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Review Article

Childhood Hyperactivity

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Severe degrees of inattentive and restless behaviour in childhood are a risk factor for later psychological disorders. They have many causes, but a pattern of severe and pervasive hyperactivity with poor concentration in the absence of affective or psychotic disorders should be recognised as a hyperkinetic syndrome. The syndrome is often associated with developmental delays in abilities such as language and motor control. Powerful short-term treatments are available, but long-term ways of promoting normal personality development need more research.

Hyperactivity means an enduring style of behaving in a restless, inattentive, and disorganised fashion. These are common complaints in children, and they have many different causes. When the problems are severe, they can handicap a child's learning and relationships with family and peers, and make him or her vulnerable to further psychiatric disability.

There has been a confusing debate about the existence of a psychiatric condition of hyperactivity. It has been argued that hyperactive behaviours are non-specific features of any psychopathology (e.g. Sandberg, 1981; Shaffer, 1980). Other authorities (e.g. Aman, 1984; Wender, 1971) recognise them as signs of a specific disorder. Most diagnostic schemes recognise a distinct category. DSM–III (American Psychiatric Association, 1980) describes a condition of Attention Deficit Disorder that may be with or without hyperactivity. The former (ADDH) is defined by the presence of specified numbers of symptoms of developmentally excessive activity, attention deficit, and impulsiveness, especially at school, and affects about 3% of schoolchildren. ICD-9 (World Health Organisation, 1980) is less precise, but describes a condition of 'hyperkinetic syndrome' that is defined by short attention span and (in younger children) by 'extreme' overactivity not attributable to any other condition (Rutter *et al*, 1975). In practice, this has led to hyperkinesis being a rare diagnosis in the UK.

Historical sketch

Severe overactivity has been recognised as a symptom of disorder in handicapped children for at least a century (Ireland, 1877). A lecture series by the pioneer of paediatrics, Frederic Still, promoted the wider idea that organic disorder of the brain was a major cause of behavioural problems in childhood, through the induction of "defects of moral control" (Still, 1902). His description of affected children was strikingly similar to modern definitions of hyperactivity: it included "an abnormal incapacity for sustained attention", restlessness, fidgetiness, violent outbursts, destructiveness, non-compliance, choreiform movements, and minor congenital anomalies. Still's work helped to create an intellectual climate in which hyperkinesis and brain dysfunction became almost synonymous.

The next step in recognition came with the encephalitis pandemics in the wake of the First World War. Schachar (1986) studied contemporary reports, and commented that severe behavioural sequelae of encephalitis were uncommon and diverse. The cases did, however, emphasise that brain dysfunction is a possible cause of overactive and disinhibited behaviour. In this tradition, Kahn & Cohen (1934) described three cases of "organic drivenness"; their paper was influential in arguing that hyperactivity was the central behavioural abnormality, and that it resulted from disorganisation at the level of the brain stem.

The idea of hyperactivity soon became more widely applied. It was accelerated by the chance discovery of amphetamine's powerful action in reducing hyperactive and disruptive behaviour (Bradley, 1937). By 1938, Levin was able to identify more than two hundred restless children, compare them with normally active controls, and conclude that cerebral lesions caused severe restlessness (while milder degrees resulted from upbringing problems). Strauss's writings in the 1940s and 1950s broadened the idea further (Strauss & Lehtinen, 1947), to the point where hyperactivity—in the absence of a family

history of subnormality—became sufficient evidence for a diagnosis of brain damage.

Laufer *et al* (1957) described "hyperkinetic impulse disorder" in children with emotional and conduct problems, most of whom did not have any overt evidence of brain damage. They reported a low threshold for induction of muscle jerks by metrazol and stroboscopic light; amphetamine restored the threshold to normal. The study was intended as a corrective to psychoanalytical views that strongly emphasised parents' contributions to children's problems. It was followed by an explosion of biological and pharmacological research, and by a great increase in the diagnosis of hyperactivity and the prescription of stimulants by American paediatricians.

A task force produced an influential document that regarded hyperactivity as a synonym for 'minimal brain dysfunction', and included an excessively wide range of children's psychological difficulties embracing most problems presented for psychiatric help (Clements, 1966). Hyperactivity, or ADDH, has remained the commonest psychiatric diagnosis for North American children. It has generated much good research, most notably into the cognitive deficits of affected children (reviewed by Douglas, 1983) and the nature of the action of stimulant drugs (reviewed by Cantwell & Carlson, 1978, and Rapoport, 1983).

