Theoretical and Epidemiological Aspects of Attention Deficit and Overactivity in Deaf Children

Peter Hindley
Leo Kroll
St. George's Hospital Medical School

Disorders of attention deficit and overactivity, ADHD (DSM-IV), and hyperkinetic syndrome (ICD-10) are common and disabling. However, a number of factors in deaf children's development make the assessment of these disorders in deaf children problematic. The prevalence of ADHD and hyperkinetic disorder does not appear to be higher than expected in children with inherited deafness alone but does appear to be higher in children with acquired deafness and/or additional impairments. Data from a re-analysis of an earlier epidemiological study of psychiatric disorder in deaf children and adolescents suggests that these disorders are commoner than expected in deaf children and share the features of ADHD and hyperkinetic disorder seen in hearing children.

Disorders of overactivity and inattention in children are common and can be disabling (Sandberg, 1996). Their etiology is likely to be multifactorial (Sandberg, 1996); biological, psychological, and social factors all playing important parts in their genesis. Other disorders, such as mood and anxiety disorders, can present with similar symptoms (e.g., restlessness and inattention), which require very different management. Differentiating between these different disorders depends, in large part, on obtaining an accurate account of the child’s personal experience of troubling symptoms.

Deaf children experience the same range of psychiatric and psychological disorders as hearing children (Hindley, Hill, McGuigan, & Kitson, 1994). However, the presentation of psychiatric disorders and their assessment presents a range of challenges for the predominantly hearing professionals whom they see. In particular, hearing professionals can experience considerable difficulty in eliciting and identifying affective symptoms seen in deaf children who use sign language (Hindley, Hill, & Bond, 1993). Given that the developmental environment of the majority of deaf children is substantially different than that of their hearing peers, is it reasonable to assume that disorders of attention and overactivity, functions closely related to language and communication, will present in deaf children in ways similar to those seen in hearing children?

In this article Attention Deficit-Hyperactivity Disorder (ADHD) and hyperkinetic disorder and related psychological constructs are briefly reviewed. This article outlines psychological processes in deaf children that influence different aspects of the behaviors that are said to constitute these disorders and discusses the differential diagnosis of disorders of overactivity and inattention in deaf children. Recent epidemiological studies of hyperactivity disorders in deaf children are critically reviewed. Finally, the findings from a re-analysis of an earlier study (Hindley et al., 1994) are presented. The findings of these various studies are used to discuss similarities and differences in the presentation of ADHD and hyperkinetic disorder in deaf and hearing children and to outline future areas of research.
ADHD and Hyperkinetic Disorder

ADHD (DSM-IV, APA, 1994) is one of the most common child psychiatric disorders diagnosed in school-age children in the United States (USA). In the United Kingdom (UK), DSM-IV is used less often by clinicians and the diagnosis of ADHD is made less frequently. Clinicians are more likely to use the International Classification of Diseases diagnostic system (ICD-10, WHO, 1992) and the diagnosis of hyperkinetic disorder. Hyperkinetic disorder is less commonly diagnosed in the UK than ADHD is in the USA. This is partly because it is a more restrictive diagnosis. Both ICD-10 and DSM-IV see inattentiveness and impulsivity/hyperactivity as the core behavioral symptoms. They both require these symptoms to be pervasive (i.e., elicited in two or more situations) but differ in two important ways: in hyperkinetic disorder, inattentiveness and overactivity must be severe and symptoms in both domains are rated separately. In ADHD symptoms from the domains of inattentiveness and/or impulsivity/hyperactivity are pooled and are less severe. Finally, ICD-10 excludes hyperkinetic disorder in the presence of pervasive developmental disorders, affective disorders, or schizophrenia, while in DSM-IV these disorders can co-occur. Hyperkinetic disorder can be seen as resembling the mixed subtype of ADHD (Taylor, 1994), and so ADHD may pick up a broader and less distinctive group of children.

