1. Child psychiatry

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Recent clinical research has re-examined various aspects of a wide variety of clinical syndromes. This seems a timely opportunity to review some of this work. In this chapter we tackle three of these themes — hyperactivity, both because it is so topical and because UK and US concepts are beginning to converge; childhood hysteria, which has been relatively neglected, perhaps because clinical diagnoses have tended not to stand up to objective scrutiny; and, finally, the non-psychotic syndromes of social isolation and withdrawal in childhood and adolescence, for we now have a better idea of their boundaries, clinical features, aetiology and prognosis.

HYPERKINESIS AND OVERACTIVITY

Many aspects of the so-called hyperkinetic syndrome have proved to be very contentious — theoretically, conceptually and clinically. This section considers some of the main questions which have been highlighted by Werry (1981). For example, is there agreement about the cardinal features of the clinical picture? What implications does this have for definition and diagnosis? Is hyperkinesis common or uncommon? Is hyperkinesis attributable to brain damage? Is there a relationship between hyperkinesis and developmental disorder? Does hyperkinesis differ from conduct disorders, and can these conditions co-exist? Finally, what is the contribution of psychopharmacological agents? A better understanding of phenomenological and aetiological issues is achieved by looking at this subject from the historical standpoint. In addition, different emphases have been given to the phenomena, aetiology and treatment in the UK and the USA.

Clinical picture

In the 1940s Strauss and Lehtinen (1947) drew attention to a possible association between hyperactivity and cerebral insult in subnormal children. Nearly a decade later, Ounsted (1955), in the UK, painted the then classic picture of hyperkinesis — a condition which he considered to be relatively rare. The features highlighted included activity, impulsivity and disinhibition, lack of fear, distractibility and short attention span, marked or subtle defects of co-ordination, excitability, emotional instability and lability, and perceptual and learning problems. The presence and relevance of most of these features have been emphasized by some workers and subjected to scientific scrutiny by others in a search for characteristic features, associations and mechanisms, predictors of change and clues to treatment.

In the 1970s, in the USA, interest became focused on those deficits of attention which were thought to be representative of hyperkinetic children: in the UK, a rapprochement was sought between UK and US concepts.

Hyperactivity

Ounsted perceived the main symptoms of hyperkinesis as pathological overactivity, with the child being constantly in motion, dashing from one activity to another, restlessly seeking, touching, grasping, investigating, manipulating and, at times, destroying objects in his environment. The child is described by parents as being 'on the go' all the time and as tireless in play. In the classroom, prominent additional features of behaviour are fidgetiness and an inability to sit still. There have been various research attempts to quantify the activity, such as the use of telemetry (Lee and Hutt, 1964), direct observation (Hutt et al, 1966), and mechanical devices, such as actometers (Schulman et al, 1965; Sprague and Toppe, 1966), but the findings have been inconclusive. The question then arises, do hyperkinetic children have greater *amounts* of activity or a different *type* of activity, compared with ordinary children (Cantwell, 1977)? It is possible that previous research, with its roots in pre-school behaviour and clinical practice, and more recent research, with its basis in population and school studies, have inadvertently addressed themselves to two different facets of activity. Some of the newer research appears to be trying to encompass both the quantitative extremes emerging from school studies and the qualitatively deviant emerging from clinical studies; in fact, these 'conditions' may overlap only at the margins. Only one piece of research has addressed itself to this theme with appropriate scientific rigour and this has shed some light on *general* hyperactivity, which reveals itself both at home and at school, and *situational* hyperactivity, which reveals itself in either the home or the school. The former type, when compared with the latter, has proved to have considerable differentiating value: the children with general hyperactivity, as a group, have lower IQs and a higher rate of psychiatric disorder than peers from a similar social background (Schachar et al, 1981).

Impulse control

Ounsted (1955) describes a lack of impulse control, so that the child appears to behave in the way he feels at that moment, with no consideration of the consequences — thus he may suddenly lash out at another child or dash on to a busy street. There is also some evidence that impulsivity and the other behavioural symptoms of hyperactive children are more responsible for problematic social behaviour than any delay in development of social skills (Campbell and Paulauskas, 1979). In addition, recent studies in the US have found that both childhood and adolescent hyperactives were more impulsive in dealing with cognitive tasks and less able to deal with confusing contextual aspects of such tasks (Campbell et al, 1971; Cohen et al, 1972).

Impulsivity is often also associated with an absence of fear and a lack of shyness, to the extent that the child may be considered to be socially disinhibited. These combinations of features suggest a degree of rudeness and disregard of social rules and, often, what appears to be calculated mischievousness. The children are often also described as being emotionally unstable and exhibiting lability of mood: they may therefore over-react emotionally to any environmental restraints, and some of their mood fluctuations may be unpredictable. Tolerance of frustration is also low, with the result that aggressive outbursts appear to be provoked easily, even by trivia.

Attention span, distractibility and cognitive development

Distractibility and short attention span are particular features which are noted by the

child's teacher. He tends to flit from one activity to another and has been described as being unable to concentrate. From his direct observations, Ounsted reports that hyperkinetic children tend to focus their attention and all their motor systems simultaneously in one direction, but quickly shift these activities elsewhere, albeit in the same circumscribed fashion.

Hyperkinetic children tend to be poor at tasks involving perceptual and visuo-spatial skills, particularly those involving recognition, manipulation or copying of visual stimuli or objects in space. Furthermore, in comparison with control groups, they function less well on tests of intelligence, such as the WISC (Palkes and Stewart, 1972), and their academic achievements are low (Keogh, 1971). Ounsted has pointed out that, despite having a lower mean IQ than the controls, these children show a wide range of intelligence. Previously, it has been widely argued that many of the deficits on psychological tests reflect cerebral dysfunction: however, modern academic psychologists question the validity of such tests as indicators of this type of dysfunction. There is some dispute about the academic achievements of hyperkinetic children: some workers claim that these are poorer than expected (Minde et al, 1971), with three-quarters of the children being retarded to some extent on academic subjects; other workers (Palkes and Stewart, 1972) claim that the academic abilities of hyperkinetic children are consistent with their intelligence.

Antisocial behaviour

In contradistinction to the picture described in the UK, North American studies report that an important minority of younger hyperactive children *initially* present with antisocial behaviour, whereas older children with hyperactive behaviour frequently display antisocial symptoms (Stewart et al, 1966; Weiss et al, 1971). This gives rise to a number of interrelated questions. First, are the antisocial symptoms an integral part of a hyperkinetic state with an early onset? Second, is there a symptom shift from overactivity in early childhood to antisocial symptoms in the course of time? Third, is this a lifelong hyperkinetic state with associated secondary antisocial behaviour? Fourth, is there a variety of hyperkinesis which first reveals itself when the child is older and is associated with antisocial behaviour? Fifth, are overactivity and impulsivity in older children part and parcel of a syndrome of conduct disorder? There are, as yet, no definitive answers to these questions, which are the subject of continuing research. Some of the research findings are discussed below.