In sharp contrast, British child psychiatry and paediatrics largely avoided the efflorescence of hyperactivity. Widely quoted papers by Ounsted (1955) and Ingram (1956) described series of severely disturbed children, all of whom were drawn from the authors' patients with overt neurological illnesses. The Isle of Wight surveys (Rutter *et al*, 1970) emphasised the infrequency of hyperkinetic syndrome in a whole population of nine year-olds. The diagnosis is still made sparingly, and around half of those receiving it are intellectually retarded or show signs of neurological illness (Thorley, 1984*a*). This narrow concept suffers, paradoxically, from vagueness of criteria, and has led to much less research than has stemmed from ADDH.

The attitudes of European nations have generally lain between these extremes, and rates of stimulant prescription vary enormously (World Health Organisation, 1985). Several different concepts of hyperactivity have evolved in different traditions, and taxonomic research is particularly necessary.

For clarity, I shall adopt arbitrary definitions to maintain some separation between concepts. 'Overactivity' will mean simply a quantitative excess of movement, without any necessary implication of inattention or disorganisation. 'Hyperactivity', as above, will refer to an excessively high level of inattentive and restless behaviour. 'ADDH' will refer to the DSM–III category and also to other, similar diagnoses used by North American, Australasian, and other workers; it is a relatively broad group, not only because there are few exclusion criteria, but also because impulsiveness and school misbehaviour can contribute to the diagnosis. 'Hyperkinetic syndrome' will be taken to mean the narrower style of diagnosis, often applied to severely disturbed children; criteria for a useful definition will be suggested below.

Taxonomy

Statistical studies make it clear that several symptoms of hyperactivity covary. Short attention span, distractibility, restlessness, constant fidgeting, and off-task activity are the most constant of these. They form a separate component in many independent factor-analytic studies of rating scales (e.g. Trites *et al*, 1982; Taylor & Sandberg, 1984). This 'hyperactivity factor' is distinct from one of defiantly or aggressively antisocial conduct. Nevertheless, some behaviours—such as disruptiveness, excitability, or impulsiveness—can appear as a part of either factor, and are probably non-specific.

The emergence of a factor of hyperactivity is only one step on the road to a rational classification. Most of the children referred for help because of their disruptive behaviour show both hyperactivity and antisocial conduct. Most hyperactive children are defiant or aggressive too; most antisocial children are somewhat hyperactive (Cantwell, 1980; Stewart *et al*, 1981; Taylor, 1986*b*). It is therefore crucial to determine whether the idea of hyperactivity has predictive and discriminative validity: does it predict anything that is different from conduct disorder? Shaffer & Greenhill (1979) demonstrated how resolutely this question had been ignored by investigators, and how little reason there was to base research or clinical practice on a widespread diagnosis such as ADDH. Researchers must therefore identify pure cases of hyperactivity that do not show conduct disorder, and pure cases of conduct disorder for comparison; otherwise, they must separately associate the two dimensions with other clinical features.

Distinction between hyperactivity and conduct disorder

Direct observations of children in playrooms or laboratories have provisionally confirmed that inattentive-restless behaviour (judged from case histories or from research interview techniques) predicts high observed levels of off-task activity, while defiance or aggression does not (Milich *et al*,

1982; Taylor *et al*, 1986*a*). Furthermore, the same groups of investigators have found that hyperactivity at home or at school correlates with the presence of developmental delays (such as motor clumsiness and low IQ) rather than with psychosocial factors. Conduct disorder, by contrast, is linked closely to indices of unsatisfactory family life, such as discord between family members, low levels of expressed warmth, and ineffective styles of coping (Loney *et al*, 1978; Taylor *et al*, 1986*a*). A comparison of pure hyperactivity with pure conduct disorder by Stewart *et al* (1981) also suggested that hyperactive children were symptomatically distinct; on the other hand, the two groups had equally high rates of antisocial relatives (Stewart *et al*, 1980).

These studies were based on clinical groups; McGee *et al* (1984) reported an epidemiologically based sample. Small numbers and missing data made conclusions uncertain, but the children with the single problem of conduct disorder had higher performance IQs than those with pervasive hyperactivity solely: socioeconomic status, perinatal history, and family relationships did not distinguish between the groups.