Apart from differences in diagnostic systems, the training of British child psychiatrists appears to place greater emphasis on social and psychological aspects of child mental health than that of their North American colleagues and so, indirectly, discourages the use of medical diagnoses. However, an increasing number of British clinicians, particularly pediatricians, are using DSM-IV as a guide and making the diagnosis of ADHD. More importantly, more parents are aware of the condition, so there is increasing pressure to diagnose and medically treat hyperactivity disorders (ADHD or hyperkinetic disorder) in Britain.

In light of these differences, this article uses the term hyperactivity disorders as a general term covering both ADHD and hyperkinetic disorder but specifies diagnoses where appropriate. The concept of ADHD has developed in succeeding editions of the DSM. ADHD, as described in DSM-III-R (APA, 1987), is very similar ADHD in DSM-IV. However, DSM-III (APA, 1980) used a substantially different model with subcategories, Attention Deficit Disorder (ADD) and Attention Deficit Disorder with Hyperactivity (ADDH). The substantial differences between these various editions mean that it is essential to specify which diagnostic term is being used.

Diagnostic systems: behaviors or relationships. In addition to inattentiveness, impulsivity, and overactivity, Barkley (1990) suggests that three further sets of characteristic behaviors can be described in children with ADHD: behavioral disinhibition, deficient rule-governed behavior, and inconsistency. Interestingly, Barkley specifically cautions against ascertaining deficient rule-governed behavior that may stem from other factors such as deafness or impaired language development (Barkley, 1990, p. 45). He goes on to suggest, after Skinner, that inattentiveness and impulsiveness may be better conceptualized as relationships between events in the environment and the child's behavior rather than cognitive or behavioral constructs. Thus, inattentiveness and impulsiveness are construed as reflecting "a problem of stimulus control or regulation of behavioural responses, especially in the area of behavioural inhibition" (p. 71). Barkley suggests that these are the result of three possible underlying mechanisms: (1) diminished sensitivity to behavioral consequences, (2) diminished control of behavior by partial schedules of consequence, and (3) poor rule-governed behavior.

Barkley's ideas should make clinicians cautious about using the hyperactivity disorder construct with deaf children. In particular, it seems likely that all three of these mechanisms can apply to deaf children in hearing families but as a consequence of language and communication differences and parental constructs of the child rather than as a biological disorder. This will be further elaborated below.

Hyperactivity Disorders in Deaf Children

Developmental aspects. Deaf children are said to be more impulsive than hearing children. Greenberg and
Kusché (1993) have conceptualized deaf children's impulsivity as a developmental delay that is primarily the consequence of early language deprivation. Excess impulsivity is seen as a result of delays in language and communication development and interpersonal problem-solving skills. In particular, deaf children are thought to have greater difficulty in developing affect regulation and verbally mediated self-control. Deaf children of deaf parents are less likely to experience early language deprivation. Harris (1978) found that deaf children of deaf parents had better impulse control than deaf children of hearing children, supporting the hypothesis that delayed language development is an important mediating factor in impulsivity in deaf children. Marschark (1993) suggests that parental responses to deaf children, immediately responding to requests and not explaining the need for delayed gratification, may unwittingly reinforce impulsive behavior. In particular, parents' limited signing skills may make it very difficult for hearing parents to explain to deaf children the long-term consequences of their actions.

These factors appear to overlap with the three mechanisms hypothesized by Barkley. Delays in language development and difficulties in communication between the deaf child and his or her hearing parents will lead to delays in the development of rule-governed behavior. The patterns of interaction described by Marschark are likely to produce diminished control of behavior by partial schedules of consequence. Finally, difficulties in explaining the consequences of actions are likely to lead to diminished sensitivity to behavioral consequences. However, the difference here is that the proposed underlying mechanisms are fundamentally different. Hyperactivity disorders are thought to reflect an underlying biologically determined disorder, interacting with social and psychological factors. In contrast, the mechanisms described in relation to deaf children are primarily social and psychological. However, it is important to point out that some causes of deafness (e.g., congenital rubella, meningitis, etc.) may lead to focal and generalized brain damage and so to hyperactivity disorders (Chess & Fernandez, 1980). Do studies of hyperactivity disorders in deaf children shed any light on this complex picture?