Natural history

Because of the different concepts of hyperkinesis used in the USA and the UK, it is difficult to obtain a clear picture of the natural history. Nevertheless, some generalisations are possible. First, the main symptoms that present in the toddler period make up a degree of hyperactivity which parents often find intolerable. Many of the more florid and intolerable symptoms reduce (Ounsted, 1955) or change (Wender, 1971) with age; nevertheless, subsequently teachers, in particular, define the problem in terms of distractibility, poor attention span, indifference to discipline, and learning difficulties. Continuity of hyperactivity into conduct disorders of adolescence is reported in about one quarter of cases in the more rigorous North American studies (Laufer, 1971; Weiss et al, 1971; Denhoff, 1973). Thus, in the toddler period the parents are more likely to complain of overactivity, impulsivity and

low threshold of frustration; in the primary school stage the teachers will complain of restlessness, indiscipline and educational problems; and in the secondary school the emphasis is more on academic problems and antisocial behaviour.

The only clinical follow-up in the UK was undertaken by Ounsted (1955) in relation to hyperkinesis associated with convulsive disorders. He describes a diminution of the hyperactive behaviour over the early school years. In North America there have been many follow-up studies, which also indicate that while gross motor activity reduces with age, the hyperkinetic children are reported as being excessively fidgety, and that other symptoms persist into adolescence: these include emotional and behaviour problems, under-achievement at school and poor attention span, and poor ability to concentrate (Minde et al, 1971; Weiss et al, 1971; Minde et al, 1972). The academic underachievement and behavioural difficulties at school are characteristic in children described as hyperkinetic in North America; the behaviour that is described is more of an antisocial variety. The fact that the disturbed behaviour of hyperkinetic children persists is not surprising, as there is already good evidence in the literature that conduct disorders in childhood have associations with antisocial behaviour at later ages (Robins, 1966). Nevertheless, one follow-up of previously hyperkinetic children into adulthood did not indicate a continuity of such antisocial behaviour or any other psychiatric disturbance (Hechtman et al, 1976). Examination of the cognitive style (i.e. measures of the individual differences used in problem solving rather than differences in intelligence) indicate that the inefficiencies in this respect shown by younger hyperactive children not only persist into adolescence, where they show an impulsive rather than a reflective cognitive style (Cohen et al, 1972), but also into adulthood. Further, such adults, despite learning to slow down their rate of response, do not show a significant improvement in accuracy on such tasks (Hopkins et al, 1979).

Classification and aetiology

In the UK, hyperkinesis has been considered to be a rare condition, even where there is demonstrable evidence of brain damage (Ounsted, 1955). In the USA, it is considered to be a relatively common syndrome with a characteristic clinical picture, natural history and functioning on psychological tests (Cantwell, 1975). The differences between the countries are not merely those of rarity and probably reflect the diagnostic criteria employed. First, there is the *context* of the diagnoses: most of the children diagnosed as hyperkinetic in the UK in the 1950s were seen in paediatric-type clinics and hence the greater likelihood of historical or clinical evidence of definite brain damage. Second, in the UK, the children were invariably *younger*, i.e. pre-school or early school age at initial diagnosis, whereas they include a wider age range in the USA. Third, the diagnostic symptoms, as described above, were not only more *circumscribed* and *characteristic* (e.g. hyperactivity, impulsivity, poor concentration), but were of marked *severity*, in contrast to the US where there is a looser concept with a wider number of symptoms, including antisocial and aggressive behaviour, with overactivity being of lesser severity (e.g. restlessness and fidgetiness). Fourth, whereas in the UK it was unlikely that a child would be labelled as hyperkinetic if he or she showed symptoms in *one specific situation*, such as the school, this was quite likely to occur in the USA (Schachar et al, 1981).

What are the cardinal or diagnostic symptoms of the hyperkinetic syndrome?

Hyperactivity, distractibility, excitability and impulsivity are commonly described in the literature (Ounsted, 1955; Werry, 1968; O'Malley and Eisenberg, 1973), and are considered to be cardinal symptoms. A hypothesis that has gained ground in the USA is that the essential features of the syndrome are not the children's overactivity, but rather the distractibility and short attention span, giving rise to the concept of 'attention deficit disorder with hyperactivity' (Cantwell, 1977). There is a suggestion that children with 'pure' hyperactivity in the school situation have associated reading and calculating disabilities which Stewart et al (1981) adduce as support for the concept of an attention deficit disorder with hyperactivity. However, in the UK, little overlap has been found between the group of children scoring high on hyperactivity and the group scoring high on inattention; furthermore, there were few differences between these groups in relation to adverse background factors (Sandberg et al, 1980).

The next key issue is the continuing dispute and debate about the importance of *brain damage* as an aetiological factor in hyperkinetic behaviour. Strauss and Lehtinen (1947) reported hyperactive behaviour in *some* mentally handicapped children with presumptive evidence of brain damage. Further, despite the fact that only *some* of the children with demonstrable brain damage exhibited hyperkinetic behaviour (Rutter et al, 1970a) and only *some* of the children with hyperkinetic behaviour are shown to have unequivocal brain damage (Werry, 1972), in the course of time hyperkinesis came to be considered as synonymous with brain damage. The circularity inherent in these arguments is patently obvious and has been fully exposed by modern research. As pointed out by Birch (1964), quite often the diagnosis of brain damage depends on 'contaminated' evidence from workers in other disciplines: thus, medical clinicians in their diagnostic formulations have tended to rely on psychometric findings; in turn, psychologists in their diagnostic assessments have tended to rely on the findings of medical clinicians. Each of these disciplines has tended to use the other as independent proof of the validity of their diagnoses. Furthermore, while first descriptions of this type of behaviour were reported in children, only some of whom had unequivocal evidence of brain damage, this illogical view has tended to persist. The terms 'minimal brain damage' and 'minimal cerebral dysfunction' have also come to be applied to children showing some or a number of the behavioural features of the hyperkinetic syndrome, the implication being that these children suffer from at least subtle neurological disturbance. This appellation continues, despite the absence of unequivocal evidence of brain damage or even presumptive evidence of minor or neurological 'soft' signs (Langhorne et al, 1976). The presence of 'soft' signs constitutes only tenuous evidence for a brain dysfunction diagnosis. Nevertheless, excessive 'soft' signs have been described by some workers (Werry, 1972), but not others (Rutter et al, 1970a; Langhorne et al, 1976). In this context, it has been suggested that hyperkinetic children with 'soft' signs show a greater likelihood of response to stimulant drugs (Satterfield, 1973), and certain workers (Cantwell, 1977) suggest that this may be a meaningful subgroup.