Existing studies of the taxonomic questions are too few to allow sharp and certain definitions. They suggest (but not conclusively) a valid separation of patterns of disorder. It is therefore worth considering the lessons from some less powerful studies, that have compared hyperactive children with other kinds of disorder, but have not included the requirement that the contrast group should be quite free of hyperactive behaviour.

Koriath *et al* (1985) have described a notable lack of differentiation between children referred to a paediatric psychiatry service in North Carolina who received different DSM–III diagnoses. Those with ADDH alone, ADDH plus other diagnoses, and other psychiatric diagnoses not combined with ADDH, were not even distinct in terms of ratings of symptoms—let alone in demographic factors or psychological test scores. Sandberg *et al* (1978) also found that different levels of severity of hyperactivity carried few implications for aetiology within a group of boys referred to a psychiatric clinic, when 'hyperactivity' was measured by a parental questionnaire, or a teacher rating scale, or by a single period of direct observation at a clinic. Each of these measures was imperfect; when they were combined, a small group of children with pervasive hyperactivity across all measures could be separated from the others, and proved to have an earlier onset, more signs of motor clumsiness, and poorer performance on a psychometric test. Similarly, Schachar *et al* (1981) reanalysed the Isle of Wight survey to find that pervasively hyperactive children had poorer performance on a psychometric test, tended to come from lower social classes, and had a worse outcome than children with other kinds of disorder.

The distinction between pervasive and situational hyperactivity may not be very sharp, and some studies find the two groups to be similar (Firestone & Martin, 1979). The general point, however, is that hyperactivity can be validly distinguished from conduct disorder, but that good measures or severe degrees need to be taken if the separation is to be found.

Status of mixed cases

If the above argument is accepted, so that hyperactivity and conduct disorder are seen as separable patterns that overlap greatly in practice, what is the status of mixed cases? If they were clearly to resemble 'pure' conduct disorder or 'pure' hyperactivity, then their position in classification would be clear.

Stewart *et al* (1981) made just such a comparison on the basis of clinical features: children with unsocialised aggression and hyperactivity (identified mainly by parental accounts) resembled those with only unsocialised aggression more than those with only hyperactivity. On the other hand, August & Stewart (1982) presented another analysis of a closely similar series of children. When hyperactivity had to have been noted in several situations, there was little difference between the children with this problem only and those who combined it with unsocialised aggression.

McGee *et al*'s (1984) epidemiological study seemed to show that the mixed group were particularly vulnerable. Their reading was worse than that of either pure group, and their short-term outcome was worse. Such a finding is in keeping with a dimensional approach to classification: children with both problems should be predicted to have the associations of both.

A separate class of hyperkinetic syndrome?

Hyperkinesis needs to be separated not only from conduct disorder but also from emotional disorders and specific disabilities of learning. Applications of cluster analysis to simple parent questionnaire ratings have given conflicting results about the presence of a discrete group of hyperactive children (Taylor *et al*, 1986*b*). Some workers find one, some do not. Halo effects and reliance on a single rater are serious problems in this research. A cluster analytical study reported by Taylor *et al* (1986*b*) was based on reliable, independent measures from several sources. The most robust feature of clustering procedures was

the emergence of a small group of pervasively hyperactive and inattentive children with few emotional symptoms. This group also showed an early onset, a high rate of delays in cognitive development, and motor clumsiness. In addition, they had a much more marked response to a stimulant drug (methylphenidate).

Definition

Current evidence favours use of a rather restricted diagnostic concept of hyperkinetic syndrome, rather than the ADDH definition. Symptoms of overactivity alone are not sufficient for the diagnosis; it should be based on:

(a) A pattern of markedly inattentive, restless behaviour (not just antisocial, impulsive, or disruptive acts) that is excessive for the child's age and IQ, and a handicap to development
(b) presence of this pattern in two or more situations, such as home, school, and clinic
(c) evidence of inattention, restlessness, or social disinhibition, from direct observation or testing by the diagnostician (i.e. not solely by unconfirmed reports from a child's caretakers)
(d) absence of childhood autism, other pervasive developmental disorders, or affective disorders (including depression, anxiety states, and mania)
(e) onset before the age of six years and duration of at least six months.