Assessment and diagnosis. A number factors may lead to a misdiagnosis of hyperactivity disorders: misattributing problems of communication, different perceptions of behavioral or learning style (in particular deaf children's reliance on visual cues), medical conditions such as seizure disorders, subtle processing disorders, and inappropriate school placements (Kelly, Forney, Parker-Fisher, & Jones, 1993a). Distractibility and restlessness can also occur in mood disorders and anxiety disorders. However, these symptoms reflect anxious and/or depressive thoughts and somatic symptoms of anxiety, which can only be elicited by asking the child directly. It appears that in hearing families with deaf children, there is a higher likelihood that parent-child interactions (see above) will occur that can mimic Barkley's hypothetical ADHD mechanisms. These interactions may be particularly marked in families with limited communication skills, in which parents may feel overwhelmed and/or rely more heavily on physical punishment (Jenkins & Chess, 1991; Gregory, 1995). In this case the child's overactivity will be situational, being most marked in the home and not reported at school. However, in circumstances where severe neglect in early childhood has occurred, often compounded by severe physical and sexual abuse, the child's social emotional development can be so severely affected that a pattern of indiscriminate affection seeking and impulsivity occurs that can be mistaken for a hyperactivity disorder.

Kelly et al. (1993a) describe a comprehensive program for evaluating deaf children who are thought to have hyperactivity disorders, including consultation with parents and school staff, direct observation of the child, and detailed medical and psychological examination. A more detailed account of the clinical assessment of hearing children with hyperactivity disorders can be found in Barkley (1990).

A variety of behavioral checklists completed by parents, teachers, and children are used in the assessment of hyperactivity disorders. Some checklists, such as the Child Behavior Checklist-Teacher Report Form (Achenbach & Edelbrock, 1986), are designed to detect a range of psychiatric disorders, including hyperactivity disorders. Others are specifically designed to detect hyperactivity disorders. Among these measures some
Table 1  Rutter hyperactivity subscale classification

<table>
<thead>
<tr>
<th>Hyperactivity at home</th>
<th>Score ≥3 on Rutter A and &lt;3 on Rutter B</th>
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<tr>
<td>Hyperactivity at school</td>
<td>Score of ≥3 on Rutter B and &lt;3 on Rutter A</td>
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<tr>
<td>Pervasive hyperactivity</td>
<td>Score of ≥3 on both Rutter A and B</td>
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Method: sum 3 hyperactivity symptom questions on Rutter A/B scales with maximum 2 for each symptom, giving maximum score of 6 on each scale.

are designed to generate information that conforms with specific diagnostic systems. For example, the Attention Deficit Disorder with Hyperactivity Comprehensive Rating Teacher Rating Scale (ACTeRS) (Ullman, Sleator, & Sprague, 1984) was designed with ADD specifically in mind. Other checklists, such as the Conner's Parent Report Scale-Revised or CPRS-R (Goyette, Conners, & Ulrich, 1978) generate information about different domains of hyperactivity disorders, such as overactivity and learning problems, without conforming to a specific diagnostic system.

Two measures commonly used in the U.K. are the Rutter A and B scales (Rutter, Tizard, & Whitmore, 1970). These are behavioral checklists completed by parents and teachers, respectively. The Rutter A and B scales contain items covering a variety of symptoms of emotional and behavioral disorders, including hyperkinetic disorder. They do not enable clinicians to make diagnoses but identify children who are likely to have a psychiatric disorder. Hyperactivity symptoms are defined by summing three questions from each of the two scales (Goodman & Stevenson, 1989; Schacar, Rutter, & Smith, 1981). Hyperactivity symptoms can then be identified in the home, school, or both (pervasive). Previous studies have suggested an association between hyperactivity scores and severity of disorder. Home hyperactivity was associated with less severe disorder. School and pervasive hyperactivity were associated with disorder of progressively increasing severity (Schacar, 1991; see Table 1).

The prevalence of hyperactivity disorders. Early studies of deaf children attending psychiatric clinics suggested that deaf children were more prone to develop hyperactivity disorders (Goldberg, Lobb, & Kroll, 1975; Williams, 1970). However, it is impossible to draw conclusions about the prevalence of these disorders in general populations of deaf children from these studies. As with any clinic-based studies, there will be inevitable biases associated with referral to psychiatric services.