Prospective studies of prenatal and perinatal complications, such as being very light for dates, have shown that such complications are associated with subsequent hyperkinetic behaviour (Neligan et al, 1976). However, as the hyperkinetic behaviour is only one component of a more widespread behavioural deviance, the associations cannot be considered to be specific.

Population studies have demonstrated that children with definite brain damage may

show some kind of psychiatric disorder (Rutter et al, 1970a). However, a comparison of brain-damaged and psychiatrically disturbed boys failed to show the hypothesized differences of gross motor activity, fidgeting and attention (Shaffer et al, 1974). On the other hand, as Shaffer and Greenhill (1979) have pointed out, a number of other factors appear to correlate with hyperactivity: these include poor environmental stimulation (Tizard, 1968), social disadvantage (Rutter et al, 1970b), and minor physical anomalies (Waldrop et al, 1968; Rapoport and Quinn, 1975). The latter authors have come to the conclusion that the symptoms of hyperactivity and impulsivity are likely to be a final common outcome of a variety of congenital, toxic and environmental influences. Their findings concerning minor physical anomalies in the USA have not been replicated in a recent UK study (Sandberg et al, 1980). It has been suggested that hyperactivity is a type of developmental delay; the evidence for this is not strong, but consists of other signs of uneven development, a tendency to improve in the course of time, and the fact that it is more prevalent in boys. Finally, the claim that allergy to artificial food additives contributes substantially to the development of hyperactivity (Feingold, 1975) has been discounted by Wender (1980): rigorous research has not been able to demonstrate any dietary effects when using the Feingold diet (Mattes and Gittelman, 1981).

In summary, there is inadequate evidence to support the notion of a specific association between hyperkinesis and demonstrable insult to the brain.

The aetiology of the *learning problems* in the hyperkinetic child has been looked at in three ways (Keogh, 1971; Douglas, 1972; Cantwell, 1977). First, it is suggested that neurological impairment may cause both the behavioural syndrome and the cognitive disability. Second, it has been suggested that the overactivity may interfere with attention and hence with the acquisition of knowledge. The third mechanism implicates impulsivity and decision-making which impairs learning.

A topical theme is the inclusion of antisocial behaviour in the hyperkinetic syndrome. Recent research findings in the UK and USA have been convergent in that they have failed to provide evidence in favour of a broad concept of a hyperkinetic syndrome: Sandberg et al (1978), in the UK, could not distinguish groups of conduct-disordered children with high or low ratings on a hyperkinesis scale on a number of measures, such as prenatal or perinatal complications, neurological abnormalities or psychiatric disturbance of mothers. Similarly, Stewart et al. (1980), in the USA, could not find any differences between hyperactive children with conduct disorders and children who merely had conduct disorders. In both studies, hyperactive children had higher rates of reading problems than conduct-disordered children, and the parents of the hyperactives had fewer psychopathological traits. Both studies support the concept of a narrower syndrome of early childhood onset of generalised overactivity with an 'erratic style of cognitive functioning and neurological immaturity' (Sandberg et al, 1978). There is also evidence from follow-up studies of hyperactive children, that while aggression strongly predicts aggressive and delinquent behaviour in adolescence, hyperactivity only weakly predicts school achievement (Loney et al, 1978); this does not support the notion of a broadly defined syndrome of hyperkinesis. However, other work suggests that if antisocial behaviour is associated with hyperactivity, the hyperactivity is predictive of a poorer outcome than when antisocial behaviour exists on its own (Offord et al, 1979). Further, Shaffer et al (1974) report that neurological dysfunction did not affect attention, as reflected

on a vigilance task, nor movement (as measured using a mechanical device), whereas inattention and overactivity were shown to correlate in a non-specific way with conduct disorder.

However, the situation is even more complex than this, as some family studies have suggested that those hyperkinetic children with antisocial behaviour form an aetiologically distinct subgroup, with a familial and probably genetic relationship between the hyperkinetic syndrome and antisocial personality in adults (Cantwell, 1975). This hypothesis was based on information derived from comparisons between families of hyperkinetic children and controls, which was insufficient in that it could not indicate that such associations were specific for hyperkinesis. For instance, there is a well-known association between social factors and conduct disturbance in childhood (Rutter, Tizard et al, 1970; Kolvin et al, 1977), which is very similar to that described in Cantwell's (1972) research on hyperkinesis. Indeed, the identified associations are likely to be general characteristics of families attending child psychiatric clinics (Stewart et al, 1980). In fact, the key association appears to be between antisocial personality of fathers and aggressive, antisocial behaviour in boys, but not hyperactivity. This recent work again suggests that the concept of hyperactivity used in the USA is too wide and that conduct disorders, at least, should be considered separately.

With regard to diagnosis, again a distinction must be drawn between hyperactivity as a symptom and hyperkinesis as a syndrome. There are also good reasons for suggesting that hyperkinesis is heterogeneous. Another problem is whether, in diagnoses, one should use behavioural symptoms and aetiology, or behavioural symptoms alone. Diagnosis based on combinations of behavioural and aetiological pathology is so fraught with difficulties that most workers apply the term hyperkinesis to a behavioural syndrome without aetiological implications (Cantwell, 1977). This, indeed, is consistent with the Newcastle view. Further, when it comes to *classification*, there are strong arguments for classifying hyperactives bi-axially, that is, to classify separately on the basis of behaviour and of aetiology.

Behavioural axis which has four sub-categories:

1. Pervasive hyperkinesis with minor, if any, problems of conduct (this is consistent with the British concept of hyperkinesis).
2. Situational hyperkinesis with minor, if any, problems of conduct.
3. Predominantly a problem of conduct with some hyperactivity, fidgetiness, etc.
4. Mixed hyperactive-conduct disorders.

Aetiological axis. Included in this axis are six possible sub-categories:

1. Idiopathic.
2. With demonstrable brain damage (Ounsted, 1955; Ingram, 1956).
3. With significant perinatal problems (Neligan et al, 1976).
4. With developmental delay (Rutter et al, 1975).
5. Genetic temperamental (Thomas et al, 1968).
6. With significant psychosocial pathology in the family background.