Such a diagnosis is useful, though not very common (Taylor, 1986*a*). It should not be missed simply because an affected child has developed antisocial conduct as well.

Hyperactivity in special subject groups

The discussion of classification above has been based on studies of normally intelligent children in the first few years of their schooling. The behaviour problems of preschool children do not yet have a stable classification (Jablensky *et al*, 1983). In one investigation, pervasive hyperactivity in children at a day nursery predicted psychological disorder later (Campbell *et al*, 1977); in another, even pervasive hyperactivity was not a predictive variable among nursery children (Cohen & Minde, 1983). The behaviour matters, as will be shown later, but its classification is obscure, and clinicians should be correspondingly slow to diagnose. Overactivity is also common among intellectually retarded children, but its causes are not yet clarified. Affective disorder, autism, and atypical pervasive developmental disorder are all important causes of overactivity in this group. I have argued elsewhere (Taylor, 1986*a*) that some individuals should be regarded as showing a distinct hyperkinetic syndrome, but more research is required.

Prevalence

No secure prevalence rate can be given, because of disputes about what constitutes a case. Hyperactivity is continuously distributed in the population, with smaller numbers at successively higher degrees (Taylor, 1986*a*). No validated cut-off yet exists, so no prevalence estimate has a scientific basis.

The numbers of schoolchildren who are in practice diagnosed vary wildly: a little over 1% in two US surveys (Lambert *et al*, 1978; Bosco & Robin, 1980), but only 0.1% in a London borough (data from the Camberwell Register)—half of the latter being intellectually retarded. By contrast, the rates derived from questionnaire surveys of teachers' ratings are much the same in the UK as in the USA and New Zealand, and range from 5% to 20% (Taylor & Sandberg, 1984). The low diagnostic rates in the UK are likely to be due to the diagnostic practice rather than the nature of the children (Prendergast *et al*, unpublished). Chinese populations are often said to be more docile and attentive, but a survey from Beijing, based on a combination of teacher ratings and medical diagnosis of ADDH, gave comparatively high rates (3–7% in different areas) (Shen *et al*, 1985). Hyperactivity, in some sense, evidently exists in many cultures; it remains to be seen whether it always takes the same form.

The prevalence of ADDH in boys is higher than in girls in the ratio of about 4:1 in American clinics (Ross & Ross, 1982). The ratio was similar for the narrower hyperkinetic syndrome at a London clinic, but fell to 1.6:1 for children with mental retardation (Taylor, 1986*a*). Girls with the diagnosis of hyperkinetic syndrome, or with the symptom of 'gross overactivity', were more likely than affected boys to show delays in cognitive development and neurological disorders such as epilepsy (Taylor, 1986*a*). The reason for the greater vulnerability of boys has not yet been explained. Several theories implicate damaged brain function. The greater vulnerability of male brains may well be a part of the explanation, but has not been shown to account for the whole of the enhanced risk. Adults are often more tolerant of hyperactivity in girls than in boys, at least before school age (Battle & Lacey, 1957).

Epidemiological studies in different cultures are needed to cast light on what is central to and

universal about the conditions and what is peripheral and culturally determined.

Aetiology

In spite of much research, the causes of hyperkinesis remain uncertain. Most studies have compared children referred to clinics, because of mixed behaviour problems including hyperactivity, with controls drawn from the normal population. When positive findings appear in such a design, it is hard to know whether they are associated with hyperactivity, with behaviour problems generally, with the learning disorders that are often associated, or with the factors that lead to referral to clinics.

Brain damage

Brain disease with localising neurological signs is uncommon in children with ADDH, and is specifically excluded from most research series. The narrower hyperkinetic syndrome, by contrast, is often diagnosed in children with damaged brains. Table I summarises the findings in those investigations that have used an ICD-9 style of diagnosis and reported the range of diagnoses in various samples of children. Brain damage gives rise to a high rate of virtually all the psychiatric syndromes of childhood, rather than to any pathognomonic syndrome (Rutter *et al*, 1970). Accordingly, the best index for comparing groups is not the numbers with the diagnosis of hyperkinesis, but the rate of hyperkinesis as a percentage of all diagnoses made (Table I).

