and may only report the more dramatic features. As a result cases are encountered where a confident clinical diagnosis cannot be made because of inadequate information. It is in such a situation that the risk of a mistaken diagnosis such as 'nightmares' or 'epilepsy' is greatest.

In these circumstances overnight video recordings can be very instructive about the nature of the attacks and their timing. Discrepancies may well be observed between the account given by relatives (or even hospital night staff) and the features revealed by analysis of the video tape. At present this facility is very little used but there is no reason why it should not be employed more widely, including recordings at home with parents' own equipment or a video system loaned by the hospital's clinical neurophysiology department. If this is not feasible then such recordings should usually be possible in any child psychiatry or paediatric in-patient unit, residential school or other setting where the child sleeps. Diagnostic accuracy can be further increased by simultaneously recording the EEG and, if necessary, other sleep parameters for conventional sleep analysis (Rechtschaffen & Kales, 1968). As illustrated in the above case reports, EEG and sleep recordings can now be conveniently achieved at home, or wherever else the child is sleeping, by means of a portable cassette system (Stores & Bergel, 1989).

Conclusions

Nocturnal disturbances of behaviour are common, especially in children. The various conditions in which such behaviour can occur seem to be a source of diagnostic confusion, perhaps especially when the possibility of epilepsy arises. The classification schemes now available for both sleep disorders and the epilepsies, as well as recent accounts of previously unrecognised disorders of both types, can serve as a useful aid to accurate clinical diagnosis.

Where diagnostic difficulty remains, especially because of inadequate clinical information, video and physiological monitoring can be helpful. Such investigations are now possible, even as out-patient procedures, by means of recently developed recording systems. These facilities are not often provided by departments of clinical neurophysiology at the present time, not because highly specialised expertise is required, but principally because of limited demand from clinicians. An all-round knowledge of the different varieties of night-time disturbance of behaviour is needed, combined with an increased awareness of the ways in which accurate diagnoses can be made.

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