Prevalence

As already indicated, the physicians in the USA appear to employ a looser and wider definition of hyperkinesis, and the UK physicians a much narrower and tighter

definition. Thus, whereas there are broad similarities in the rates of symptoms of overactivity in the cross-national studies, there are considerable discrepancies in the rates of the hyperkinetic syndrome. Some 50 per cent of USA parents describe their sons as having symptoms of hyperactivity (Lapouse and Monk, 1958) and up to 20 per cent of elementary school children are thought to have a hyperkinetic syndrome (Stewart et al, 1966; Huessy, 1967; Wender, 1971). In the UK, in the Isle of Wight study (Rutter, Tizard et al, 1970), one-third of all the boys aged 10–11 years were described as overactive and inattentive by parents, and one-fifth by teachers. Nevertheless, these workers concluded that there were only two truly hyperkinetic children in over 2000 11-year-old children which they studied. However, in Europe, higher prevalence rates are reported — about 5 per cent of boys in a school population in Holland (Prechtl and Stemmer, 1962). Finally, in his series of children with convulsive disorders in the UK, Ounsted describes an 8 per cent rate of hyperkinesis which is far below the 40 per cent rate described by Safer and Allen (1976) for their USA psychiatric clinic population.

Treatment

Pharmacotherapy

In North America, the treatment of choice is the *central nervous system* stimulants. The most widely used psychostimulants are methylphenidate (Ritalin) and dextroamphetamine (Dexedrine). Paradoxically, in the short term, stimulant medication is described as having a positive effect both on overactivity and other aspects of behaviour and learning problems. Up to two-thirds of the children are reported as showing considerable improvement, but up to 10 per cent become more irritable and distractible (O'Malley and Eisenberg, 1973; Cantwell, 1975; Ross and Ross, 1976; Safer and Allen, 1976). Improvement has been reported in terms of an increase in control of motor activity (Witt et al, 1970), intelligence and learning (Conners, 1967; Werry, 1970). Such psychostimulants appear to have much less effect in free play situations, either at home or school, than in structured situations, such as during psychological testing or in formal classroom activities. Side effects are usually temporary, lasting not more than a few weeks. These include irritability, anorexia, insomnia, headache, nausea, moodiness and depression. Psychotic episodes and growth suppression have also been reported. It is asserted that the worrying danger of later drug abuse in adolescence has been exaggerated (Beck et al, 1975).

Because of reported side effects with amphetamines, clinical trials have been carried out using a milder central nervous system stimulant known as magnesium pemoline. Recent studies suggest that its clinical efficacy compares favourably with that of dextroamphetamine (Conners et al, 1972) and that it may have practical advantages over other stimulants as it requires only a once-daily regimen (Conners and Taylor, 1980) and may also have fewer side effects, particularly on growth (Friedmann et al, 1981). Moreover, the amphetamines may aggravate any pre-existing tendency to epileptic seizures. Finally, as Shaffer and Greenhill (1979) have pointed out, hyperactivity is associated with a multitude of other problems and it is not clear what aspect of behaviour the treatment influences. Similarly, there has not been any attempt to sort out the effects of such drugs in relation to the diagnostic heterogeneity of hyperkinesis.

The major tranquillizers, such as chlorpromazine (Largactil) and thioridazine (Melleril), have been more frequently used in the UK, albeit conservatively, as they also have many undesirable side effects. One of the important, and yet poorly reported effects, is their convulsive properties and hence these drugs should be used with caution whenever there is demonstrable evidence of brain damage or an abnormal EEG. Even on low doses, photosensitivity and drowsiness may occur. On higher doses, jaundice, blood dyscrasias and dyskinetic reactions have been reported.

A major question is which of the above two groups of psychopharmacological agents is more effective? In a recent clinical trial, no long-term differences of outcome were found between phenothiazines, amphetamines and placebos on behavioural or cognitive measures (Weiss et al, 1975), which suggests that neither group is particularly effective in the long term.

ANXIOLYTICS AND ANTIDEPRESSIVES

Anxiolytics are often prescribed but there is no evidence that these drugs are of any real benefit. Furthermore, barbiturates are particularly contraindicated as they often lead to an increase in overactivity and a deterioration of general behaviour (Conners, 1972). Other workers have recommended the *imipramine group* (Huessy and Wright, 1970; Winsberg et al, 1972), and recent work indicates positive effects on learning, motor performance and social behaviour (Werry et al, 1980). These latter authors suggest that the effect of imipramine is basically similar to that of methylphenidate, suggesting a stimulant-like action. However, the safety margin for imipramine is small and cardiotoxic effects and weight loss have been reported on moderate doses (Werry et al, 1980); seizures have also been described in previously seizure-free children. The child may also complain of dry mouth, constipation, blurred vision, sweating, urinary retention and postural hypotension. These drugs are, therefore, particularly contraindicated in pre-school children.

ETHICAL MATTERS

The use of inaccurate or inappropriate labels which is, in essence, a form of pigeon-holing without a scientific basis, may result in the unnecessary use of chemotherapy, with potentially harmful side effects, in an attempt to control a child's social behaviour. In this situation the ethical responsibility of the psychiatrist or physician requires him or her to take a full account of *all* the available facts when coming to a diagnosis (Graham, 1981).

Schooling

One of the crucial questions is what to do about schooling. Previously, in the case of the hyperkinetic syndrome, *a laissez-faire* policy was recommended, but this often led to excitable behaviour and chaos in the classroom. On the basis of empirical observations and rigorous research, this era of permissiveness has given way to more sensible approaches. These children appear to function better in smaller groups or classes, particularly where a greater degree of structure and control is provided. Close personal supervision also reduces the child's distractibility. However, the effectiveness of remedial education programmes has still to be demonstrated conclusively. Behaviour modification techniques have been demonstrated as being useful at school, and so has an environment with minimal stimulation (Cruickshank et al, 1961).

Psychotherapy and casework

It is important to point out that, in some cases, overactivity is associated with family problems. In such circumstances, casework with the parents, individual psychotherapy or family therapy may be useful. The parents' help can be enlisted in the implementation of operant conditioning programmes. Finally, most parents need support in accepting their child's problems and understanding his needs. Such therapy often goes a long way to helping the family cope with life's stresses and reducing the frustrations which hyperkinetic children so often experience.

HYSTERIA IN CHILDHOOD

Although hysterical disorders in adults have been the subject of much research, less attention has been paid to childhood hysteria. The history of hysteria has been well reviewed by Merksey (1979); it is important to link the earlier work with those modern developments in the study of hysteria that are relevant to childhood and adolescence.