The relatively high rate in neurologically handicapped groups implies that there may be a specific vulnerability to hyperkinesis in brain-damaged children. This conclusion is not secure, for the diagnosis could in part be determined by judgements about neurological status. Explicit and independent criteria for case definition need to be developed. If there is a specific vulnerability, we do not know whether it resides in the brain damage itself or in the generalised intellectual retardation which can result. High rates of structural brain damage were reported by Reid (1980). Jenkins & Stable (1971) matched severely hyperkinetic cases (from a case register of intellectually subnormal people) with subnormal but not hyperkinetic controls: they found higher rates of cerebral palsy and other evidence of structural lesions in the hyperkinetic patients. However, the hyperkinetic patients were also more retarded on a global measure of development. Thorley (1984*a*), using the case records of severely hyperkinetic children, matched psychiatric controls individually for IQ level: no significant difference in rates of brain damage then appeared.

Severe hyperkinesis may have a specific neuropsychiatric predisposition; this should be investigated further. However, there is no reason to suppose that mild hyperactivity is therefore caused by mild brain dysfunction.

Perinatal trauma to the brain

This is not usually the cause of hyperactive behaviour. Case-control studies of children showing high levels of hyperactivity have usually shown little or no increase in rates of perinatal injury, retrospectively assessed (e.g. Minde *et al*, 1968; Gillberg *et al*, 1983). Cohort studies of children of low birth weight (Neligan *et al*, 1976), or of children with complicated deliveries (Nichols & Chen, 1981), make it clear that

TABLE I

Prevalence of clinically diagnosed hyperkinetic syndrome (ICD–9) as a percentage of children with psychiatric diagnoses

Study	*Type of study*	*Patient group*		
		Neurologically normal children: %	*Neurological disorders or epilepsy: %*	*Mental subnormality: %*
Rutter *et al* (1970)	Epidemiological survey	~1	12	26[1]
Corbett (1979)	Case register	—	—	8
Reid (1980)	Clinic outpatients	—	—	15
Thorley (1984*a*)	Clinical records	0.9	—	2
Gillberg *et al* (1986)	Epidemiological survey	—	—	10

1. Severely handicapped children, not attending school.

hyperactivity is not particularly common in the later lives of such children. There is a small increase—as there is for other kinds of behaviour problem—but even this increase is partly attributable to coexisting family problems. Indeed, the Isle of Kauai study (Werner & Smith, 1977) suggested that perinatal damage only increased vulnerability in children with less advantaged backgrounds.

Minimal brain dysfunction

Minimal brain dysfunction has been intensively sought, so far largely in vain. Extensive reviews have concluded that there are few unequivocally positive findings of biological abnormalities in hyperactive children (Rie & Rie, 1980; Rutter, 1983*b*). When abnormalities have been found, their meaning is usually unclear. 'Soft' neurological signs (i.e. clumsiness and lack of coordination) are commoner in markedly hyperactive children than in other sorts of psychological disorder (Werry *et al*, 1972; Sandberg *et al*, 1978; Taylor *et al*, 1986*b*). However, they are not yet valid as a sign of cerebral or cerebellar pathology because of uncertainty about what causes them (Taylor, 1983*a*).

Neurophysiological unresponsiveness

Neurophysiological unresponsiveness to stimuli is another replicated finding. Hyperactive children, by comparison with controls, probably show normal resting levels of autonomic nervous system activity. However, peripheral autonomic responses and central averaged evoked EEG responses both suggest that a new or important stimulus produces a smaller change in hyperactive children (Taylor, 1985*a*). This lack of reaction may not be specific to hyperactive children: diminished physiological responsiveness also characterises children with learning disorders (e.g. Maxwell *et al*, 1974) and unsocialised aggression (Delamater & Lahey, 1983). Since these three clinical problems often coexist, which of them (or what other associated problem) is linked to unresponsiveness? We shall only know when further research untangles the various problems.