There is only one published study (Kelly, Kelly, Jones, Moulton, Verhulst, & Bell, 1993) that has used standardized instruments to estimate the prevalence of ADD (Attention Deficit Disorder, DSM-III; APA, 1980) in a nonclinic-based population of deaf children and young people. Kelly et al. used the CPRS-R, completed by house parents, (Goyette et al., 1978), the ACTeRS (Ullman et al., 1984), and the Aggregate Neurobehavioral Student Health and Education Review (ANSER) System Questionnaires (Levine, 1987); the latter two scales were completed by teachers. The children studied were 238 out of 254 students at a residential school for deaf children. They were 4 to 21 years, with a mean age of 16; the authors also grouped the children according to the etiology of deafness.

There were significant correlations between the different hyperactivity factors of all three scales. The mean scores on three subscales of the CPRS-R (learning problem factor, impulsive-hyperactive factor, and hyperactivity index) were compared with hearing norms. There were no differences for deaf girls, and deaf boys' scores were significantly lower on the impulsive-hyperactive factor and hyperactivity index.

There was no association between the attention and hyperactivity factors of the ACTeRS and the age, gender, degree of hearing loss, or use of hearing aids. However, significant associations were noted between these factors and etiology of deafness. The mean scores of children with acquired deafness were significantly higher than those of children with hereditary deafness on the learning problem factor (acquired: 0.68, hereditary: .34, t = 3.18, p = .002), the hyperactivity index (acquired:0.62, hereditary: 0.41, t = 2.28. p = .024), and significantly lower, and so indicative of greater disturbance, on the ACTeRS subscale attention factor (acquired: 39.6, hereditary: 49.4, t = 2.18, p = .031).
Using a clinical cut-off of the bottom 20th percentile for the ACTeRS, 38.7% of the acquired group against 14% of the hereditary group had clinically significant problems ($p = .002$). This finding was replicated with the CPRS-R learning problem factor but not with the impulsive hyperactive factor or hyperactivity index.

Kelly et al. (1993b) conclude that deaf children with hereditary deafness are not at greater risk of developing ADD but that children with acquired deafness are and that this difference is most probably accounted for by generalized brain abnormalities associated with congenital rubella, congenital cytomegalovirus, and bacterial meningitis. However, they note that hereditary deafness, in the presence of a family history, is more likely to lead to early intervention including appropriate communication. It may also be noteworthy that the hereditary group is also more likely to contain the deaf children of deaf parents, whose developmental pathway is not affected by communication differences.

Kelly et al.'s (1993b) findings should be interpreted with some caution. First, their study population does not reflect the whole population of deaf children, who are educated in a variety of settings. Second, the age range of the children studied was skewed toward an older age group and levels of hyperactivity and inattention are often said to decline during adolescence (Barkley, 1990). Finally, they did not have a contemporaneous comparison group, relying on previously established norms instead. Earlier in this article we argued that either psychosocial or biological factors might account for differences in rates of hyperactivity disorders in deaf children. Kelly et al.'s finding that acquired deafness is associated with higher rates of the DSM III diagnosis of ADD lends weight to the theory that ADHD in deaf children is more likely to be a consequence of biological factors rather than social and psychological factors.

A recent study of Finnish deaf and hard-of-hearing children (Sinkkonen, 1994) used a different method and diagnostic system from that used by Kelly et al. Sinkkonen studied 414 out of 445 deaf and hard-of-hearing children and young people attending school in Finland. There were more boys than girls (224 versus 190) and their ages ranged from 6–21 years (mean = 11.92, standard deviation [SD] = 3.48). However, Sinkkonen reports results from the children ages 16 or younger only ($n = 379$). Sinkkonen collected data on 224 hearing children. There were proportionately more girls in the comparison group, but their age distribution is not reported.