The work of Charcot (1889) permitted a distinction to be made on positive neurological grounds between those symptoms with a demonstrable physical basis and those with a psychological origin. Janet (1907) considered that a patient's emotions, thoughts and physical symptoms were intimately associated and he emphasized the presence of specific personality traits such as egocentricity and shallow affect. Breuer and Freud (1893–1895) advanced hypotheses that unconscious mental conflicts produce anxieties which are then converted into physical symptoms. All these concepts and hypotheses have particularly influenced child psychiatrists in their clinical practice.

It has been pointed out that some of the concepts and meanings which have become associated with hysteria over the last 60 years have given rise to more confusion than clarity. More recently, new ideas have emerged, two of the most important of which are the concepts of sick role and illness behaviour (Kendell, 1974; Mechanic, 1962; Parson, 1951): however, the relevance of these new ideas have yet to be established fully in relation to childhood and adolescence.

Concepts and classification of hysteria

The first succinct analysis of this problem was undertaken by Mildred Creak (1938). She classified hysteria in childhood into three sub-groups: first, true conversion hysteria, in which neurological symptoms are attributable to conversion of anxiety into somatic manifestations; second, hysterical prolongation of a symptom which was originally part of an organic illness; third, organic disease in which there are associated complicating psychological factors. Many clinicians continue to use this classification because of its simplicity and utility.

The crucial question raised by many workers over the years is whether or not hysteria is a homogeneous phenomenon and whether the protean symptomatology manifested by patients can be considered to be a distinct clinical entity. Some clues are emerging from research in adults and from the results of the classic studies of Guze and his colleagues (Guze, 1967; Guze et al, 1972). The operational definitions which they have developed (Perley and Guze, 1962) have led to a series of studies on adult patients and have identified two populations whose presenting symptoms, family

factors, natural history and outcome are clinically distinct from each other. In summarising these studies, Guze (1967) describes his first group as having '*monosymptomatic hysteria*' consistent with the conversion and dissociative reactions classically described by Freud; patients in this group are considered to have a good prognosis. Guze's second group were described as having '*polysymptomatic hysteria*' or *Briquet's syndrome* because their clinical characteristics resembled those first outlined by Briquet (1859). These patients possess a dramatic and complicated medical history, beginning before the age of 35; they exhibit at least 25 physical symptoms which may include conversion or dissociative symptoms and in these cases no other diagnosis can be made to explain such symptoms. The prognosis in this group is considered to be poor. A further variety has been labelled '*epidemic*' or '*communicable*' *hysteria* (Moss and McEvedy, 1966; Levine et al, 1974; Sirois, 1975). Unlike the individual form, this variety typically refers to the spread of physical symptoms among closely knit groups of adolescents or young adults. We will discuss these clinical types with particular reference to children and adolescents.

Finally, the concept of hysterical personality should be mentioned, as recent work has indicated that this may be relevant to adult psychiatry (Chodoff and Lyons, 1958; Chodoff, 1974; Lazare and Klerman, 1968). However, its place in child psychiatry is uncertain, as even normal children may exhibit immaturity and dependency, to varying degrees.

Monosymptomatic hysteria

Monosymptomatic hysteria has been the main focus of interest and research in childhood and adolescence and the clinical picture has been well described. Common conversion reactions are disorders of limbs, such as gait or posture, and of sensory organs, such as blindness, deafness and aphonia. Dissociative reactions are characteristically disorders of higher cerebral function such as amnesia, states of wandering and fugues.

From the clinical standpoint, monosymptomatic hysteria is unlikely to present before five years of age: slightly more girls than boys present with this disorder (Caplan, 1970; Rock, 1971; Stevens, 1969; Goodyer, 1981).

In most studies, symptoms are reported to arise rapidly and appear to be related to a recent threatening event in the child's life or in the lives of people important to the child. Symptoms tend to be static and fluctuate little: although the child's symptoms may get worse over a period of hours, or occasionally days, rarely is there an actual change of physical locus (Goodyer, 1981). Furthermore, clinical studies suggest that symptoms are unlikely to remit before appropriate psychological intervention.

There is little evidence, in childhood, of the clinical finding of '*la belle indifférence*' to the physical complaint. Most authors report that it is either absent or extremely uncommon; if indifference is present, it tends to fluctuate. Psychophysiological studies in adults have shown that patients with monosymptomatic hysteria are as anxious as phobic patients, even in the presence of apparent unconcern for their symptoms (Lader and Sartorius, 1968). The current view is that such children are aware, to a variable degree, both of their physical and of their psychological difficulties: it is therefore understandable that other symptoms which are reported include low self-esteem, lack of confidence and thoughts of being bad.

Most workers nowadays agree that these children exhibit the full range of

intelligence: furthermore, it would seem that there are essentially no distinguishing features of temperament or personality.

The relationship to organic illness

The problem of missed organic disease labelled as psychiatric, has been of concern to clinicians working with adults or children alike (Slater, 1965; Rivinus et al, 1975). Systematic studies of children admitted with a diagnosis of monosymptomatic hysteria have revealed that, by discharge or follow-up, some 25 to 50 per cent are suffering from an organic illness related to the original complaint (Caplan, 1970; Goodyer, 1981). Caplan (1970) showed that true cases of monosymptomatic hysteria differed from misdiagnosed cases in terms of a more rapid onset of symptoms and a more frequent family history of hysteria. Furthermore, it did not appear that a recent physical illness in the child predisposed towards the development of hysteria any more than towards other psychiatric disorders.

Perhaps the most difficult diagnostic group of patients are adolescents with clinical evidence of seizure phenomena. In these cases the presence of a normal routine EEG and absent neurological findings with a history of seizure activity is not sufficient to rule out organic disease. The clinical picture is often confused by psychogenic seizures which can occur in both epileptic and non-epileptic patients (Currie et al, 1971; Merskey and Buhrich, 1975). Furthermore, both anxiety and hysteria can coexist with a real physical illness (Creak, 1938; Stevens, 1969). Serial EEGs, with the use of sleep recording or even EEG telemetry, may be necessary before an organic disease can be ruled out (Stores, 1978).

Hysterical blindness in children is now recognised as being more common than clinicians have realised: it is well known to ophthalmologists, who report a course and prognosis similar to that for other symptoms of hysteria (Yasuna, 1963; Krill, 1967; Rada et al, 1973).