Attention deficit

Hyperactive children fail to sustain organised attention, and score poorly on psychological tests related to attention (Douglas, 1983). However, test performance can also be upset by many different sorts of psychopathology (Shaffer, 1980), and the impairment has not yet been shown to be specifically an attention problem. Although hyperactive children behave impulsively, psychological tests of impulsiveness suggest that they respond too slowly, not too quickly (Sergeant, 1981; Sandberg *et al*, 1978; Firestone & Martin, 1979). Although they behave distractedly, the addition of distracting information does not particularly disrupt test performance (Douglas & Peters, 1979). The deficit is present in both short tests, such as the digit span, and long ones, such as continuous performance tests. It is even manifest in a downwards shift of IQ; indeed, when IQ is allowed for, hyperactivity shows much reduced (but still present) association with tests of concentration. There is as yet no sign of a structural deficit at any one stage of processing information—rather, there is a high-level failure to allocate resources sensibly to the task in hand and to maintain self-control in organising responses.

Animal models

Animal models of hyperactivity emphasise its heterogeneity. Prefrontal damage in monkeys, ventral tegmental lesions in rats, chemical damage to dopamine and noradrenaline synthesis, foetal exposure to lead, neonatal hypoxia, and social isolation can all produce a combination of overactivity and abnormal learning that is reversible by amphetamines (Robbins & Sahakian, 1979).

Genetic inheritance

No really persuasive study has yet been made on the effects of genetic inheritance. Full siblings of hyperactive children are more likely to be affected than are half-siblings (Safer, 1973), and hyperactive children, like antisocial children, have many relatives who themselves show sociopathy or alcoholism (Stewart *et al*, 1980). However, family interactions could explain these findings as well as genetic inheritance. The biological parents of hyperactive children living with their natural families were more likely to have had behaviour problems than the adoptive parents of adopted children (Cantwell, 1975), but this study could not compare biological with adopted parents of the same children. The adopted-away offspring of psychiatric patients were unusually likely to be hyperkinetic (Cunningham *et al*, 1975), but a transmission of hyperactivity itself was not demonstrated. Comparisons of dizygotic with monozygotic twins suggested that the latter were more concordant with regard to several temperamental dimensions, including overactivity (Torgersen & Kringlen, 1978); twin studies of children with diagnosable conditions have not yet been reported.

Chemical agents

The most controversial aspects of the aetiology of hyperactivity have come from theories implicating exogenous toxins, such as lead and food additives.

Severe lead poisoning causes brain damage, and its sequelae include hyperactivity. Lower levels of exposure can cause hyperactivity in animals, but their significance in children is still debated. High levels within the normal range are associated with small increases in problem behaviour and with lower IQ (Needleman *et al*, 1979; Yule *et al*, 1981). It is still uncertain whether these correlations reflect the damaging effect of lead at low levels of exposure, or an association with other kinds of social adversity (Rutter, 1983*a*). In any event, lead exposure is not the major cause of hyperactive behaviour.

In spite of the scientific uncertainties, there is a brisk trade in testing hair for lead and other minerals and in selling supposed therapies. The enterprise should not be encouraged: commercial hair measurement is unreliable (Barrett, 1985), and the proprietary treatments are unevaluated.

Food dyes and preservatives were implicated by Feingold (1975), with the suggestion that hyperactive children showed a genetically determined intolerance. Unfortunately, most trials involving elimination of food additives have given rather disappointing results: some children's behaviour improves for a while when a diet is started, but it does not usually worsen again if additives are given in a double-blind placebo-controlled fashion (Mattes & Gittelman, 1981). Occasionally, however, some children do seem to show a genuine improvement in behaviour due to the physical effect of removing additives (Weiss *et al*, 1980). The improvement is not confined to hyperactivity, but extends to 'difficult' behaviour generally. A recent trial involving exclusion of a wide range of different foods from the diet suggested that more children can be helped by a radical exclusion of many allergens; however, the trial was based on a selected sample with a high prevalence of physical allergies (Egger *et al*, 1985). Since some parents become preoccupied with diet to the exclusion of all else, more research is quite pressing.

Psychosocial factors

The psychological environment can determine the extent to which children are attentive (Taylor, 1980). It is therefore logical to seek evidence of psychosocial adversity in the hyperactive. Affected children, ascertained in a population survey of children with minimal brain dysfunction, can be distinguished from normal controls by signs of unsatisfactory family life (Gillberg *et al*, 1983). Children who have grown up in institutions are particularly likely to be hyperactive in their classrooms, even after they have been adopted into a family home (Tizard & Hodges, 1978; Roy, unpublished). Children known to have been unwanted (because their mothers sought a termination of pregnancy unsuccessfully) are more likely to be excitable, unsociable, and hyperactive when they go to school (Matějček *et al*, 1985).

