33. Nordstrom, P., Samuelsson, M., Asberg, M., *et al.* (1994). CSF 5-HIAA predicts suicide risk after attempted suicide. *Suicide and Life Threatening Behavior*, 24, 1–9.
34. Juon, H.S. and Ensminger, M.E. (1997). Childhood, adolescent, and young adult predictors of suicidal behaviors: a prospective study of African-Americans. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 38, 553–63.
35. Pfeffer, C.R., Klerman, G.L., Hurt, S.W., Lesser, M., Peskin, J.R., and Siefker, C.A. (1991). Suicidal children grow up: demographic and clinical risk factors for adolescent suicide attempts. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30, 609–16.
36. Pfeffer, C.R., Klerman, G.K., Hurt, S.W., Kakuma, T., Peskin, J.R., and Siefker, C.A. (1993). Suicidal children grow up: rates and psychosocial risk factors for suicide attempts during follow-up. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 106–13.
37. Harrington, R., Bredenkamp, D., Groothues, C., *et al.* (1994). Adult outcomes of childhood and adolescent depression. III: Links with suicidal behaviours. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 35, 1309–19.
38. Drye, R., Goulding, R., and Goulding, M.E. (1973). No-suicide decisions: patient monitoring of suicide risk. *American Journal of Psychiatry*, 130, 171–4.
39. Clarke, G.N., Rohde, P., Lewinsohn, P.M., Hops, H., and Seeley, J.R. (1999). Cognitive–behavioral treatment of adolescent depression: efficacy of acute group treatment and booster sessions. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38, 272–9.
40. Linehan, M.M. (1993). *Cognitive behavior therapy of borderline personality disorder.* Guilford, New York.
41. Miller, A.L., Rathus, J.H., Linehan, M.M., Wetzler, S., and Leigh, E. (1997). Dialectical behavior therapy adapted for suicidal adolescents. *Journal of Practical Psychiatry and Behavioral Health*, 3, 78–86.
42. Tondo, L., Jamison, K.R., and Baldessarini, R.J. (1997). Effect of lithium maintenance on suicidal behavior in major mood disorders. *Annals of the New York Academy of Sciences*, 836, 339–51.
43. Letizia, C., Kapik, B., and Flanders, W.D. (1996). Suicidal risk during controlled clinical investigations of fluvoxamine. *Journal of Clinical Psychiatry*, 57, 415–21.
44. Montgomery, S.A. and Montgomery, D. (1982). Pharmacological prevention of suicidal behaviour. *Journal of Affective Disorders*, 4, 291–8.
45. Fava, M. and Rosenbaum, J.F. (1991). Suicidality and fluoxetine: is there a relationship? *Journal of Clinical Psychiatry*, 52, 108–11.
46. Beasley, C.M., Jr, Dornseif, B.E., Bosomworth, J.C., *et al.* (1991). Fluoxetine and suicide: a meta-analysis of controlled trials of treatment for depression. *British Medical Journal*, 21, 685–92.
47. Rothschild, A.J. and Locke, C.A. (1991). Reexposure to fluoxetine after serious suicide attempts by three patients: the role of akathisia. *Journal of Clinical Psychiatry*, 52, 491–3.
48. Montgomery, S.A. (1997). Suicide and antidepressants. *Annals of the New York Academy of Sciences*, 29, 329–38.
49. Shaffer, D., Garland, A., Gould, M., Fisher, P., and Trautman, P. (1988). Preventing teenage suicide: a critical review. *Journal of the American Academy of Child and Adolescent Psychiatry*, 27, 675–87.
50. Cummings, P., Grossman, D.C., Rivara, F.P., and Koepsell, T.D. (1997). State gun safe storage laws and child mortality due to firearms. *Journal of the American Medical Association*, 278, 1084–6.
51. Shaffer, D., Vieland, V., Garland, A., Rojas, M., Underwood, M., and Busner, C. (1990). Adolescent suicide attempters: response to suicide-prevention programs. *Journal of the American Medical Association*, 264, 3151–5.
52. Rihmer, Z., Rutz, W., and Pihlgran, H. (1995). Depression and suicide on Gotland: an intensive study of all suicides before and after a depression-training programme for general practitioners. *Journal of Affective Disorders*, 35, 147–52.

9.2.11 Speech and language disorders of childhood and psychological mutism

Israel Kolvin

Speech and language disorders

A common question asked of clinicians in general practice, child health, and child psychiatry concerns the long-term consequences of delayed speech and language development in the early years of life.[1] A previously held view was that most young children with speech and language problems would eventually develop normally. But this is an optimistic view—it is more likely to be true of children without major mental and physical handicaps. Allied questions which have to be addressed are whether the speech and language is abnormal? If so, which children will eventually develop normally? What is the long-term outcome? How do we understand and explain the causes? Therefore it is essential to have some skills in the diagnosis and management of these disorders.