Aetiological factors

According to classic psychoanalytic theory, monosymptomatic hysteria is the conversion of anxiety into a dysfunction of bodily structures supplied by the central nervous system. This serves to decrease conscious anxiety, while the symptoms symbolise inner mental conflict. The neurotic conflict needs to be uncovered to help the child to 'reconvert' (Freud, 1954; Anthony, 1967)

However, recent studies on childhood hysteria have suggested alternative explanations. Some authors report that these children are at least partly conscious of their actions and learn through experience to use their physical symptoms as a maladaptive defence against anxiety (Gold, 1965; Leybourne and Churchill, 1972). Others have claimed that, in many cases, symptom formation itself can be understood in terms of identified or imitated phenomena from the child's environment, particularly where the identified patient had an emotional investment in a member of the nuclear or extended family (Goodyer, 1981). Furthermore, as previously indicated, most workers seek an explanation for anxiety in terms of recent threatening life events. In addition, the very nature of being physically ill increases a child's anxieties and even normal life events may be perceived as threatening.

The concepts of sick role and illness behaviour may be relevant in aetiology. The 'sick role' may be adopted by the child when its advantages are greater than health.

Abnormal illness behaviour and the development of dependency and being cared for have their advantages and may develop when symptoms can be prolonged by extensive physical investigations and reluctance to consider a psychiatric diagnosis (Dubowitz and Hersov, 1976).

Chodoff has suggested that monosymptomatic hysteria may be that the result of poor verbal communication skills, or the imprecision of language of certain individuals who turns to non-verbal methods of communicating (Chodoff, 1974). There is some evidence that, in children, monosymptomatic hysterics do have more difficulty in the communication of affect compared with children suffering from other psychophysiological disorders such as disturbances of the gastrointestinal tract (Looff, 1970).

There remains the question of the importance of previous psychiatric disturbance. Whereas some authors report that their cases are devoid of such previous psychiatric disturbance, others report that considerable psychiatric difficulties were noted before the onset of the hysterical disorder (Dubowitz and Hersov, 1976; Goodyer, 1981). Furthermore, monosymptomatic hysteria can be an isolated incident which, in some cases, may be secondary to a physical illness, whereas for others it will represent a period of physical symptom formation as a reflection of continuing psychological disturbance.

How common is monosymptomatic hysteria?

Estimates of the prevalence of monosymptomatic hysteria in childhood and adolescence have varied with the type of population studied and definitions employed. Studies in the United States suggest that the diagnosis is more frequent in rural than in urban populations (Proctor, 1958; Looff, 1970; Forbis and Janes, 1965). Low socioeconomic status, poor educational attainment and the presence of more primitive child-rearing practices in the rural areas are suggested as important influences in the acquisition of monosymptomatic hysteria. Studies in the UK have concentrated on in-pateint populations, where much lower rates (less than 1 per cent) have been found than in the US (Caplan, 1970; Goodyer, 1981). Epidemiological studies suggest that this condition is rare (Rutter et al, 1970, 1976).

Treatment and outcome

Treatment methods consist of symptom removal and attention to any underlying psychopathology. Although some workers have suggested the use of chemical abreaction to remove symptoms, others have been impressed by the use of simple suggestion in most cases (Proctor, 1958; Stevens, 1969; Rock, 1971). Other workers advocate a well-planned programme of physical rehabilitation combined with a minimum of physical investigations and report considerable success in symptom removal (Dubowitz and Hersov, 1976). A recent review of behavioural approaches to hysteria suggests that these could merely be giving the patient an excuse to get better (Bird, 1979). Other authors attend to any underlying psychopathological features, using individual psychotherapy for the child and casework for the parents. Simple counselling has often proved to be sufficient in uncomplicated cases. Goodyer (1981) has suggested that family therapy techniques may be useful.

The outcome of this disorder appears to be good: most patients are reported to be free of presenting symptoms and showing good psychosocial adjustment within one to

eleven years of treatment, with no reports of physical symptom substitution (Stevens, 1969; Caplan, 1970; Goodyer, 1981). Although there is a suggestion that a few of these children, as young adults, may show minor neurotic disturbance, such continuity is more likely in those cases of confirmed cerebral dysfunction (Launay and Col, 1964; Sulestrowka, 1973).

Polysymptomatic hysteria

This handicapping disorder is said to be present almost exclusively in women and has an age of onset generally in the early to mid twenties (Guze et al, 1972). However, there are now some indications that polysymptomatic hysteria may be found in older children, as well as in young adulthood.

Clinical picture

The cardinal features of this condition are recurrent or chronic ill health, associated with an extensive and complex medical history which is often rather dramatically described (Guze, 1967). The manifold features include many and varied pains, anxiety symptoms, gastrointestinal disturbances, urinary symptoms, menstrual difficulties, sexual and marital problems, mood disturbances and conversion symptoms, most of which are seen in the majority of cases. In children, features that should alert the clinician include the presence of recurrent vague abdominal (or other) aches and pains, which may be accompanied by transient mood disturbance, particularly anxiety. In many of such cases these physical symptoms may be related to recent threatening significant events such as starting school or disturbed family relationships.

There is documentary evidence for the existence of this syndrome in children. First, in a longitudinal study of child psychiatric illness, a small number of children presented with multiple somatic complaints for which no physical cause could be found (Robins and O'Neal, 1953). More recently it has been pointed out that some children with abdominal pain may go on to develop polysymptomatic hysteria (Kolvin and Nicol, 1979).

Epidemic hysteria

This syndrome has been described in various age groups, but most typically in adolescence, and in many different cultures and socioeconomic settings (Kagwa, 1964; Moss and McEvedy, 1966; Lyons and Potter, 1970; Levine et al, 1974).

Clinical picture

Clinical features may include episodes of fainting, headache, pins and needles and hyperventilation. Symptoms have a sudden onset: they always occur in more than one person and mainly in girls. There is a spread to include these people in close proximity to the original person or persons and soon a large number of people may complain of identical symptoms. These episodes of 'contagion' are interspersed with days when no new cases are reported (Moss and McEvedy, 1966; Levine et al, 1974).

Psychological and social factors

The epidemic often starts in a small number of disturbed individuals but most cases occur in children with no previously reported psychopathology. Those children with a

dominant and influential position in the school or institution appear to be important in the 'spread' of symptoms. Transmission occurs more readily within closely knit groups and the direction usually is from older to younger children. Often, the epidemic starts at a time when group anxieties, such as academic stresses or recent illnesses have created a kind of emotional vulnerability. However, some workers have suggested that the condition does not appear to affect more intellectually and socially competent individuals (Benaim et al, 1973).