Sinkkonen used modified Rutter B scales (Rutter et al., 1970) to collect information about psychiatric disturbance and collected data about the children's additional disabilities, communication abilities, and their parents' communication methods. Using the standardized cut-off score of 9 on the Rutter B scale, the overall rate of psychiatric disorder was not significantly increased among the deaf children, although it was among children with additional impairments. Sinkkonen looked at correlations between subscales of the Rutter B and communication ability. While he found no correlation between the neurotic and antisocial subscales and low communication ability, he found a highly significant correlation between the hyperactivity subscale and low communication ability ($p < .001$, no correlation coefficient cited). Deaf children with additional impairments had significantly higher mean hyperactivity subscale scores than children with deafness alone. Sinkkonen (p. 62) found that children with poor communication and additional impairments were four times as likely to be identified as having a psychiatric disorder (65%), as defined by scores on the Rutter B, than children with poor communication but without additional impairments (15%). Finally, Sinkkonen found no significant differences on the hyperactivity subscale between his hearing controls and children with deafness alone.

Sinkkonen's study lends support to Kelly et al.'s (1993b) suggestion that additional impairments such as brain abnormalities account for higher rates of hyperactivity disorders in children with acquired deafness. His finding that low communication ability correlated with hyperactivity raises the possibility that communication may be a mediating factor. However, the markedly higher rate of disorder among children with additional impairments and poor communication against children with deafness alone and poor communication runs counter to this.
Hyperactivity Disorders in British Deaf and Hard-of-Hearing Children

The study reported in this article used different methods from Kelly et al.'s but used concepts of hyperactivity disorders similar to both Sinkkonen's (the Rutter hyperactivity factor) and Kelly et al.'s study (DSM-III, ADD/ADHD).

Kelly et al. (1993b) suggest that hyperactivity disorders are no more common in children with hereditary deafness than in hearing children but that they are more common in deaf children with coexisting brain damage and/or acquired deafness. If this is so, hyperactivity disorders in deaf children, with hereditary deafness and without brain damage, should share the same characteristics as those seen in hearing children. In particular, children's communication ability should not be a significant factor in the etiology of hyperactivity disorders, in contrast to Sinkkonen's finding. This article describes a post-hoc analysis of hyperactivity disorders in a heterogeneous sample of hearing-impaired children who were involved in a study of the prevalence of psychiatric disorder (Hindley et al., 1994).

Three main questions were posed in conducting the analysis:

1. What is the prevalence of hyperactivity symptoms in the sample as defined using Rutter A and B questionnaires (Rutter et al., 1970); and of ADD (DSM-III; APA, 1980), using the Child Assessment Schedule (CAS, Hodges, 1987)?

2. Does this sample of children with a wide range of hearing impairment and hyperactivity symptoms show similar correlates with psychiatric disorders (DSM III) and Rutter scale deviance scores to those described in hearing children (Goodman & Stevenson, 1989; Schachar, 1991).

3. What associations exist between the presence of hyperactivity symptoms/ADD and the degree of hearing loss, communication competency, and academic achievement or adversity?

Method

Instruments and procedures. Four schools were surveyed in the original study, one residential day school for deaf children and three hearing-impaired units (HIUs); 81 children were screened using teacher checklists (TCLs) and parent checklists (PCLs) (Hindley et al., 1994) and the Rutter A and B questionnaires (Rutter et al., 1970). The TCL and PCL were developed as psychiatric screening instruments for deaf children. The front sheet of the teacher form asked about method of communication (British Sign Language [BSL]; Sign-Supported English [SSE]; spoken English). The teacher was also asked to rate the child's ability to communicate on a visual analogue scale, and three categories were created corresponding to poor, average, and good communication competence. The Rutter A has been extensively used as a psychiatric screening questionnaire in conjunction with the Rutter B questionnaire (see above).

Children who screened positive for psychiatric disorder and a sample of those screening negative on the TCL and/or PCL were interviewed using the CAS (Hodges, 1987). The CAS is a psychiatric assessment interview that has been used with a wide range of hearing children (Hodges, 1993), in both clinical and community populations. The validity of the CAS when used with deaf children was assessed in an initial pilot study (Hindley, 1993). Deaf children who used BSL were interviewed by a child psychiatrist and a BSL interpreter. Children who used spoken English were interviewed by the child psychiatrist alone. The parents of children were also interviewed using the parental form of the CAS (P-CAS, Hodges, 1987).