Studies such as these are vulnerable to the same criticisms that have already been levelled at investigations of physical causes: they show correlations, not causes, and point the clinician only towards the need for comprehensive assessment.

Developmental course

Hyperactivity most commonly presents during the early years of schooling. Retrospective enquiry usually finds an onset before the age of five and often before the age of two.

Infants vary greatly in their activity levels, but the individual differences are not very stable over time. Indeed, one careful study suggested an inversion in the intensity of behaviour between the age of 3–4 days and that of $2\frac{1}{2}$ years (Bell *et al*, 1971): the neonates with highest magnitude, frequency, and speed of behaviours became the two year-olds with least vigour and lowest responsiveness.

In preschool children, overactivity is a common complaint made by parents, but sometimes reflects impaired parental tolerance rather than an abnormality of development. Although (as considered above) no clear hyperkinetic syndrome has been validated for this age-group, nevertheless, the complaint of overactivity should not be ignored: it predicts later antisocial disorder quite strongly (Richman *et al*, 1982).

From the age of about three years, the normal course of development involves a reduction of general level of activity in some settings but not in others (Routh, 1980). The appropriate modulation of activity can therefore be impaired by developmental delay, whatever the cause. The resulting restlessness becomes more of a problem as schooling proceeds and successively greater demands are made on children's powers of attention and self-control.

The continuities between the early years of schooling and outcome in adolescence have been examined by many follow-up studies, mostly of children referred to clinics with ADDH, contrasted with normal controls from the community (see reviews by Weiss, 1983, and Thorley, 1984*b*). The investigations are limited by design weaknesses (highlighted by

Shaffer & Greenhill, 1979), but taken as a whole, they suggest that the broad group of hyperactive children do not simply grow out of it. In adolescence, they tend to remain somewhat impulsive and inattentive, and to be characterised by high rates of academic failure, antisocial behaviour, and delinquency. An epidemiological study in England also found pervasive hyperactivity to be a strong predictor of persistence of psychiatric disorder between the ages of nine and 14 (Schachar *et al*, 1981).

There is even some evidence that severe overactivity remains a risk factor for adolescents when the controls themselves show antisocial conduct. Thorley (1984*b*) identified children with the hyperkinetic syndrome seen at a psychiatric hospital, followed them into adult life, and compared them with matched cases of conduct disorder from the same hospital. Their adolescent outcome was characterised by more episodes of psychiatric treatment, more epileptic fits, more accidental injuries, and more placements in special schools.

In adult life, most children with ADDH do not show diagnosable illness, but the rate of antisocial and impulsive personality disorder is high (Hechtman *et al*, 1984). It is probably no higher than that of less hyperactive children with conduct disorder (Thorley, 1984*b*); however, the degree of conduct disorder in childhood and the disturbance of family relationships are better predictors of an adult antisocial adjustment than is hyperactivity itself. It seems likely that pervasive hyperactivity has its major effect on development by increasing the risk that affected individuals will develop complicating problems such as educational failure or antisocial conduct disorder. It they can be protected from such complications, then a gradual improvement in adjustment in adult life can be expected.

Treatment

Multiple factors interact to cause hyperactivity; its course is largely determined by complicating adversity. Diagnosis is therefore not enough to dictate treatment; a wide assessment and a range of interventions are needed.

Drug treatment

Amphetamines and related central nervous system stimulants are the commonest treatment for ADDH in the USA, being given to more than 1 schoolchild in 100 (Bosco & Robin, 1980; Lambert *et al*, 1978). By contrast, they are so rarely used in the UK that the first-line drug, methylphenidate, has recently been withdrawn for lack of demand. Dogmatic guidelines are therefore inappropriate.

Scores of double-blind trials have shown that stimulants are more effective than placebo in suppressing hyperactive behaviour for children with ADDH (Taylor, 1985*b*). These trials are based on relatively short periods, from a few weeks to a few months; the long-term effect is less clear. Some children who have taken amphetamines for long periods are no longer getting any benefit (Charles *et al*, 1979). Others show worsened symptoms when their drug is removed (Sleator *et al*, 1974). Even if symptoms are controlled in the long term, it does not follow that long-term development will be improved. Follow-ups of treated and untreated groups leave it uncertain whether drug treatment is of any use over a period of years (Weiss, 1983). Accordingly, long-term treatment should only be embarked on when there is clear evidence of short-term benefit, and good supervision (including regular periods off-drug) can be provided to monitor the continuing need for therapy.