Hints on management

Press publicity of the event may be harmful, but publication of the view that the disease is psychological can be helpful (Adomakoh, 1973). The school may have to be closed for a short time to prevent further bouts of contagion.

Persons of high prestige such as doctors and school teachers can allay effectively the anxieties of the children with support and reassurance and can thereby help to control the spread of symptoms. Most cases recover with simple support and reassurance given in this manner. However, it is important that the main instigators are isolated and their personal problems are examined; there should be appropriate referral to psychiatric services where necessary. In a small number of cases, psychiatric intervention may be indicated.

NON-PSYCHOTIC SYNDROMES OF SOCIAL WITHDRAWAL AND ADJUSTMENT IN CHILDHOOD AND ADOLESCENCE

There continues to be diagnostic confusion between marked social withdrawal and adjustment problems in childhood. There is little diagnostic difficulty with the early or late onset psychoses of childhood, about which there is now considerable agreement (Kolvin, 1971; Kolvin, Ounsted, Humphrey et al, 1971; Kolvin, Ounsted, Richardson et al, 1971; Kolvin, Ounsted and Roth, 1971; Rutter, 1977): similarly, the minor variations of withdrawal or fluctuating withdrawal which may occur in adolescence, do not pose any problems of diagnosis. One is therefore left with the non-psychotic syndromes of later childhood and adolescence, which can be classified as those in which the children display mutism but are also markedly withdrawn, and those in which marked withdrawal is the main feature. In this section we provide an account of these two groups.

Mutism

The various forms of mutism can conveniently be divided into those with a presumed physical basis and those considered to have psychogenic origins. The first includes that type of mutism which may be associated with profound deafness, serious mental handicap, autism or with the akinetic type of mutism. There are two types of psychogenic mutism, both of which are rare and have a dramatic presentation. Traumatic mutism is said to follow a severe psychological or physical schock and is considered by some to be a hysterical phenomenon. The media and fictional literature suggests that it is common, but a clinical survey does not corroborate this and suggests that it is an extremely rare condition (Kolvin and Fundudis, 1981). Elective mutism is the term coined by Tramer (1934) to describe a rare and strange condition in which talking is confined to a small group of intimates in familiar situations, the most

common of these being the child's home. The rest of this section is confined to a discussion of elective mutism.

Clinical picture and diagnostic factors

Confusion may arise with those excessively shy infant-school children who do not speak when first attending school (Brown and Lloyd, 1975), but these can usually be distinguished from electively mute children by the severity and persistence of the latter condition. For instance, some 90 per cent of the former improve spontaneously over the first school year. Kolvin and Fundudis (1981) consider that such excessive shyness constitutes transient adaptation reactions to the usual stresses and unfamiliarity of the new school situation, and that only the residual small percentage of children not speaking for as long as one year after starting school would be similar to those children clinically diagnosed as elective mutes.

Previously there was both a tendency to emphasise the importance of motivation in the diagnosis of elective mutism and acceptance of the notion that the presence of abnormalities in speech or language precluded such a diagnosis. However, such earlier views were based on anecdotal material or small case studies without controls. Recent studies of a more representative series of cases indicates that such previous accounts were often misleading. In these recent studies the picture that is emerging is as follows: first, more girls than boys are affected, which is unusual for childhood disorders (Wright, 1968; Kolvin and Fundudis, 1981); second, there is also evidence of slow or uneven development, including delay in onset of speech, an excess of developmental mispronunciations, signs of immaturity on the EEG and associated problems of speech, bowel and bladder function (Kolvin and Fundudis, 1981); third, these children also exhibit poorer non-verbal intelligence, a high rate of associated behavioural problems and certain adverse temperamental traits. Further, there are descriptions not of an acute onset but rather of the insidious development of shyness from the earliest years of life in most of such children (Wright, 1968; Kolvin and Fundudis, 1981).

Aetiology and outcome

Several workers in the past have postulated a psychogenic basis for elective mutism — there are many single-case reports and small-group studies which have emphasized the importance of psychopathology, such as a faulty mother-child relationship (Parker et al, 1960), family neurosis (Browne et al, 1963) or psychological trauma in infancy (Salfield, 1950). Some workers suggest that elective mutism may be a secondary psychological reaction to some biologically based symptoms: for instance, some children may avoid speaking because they are teased when they mispronounce words (Rutter, 1977). Furthermore, newer work suggests that there may be an important maturational component which slowly diminishes with age (Kolvin and Fundudis, 1981). In the recent Newcastle study, personality problems of parents, particularly those concerning social relationships and also parental psychiatric disturbance, proved to be common (Kolvin and Fundudis, 1981). The diversity of aetiological factors suggests that the origins are multifactorial and the condition heterogeneous.

Finally, follow-up reveals that less than half the elective mute children subsequently improve in both speaking and social adjustment, irrespective of the treatment they receive. This suggests that the syndrome is more intractable than most of the other

non-organic, non-psychotic psychiatric disorders of childhood (Kolvin and Fundudis, 1981).

Schizoid personality in childhood

There has been a re-emergence of interest in the personality syndrome of childhood comprising gross lack of social skills associated with a degree of naiveté giving rise to significant impairment of social relationships (Asperger, 1944). Historically, two different paths have been taken in delineating the characteristics of this syndrome. First, there is the work of Asperger (1944), whose concepts evidently derive from clinical studies of autism: not surprisingly, his diagnostic criteria are reminiscent of those described in autism. He has applied the label 'autistic psychopathy' to this syndrome, but this had led to misunderstanding because of confusion with sociopathic behaviour (Wing, 1981) of infantile autism. Second, Wolff's concepts (Chick et al, 1979; Wolff and Barlow, 1979; Wolff and Chick, 1980) derive from the schizoid personality patterns described in the adult literature (Kretschmer, 1925; Bleuler, 1954). Nevertheless, it is evident from recent work that the syndrome described by Asperger and elaborated by Wing (1981), and the syndrome of 'schizoid personality in childhood' studies by Wolff and colleages, overlap to such an extent that they should be considered to be almost synonymous (Wolff and Barlow, 1979).

The clinical picture

The cardinal or core characteristics have been variously described as solitariness; impaired empathy and sensitivity for the feeling of others; emotional detachment; increased personal sensitivity; rigidity of mental set (i.e. fixed ideas, interests and views) which at times achieves obsessional proportions; and an unusual or odd style of communication (Wolff and Chick, 1980). Additional clinical features include displaying negativism, obstinacy and aggressive outbursts in response to pressure to conform. The children affected are predominantly school-age boys, most of whom are of at least average intelligence, and are described by their parents as showing a tendency to find social activities stressful. Some tend to avoid school; many refuse to do games; and a few refuse to speak in class.