The severity of deafness was recorded from school records of pure tone audiograms conducted during the previous two years. The parents were also interviewed about various aspects of their children and themselves. This included an impressionistic rating of their child's academic ability, using three categories: below, average, and above average ability. A social adversity index was calculated from the results of questions asking whether the mother or father had a mental illness or criminal record, the presence of overcrowding, the need to share beds, a history of children in care, and the parents' socioeconomic group. The total possible score was 9. A score of 2 or more was taken as a positive index of social adversity and corresponded to two of the items rated present. This method replicates a standardized assessment of psychosocial adversity (Rutter, 1978).
Table 2  Summary of CAS/CAS(P) and Rutter hyperactivity (h/a) classifications

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<th>Home h/a</th>
<th>School h/a</th>
<th>Pervasive h/a</th>
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<tbody>
<tr>
<td>Number of children</td>
<td>8</td>
<td>13</td>
<td>7</td>
</tr>
<tr>
<td>Number with CAS/CAS-P diagnosis</td>
<td>6</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>Number with ADD</td>
<td>0</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Number with conduct or oppositional disorder</td>
<td>2</td>
<td>4</td>
<td>4</td>
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In the secondary analysis, hyperactivity symptoms were defined using a described and recognized method (Goodman & Stevenson., 1989; Schachar et al., 1981), which identifies symptoms in the home, school, or both (pervasive) (Table 1). This only partially corresponds with the method used in Sinkkonen's (1994) study, in which the Rutter B was used alone.

**Subjects.** There were 81 children, ages 11–16 (mean age = 13.5), 39 boys and 42 girls; 46 of the total were at the deaf school, the others (35) at the three HIUs. Teachers in the HIUs did not identify any children who used sign language, whereas in the deaf school, 80% of the children were identified as using BSL or SSE.

**Results**

In the original study, 43 children screened positive using TCL and PCL questionnaires, and 39 of these were interviewed using the CAS and P-CAS as well as 10 of those that screened negative. The prevalence of any psychiatric disorder found on clinical interview was in the range 33%–42% and 57%–61% in the deaf school and HIUs, respectively. The results of the study are reported in more detail in Hindley (1993) and Hindley et al. (1994). The rate of psychiatric order is higher than has been seen in comparable community studies of hearing children living in inner city areas. Rutter, Cox, Tupling, Berger, and Yule (1975) reported a prevalence of 25%, using a similar two-stage design. The increased prevalence is primarily accounted for by higher rates of social phobias and conduct disorder in the deaf and hard-of-hearing population in this study (see Hindley, 1993, for a more detailed account). In the U.K. most of these children's difficulties would have been addressed at the primary care level by teachers and social workers and would not have required formal psychiatric intervention.

Eight children in the re-analysis fell into the home hyperactivity group, 13 into the school group, and seven in the pervasive hyperactivity. Only 49 out of the 81 children had been interviewed, and we had complete data on six children in the home group, 10 in the school group, and six in the hyperactivity group. Because of the small numbers involved, we collapsed these three groups and compared hyperactive and nonhyperactive groups in our analysis.

There was not a significantly larger proportion of children with severe or profound deafness and hyperactivity (Fisher's exact, 2-tailed, 0.15) nor children with low teacher ratings of communication competence and hyperactivity. None of the children in the home hyperactivity group had DSM III diagnoses of ADD, four out of 10 children in the school group had ADD diagnosed at interview, and four out of five children in the pervasive group had a diagnosis of ADD. This suggests that the Rutter hyperactivity subscale was functioning in this group as it does in hearing children, with greater specificity for hyperactivity disorders among the pervasive hyperactivity group (Table 2; Schachar, 1991).