The short-term action of drugs is not paradoxical or unique to hyperkinetic children: the effects are qualitatively similar in hyperactive children, normal children (Rapoport *et al*, 1978), normal adults (Weiss & Laties, 1962), and conduct-disordered children (Taylor, 1983*b*). In all these groups, they act as stimulants, not sedatives; they reduce high-frequency and off-task behaviours, and they improve performance on a variety of psychometric tests. The mechanism probably involves catecholamine neurotransmitters (Rapoport & Zametkin, 1986). Though the action is similar in kind in different groups, it varies greatly in degree.

The best and clearest indication for stimulant medication is the uncommon nuclear hyperkinetic syndrome of pervasive and severe hyperactivity and cognitive impairment in the absence of overt emotional disorder, presenting in primary schoolchildren of normal or only mildly retarded intelligence. There are, however, other indications too. Sometimes it is desirable to induce a rapid reduction in restless, disruptive behaviour in order to bring about a change of attitude in the child's caretakers. Sometimes medication is necessary to allow a programme of learning to operate. In these circumstances, stimulants may be given to children with lesser degrees of hyperactivity. Whatever the indication, stimulants need to be planned as an adjunct to educational or psychological help.

Careful monitoring is needed, and the reader is referred to fuller accounts of drugs and their prescription (e.g. Barkley, 1981; Taylor, 1985*b*).

Dietary treatments

Dietary treatments are very popular with parents, and present a dilemma because of the difficulty of recognising those few children who may be helped. The diets carry the hazards of poor nutrition and the neglect of other treatments. In present knowledge, they should only be prescribed with expert help, but it is reasonable to support parents who have themselves decided on a trial.

Psychological treatments

The techniques of behaviour modification can be applied in many different ways to the treatment of hyperactivity (Yule, 1986). Operant conditioning is of demonstrated value in the short-term reduction of off-task behaviour and increase of constructive activity in the classroom. Although it may sometimes be less potent than stimulant drugs (Gittelman *et al*, 1980), it is also more widely acceptable.

Reward-based schemes of learning can also be used to reduce gross motor activity and fidgetiness (Christensen & Sprague, 1973), but the mere lessening of activity is seldom enough to help children's adjustment. They also need to learn more positive skills of learning and social interaction.

Cognitive-behavioural therapy has promise as a tool for teaching techniques of self-control and problem-solving (Bornstein & Quevillon, 1976). Systematic evaluations have indicated that it can improve performance on laboratory tests, for instance of impulsiveness (Douglas, 1983). Its value in promoting longer-term adjustment and real-life performance is not yet established.

Considerations of developmental course emphasise the importance of helping wider aspects of a child's predicament than hyperactivity alone. Adverse and coercive styles of family interaction can readily develop, and may indicate behavioural techniques of modifying such interactions, or the training of parents in behavioural skills, or the approaches of conjoint family therapy. Educational failure is so common and important that a liaison with schools, and a consideration of special education needs, should be part of every psychiatric assessment. Many affected children acquire a very negative view of themselves as 'weird', 'rubbish', or 'stupid'; individual sessions therefore have a counselling purpose as well as that of instilling skills. If drug treatment is to be used, it needs full and repeated discussion with child and parents, lest it lead to scapegoating or to a glib alibi for bad behaviour.

Conclusions

Research continues, but provisional conclusions can be made. Severe degrees of inattentive and restless behaviour constitute a major problem for children's development, and are different from (but overlap with) conduct disorder. They probably need more recognition by psychiatrists and teachers in the UK, but may be over-diagnosed in the USA. Delays in motor and cognitive development are often associated, and may well be aetiologically important; however, they are not yet sufficiently specifically described to justify the view that affected children have a structural deficit in the processing of information from the environment. Powerful short-term treatments are available, in the shape of stimulant drugs and behaviour modification, but their long-term effect is inadequately known. Family, school, and peer relationships are usually more important than the core problem in determining eventual adult outcome. Clinical services therefore need to develop a range of treatments for affected children.

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