In Wing's account (1981) there are variations and differences in emphasis and elaboration of the above picture, particularly in relation to the basis of the solitariness, communication defects and the children's cognitive skills. This work suggests that: first, the basis of the solitariness is an impairment of two-way social interaction and that there is no evidence that it is primarily due to a desire to withdraw from social contact. Furthermore, some of the children are aware of their difficulties and strive to overcome them. Second, though speech and language may initially be delayed, these children eventually achieve full command of grammar and may even have an extensive vocabulary, but they tend to be pedantic, repetitive and circumstantial. Third, even their non-verbal communication is poor — there is little in the way of facial and gestural expression and this is combined with a poor reaction to the expression and gesture of others. Fourth, some have anomalous cognitive skills, such as excellent rote memories, and many have a fascination with a circumscribed area of knowledge, but appear to have little grasp of the meaning of the facts they learn. At school, they are likely to follow their own interests regardless of instruction.

Prevalence, aetiology and pathology

There have been no specific epidemiological studies, but there is some evidence to suggest that the syndrome is rare — perhaps less than one per 10 000 children (Wing and Gould, 1979).

Asperger (1944) thought that the syndrome was genetically transmitted, particularly through fathers: a recent account suggests that about one-third of fathers show a similar personality pattern to their children. Wing (1981) tends to support this view. A history of adverse pre-, peri- or post-natal experiences has been reported in about half of the cases (Wing, 1981). It has also been postulated that such a personality variant could be turned into an autistic psychosis by earlier brain damage (Van Krevelen, 1971) but there is no evidence to support this idea.

Differential diagnosis

Many of the clinical features described above may be found to varying degrees in the normal adolescent population (Wing, 1981) and this may give rise to confusion. However, in those children in whom there is grave eccentricity, or the co-existence of a widespread number of such clinical features, all of which are rather extreme, there is little difficulty in arriving at a diagnosis. Schizoid personality in later childhood may be confused with juvenile schizophrenia, only if the latter is loosely defined.

There remains the question of the distinction between the child with a schizoid personality and the older child who previously has been diagnosed as suffering from infantile autism. Some workers see a sharp distinction between the conditions, with autism not only being more severe, but different (Van Krevelen, 1971; Wolff and Chick, 1980). Other workers seem to indicate that the distinction is less clear-cut (Bosch, 1970; Wing, 1981), with certain milder autists showing a picture that resembles the schizoid personality syndrome (Asperger, 1944, 1979). However, there should be no diagnostic confusion, as the child with a schizoid personality does not exhibit the cardinal features of autism (Kolvin, 1971; Wolff and Barlow, 1979).

Thus, where the syndrome is based on concepts which derive from a study of personality traits (Wolff and Barlow, 1979) considered to be reminiscent of schizophrenic symptoms (Bleuler, 1954), then there is an increased distance between infantile autism and schizoid personality in childhood. On the other hand, where the syndrome is based on concepts related to infantile autism (Wing, 1981), then the distinction is less clear-cut.

Prognosis

Asperger (1979) reported a stable clinical picture in which there is a gradual improvement of social skills through maturation. There is, however, less improvement when there is associated mental subnormality. Nevertheless, he and other writers emphasise an early onset in childhood and a persistence into adulthood. Asperger did not consider that a schizoid personality was a pre-schizophrenic condition, for only one patient out of 200 later became schizophrenic. Wolff and Chick (1980) have described a more serious prognosis during early adult life: there is continuity of the schizoid personality characteristics consisting of difficulties with heterosexual relationships and impaired empathy; there is also a continuing single-minded pursuit of interests; a continuing unusual style of communication, and,

finally, a wide variety of psychiatric illness (50 per cent) and a high risk of suicide (25 per cent). Nevertheless, despite their impaired intimate human relationships, work records of such adults usually proved satisfactory and, at times, quite outstanding.

Relationship between elective mutism and schizoid personality in childhood

The next question concerns the relationship between elective mutism and schizoid personality in childhood. This theme has already been reviewed elsewhere (Kolvin and Fundudis, 1981). Although both groups have initial problems of social adjustment, mainly manifesting outside the home or school, there are many important differences. In highlighting these differences we draw freely on material from Edinburgh (Wolff and Barlow, 1979; Wolff and Chick, 1980) as representative of schizoid personality disorders, and Newcastle (Kolvin and Fundudis, 1981) as representative of elective mutism.

In both groups there is evidence of lifelong personality deviations in most of the children, rather than an illness with a definite onset (Wolff and Barlow, 1979; Kolvin and Fundudis, 1981). Whereas the children falling into the schizoid personality group are often described by their mothers as 'solitary', 'remote' and 'strange' (Wolff and Barlow, 1979), in the case of the elective mute group the parents are often not aware of anything unusual until the children enter school (Kolvin and Fundudis, 1981). The children in the schizoid personality group are described as showing obstinacy and aggressive outbursts, particularly when attempts are made to get them to conform, but in the case of the elective mutes the obstinacy is more usually combined with degrees of withdrawal or retreat. While obsessionality is universal in the schizoid personality group, it occurs only occasionally in the elective mutes.

By definition, none of the elective mutes speak outside the home or at school whereas, in the case of the schizoid personality group, only a few refuse to speak in class. Electively mute children may use gesture and other forms of non-verbal communication, but children with schizoid personality do not communicate well by such means. Delay in milestones and speech problems are common in elective mutes (Kolvin and Fundudis, 1981); whereas Asperger claimed that these delays were often present in the schizoid personality group, Wing (1981) reports such delays in some cases only, and Wolff and Barlow (1979) rather infrequently.

Finally, the sex ratios are quite different: in the schizoid personality group it is as high as 9 boys to 1 girl whereas, in the elective mute group, it is the reverse, i.e. 1.1 girls to 1 boy. These sex ratios are difficult to explain, especially as a few of the children in Wolff and Barlow's schizoid personality group actually refused to speak in class and technically might be considered to be elective mutes. One explanation might be that a greater number of girls than boys with elective mutism improve in the course of time and that the schizoid personality disorder syndrome constitutes the residuum of elective mutes. However, the Newcastle data do not fully support this theory, as the greater improvement of girls than boys is only a trend. Thus, there is only tenuous evidence of a continuity of elective mutism in younger children into schizoid personality disorder in somewhat older children. The differences which exist, including those of sex ratio, suggest that the condition of elective mutism is, for the most part, distinct from the condition of schizoid personality disorder, but there may be a marginal overlap between the two conditions.

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