More boys than girls were rated as hyperactive (Fisher's exact 2-tailed $p = .03$), but there were not significantly more children with high adversity scale scores (Fisher's exact 2-tailed $p = .067$) nor oppositional disorder (Fisher's exact $p = .06$). In addition, parents rated the academic achievement of children in the hyperactivity group lower than children in the non-hyperactive group.

**Discussion**

These findings appear to partially confirm the hypothesis that hyperactivity disorders in deaf children are
similar to those seen in hearing children. Hyperactivity disorders were commoner in boys, and parents' impressions of their children's academic attainment suggested that they were poorer in children with hyperactivity disorders. However, the proportion of children with hyperactivity disorders and high social adversity scores or oppositional disorder was not significantly higher than expected.

When the findings were compared with those from a study of British hearing children, there was a strong suggestion that the prevalence of hyperactivity symptoms, as rated by the Rutter A and B scales, was higher in the school and pervasive groups (Table 3). There appears to be a significantly greater number of deaf/hard-of-hearing children with hyperactivity disorders. However, re-examination of the data shows that a substantial proportion of children rated as hyperactive in school were identified as such by one teacher. For this reason we are uncertain as to the wider significance of this finding.

Unfortunately, the original study used a limited assessment of the presence or absence of additional CNS disorders, so it is not possible to say whether or not this difference can be accounted for by the factors described by Kelly et al. (1993b) and Sinkkonen (1994).

Conclusion
The findings from the re-analysis of Hindley et al.'s (1994) study suggest that hyperactivity disorders in deaf children share some of the features of hyperactivity disorders seen in hearing children, with a high proportion of boys and a suggestion that deaf children with hyperactivity disorders have poorer academic attainments, based on parental impression. However, this study does not show a higher proportion of children with hyperactivity disorders and psychosocial adversity or oppositional disorder. This study suggests that hyperactivity disorders are commoner in deaf and hard-of-hearing children than in hearing children. However, it does not offer an explanation as to why this might be so. The finding that children with low communication competence ratings are not disproportionately represented among deaf children with hyperactivity disorders suggests that delays in communication and language development may not play as significant a part in the genesis of these disorders as the developmental literature would suggest. By negative inference, this lends support to the hypothesis that biological factors are more likely to account for the difference.

Although communication and language development, and their impact on social and emotional development, do not appear to play a significant part in the etiology of hyperactivity disorders in deaf children, there are remaining doubts about these findings. At a methodological level, the techniques used to assess communication and language competence in these studies are crude and offer no information about the child's interaction with his or her environment. Second, the cross-sectional nature of all three studies poses problems in determining etiological mechanisms. Although Sinkkonen's (1994) findings seem to support the hypothesis that additional impairments, presumably through the effects of brain abnormalities, explain higher rates of hyperactivity disorder, it is possible that there is an interaction between such impairments and communication. The best way of clarifying these outstanding uncertainties would be by means of a longitudinal study employing reliable measures of language development, intrafamilial communication, and additional impairments.

Finally, although the nature of the etiology of hyperactivity disorders in deaf children is now better understood, there is a need to exercise caution when un-

Table 3 Comparison of prevalence rates for this study and a similar study of hearing children

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<th>Home h/a</th>
<th>School h/a</th>
<th>Pervasive h/a</th>
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<tr>
<td>This study (%)</td>
<td>9.9</td>
<td>16.0</td>
<td>8.6</td>
</tr>
<tr>
<td>95% CI</td>
<td>3.4, 16.0</td>
<td>8.0, 24.0</td>
<td>2.5, 15.0</td>
</tr>
<tr>
<td>Goodman and Stevenson (1989)</td>
<td>12.7</td>
<td>7.2</td>
<td>4.6</td>
</tr>
<tr>
<td>95% CI</td>
<td>9.7, 15.6</td>
<td>5.9, 12.7</td>
<td>2.8, 6.5</td>
</tr>
<tr>
<td>Difference in proportions</td>
<td>p = 0.24</td>
<td>p = 0.0041</td>
<td>p = 0.065</td>
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deriving assessments in deaf children. Behavior rating scales and computerized assessments are of immense value, but the information that they provide must be interpreted in the context of an understanding of the effects of deafness on children's social and emotional development.

References