In a brief chapter there is no space to discuss theoretical aspects of the development of speech and language, particularly the newer neurolinguistic concepts which are competently reviewed elsewhere.[2] The focus will be on those types of disorders that present in clinical settings, such as specific developmental speech and language disorders, deafness, and selective mutism. Classification and prevalence will only be addressed briefly for those disorders associated with mental handicap, brain damage, or autism.

Classification and concepts

To produce normal speech there must be a functional neurological system capable of learning the 'underlying structure of language',[3] including a sensory component which can perceive and decode the incoming signals, an intact apparatus subserving, directing, and programming speech sound production, and available stimulation by exposure to language in its various forms. However, in most speech and language disorders there is no evident physical aetiology—these are the developmental disorders.

Functional clinical classification

One of the older, but more useful, functional clinical classifications is that of Ingram[4] who described three main categories of speech and language disorders: primary, secondary, and developmental. Modifications of this classification were developed by other workers.[1]

1. **Primary disorders** are disorders of word sound production, which occur in dysarthria or with cleft palate, with either demonstrable dysfunction or structural abnormality of tongue, lips, teeth, or palate.
2. **Secondary disorders** are disorders of speech sound production secondary to other diseases or environmental factors. These include marked intellectual impairment, demonstrable neurological disorders and cerebral palsy, deafness and specific psychi-

atric syndromes such as infantile autism, acquired dysphasia, and the effects of an adverse psychosocial environment.

3. **Developmental speech and language disorder** is a descriptive label for retardation of speech and language development in children who are otherwise apparently normal.

Ingram[4] views the last condition as a heterogeneous group of articulatory and language disorders in which speech and language development may not only be delayed but also be deviant. Nevertheless, he finds it useful to regard it as a spectrum of clinical disorders that range from mild to very severe.

Further, a distinction needs to be made between speech and language. **Speech** can be defined briefly as the articulatory skill associated with the production of word sound and spoken language. **Language** is a cognitive skill that utilizes a system of symbols[5] and whose main purpose is communication. It has various forms such as spoken, written, gesture, and sign.[6]

Stammer is a disturbance in fluency and patterning in time of speech which is disrupted by sound and syllable repetitions or sound prolongations (DSM-IV). Neither physical nor environmental factors have been shown to be consistent major determinants. However, it occurs in families and is more common in boys than girls—which suggests a genetic predisposition. Some young children have a transient trivial dysfluency, and parental anxieties and concerns may hinder the spontaneous improvement. In these circumstances, parental counselling by a speech therapist is usually helpful. Psychiatrists are seldom involved in the treatment of stammer, and the condition is not considered further in this chapter.

The ICD-10 classification

Specific developmental speech and language disorders are subdivided into those with a specific speech articulation disorder, an expressive language disorder, and a receptive language disorder. Following ICD-10 criteria,[7] specific developmental disorders of speech and language are those in which language delays are evident from the early stages of development, and are not a direct consequence of 'neurological or speech mechanism abnormalities, hearing impairment, mental retardation or environmental factors'. In addition, the impairments or delays are related to biological maturation of the central nervous system which become evident as the child grows older. There are wide variations in the age at which spoken language is acquired in normal children, and any delays or impairments are usually 'of little or no clinical significance as the great majority of "slow speakers" go on to develop entirely normally'.[7] This is the basis of the specific speech and language disorder syndrome. In this sense the syndrome may be considered to represent an extreme variation in normal development. As a general rule, any language delay must be sufficiently severe to fall outside the limit of two standard deviations on relevant measures to be regarded as abnormal. These conditions are much more common in boys than girls. The subclassification is as follows.

- **Specific speech articulation disorder**: the child's use of speech sounds is developmentally below that expected or appropriate for his or her mental age, although there is a normal level of language skills and non-verbal intelligence. The child's acquisition of speech sounds is delayed or deviant, and there are associated misarticulations with consequent problems for others in understanding the speech. The identified abnormalities do not have a clear basis in secondary pathology consisting of structural or neurological abnormalities.
- **Specific developmental expressive language disorder**: the child's ability to use expressive language is substantially poorer than the appropriate level for his or her mental age, but 'language comprehension is within normal limits'. There may or may not be associated articulation abnormalities.[7] The diagnosis should only be made when the severity of the delay in development of expressive language is beyond the limits of normal variation. A range of associated abnormalities and language difficulties have been described:[7] after the age of 3 years there may be a 'restricted vocabulary ... , overuse of small number of general words, ... short utterance length and word substitution ... , errors of syntax, especially omissions of word endings or prefixes, grammatical abnormalities' with prepositions, pronouns, verbs, etc., problems with fluency and sequencing, and possibly associated problems in word sound production.[7] Helpful non-verbal diagnostic features are that the child may employ appropriate non-verbal cues, such as gesture, facial expression, and mime, to communicate.
- **Specific developmental receptive language disorder**: 'the child's understanding of language is below that appropriate for his or her mental age'.[7] Commonly, expressive language is also affected and there are associated abnormalities in word sound production with the picture being one of a mixed receptive and expressive language disorder. Again, the diagnosis should only be made when the severity of delay in receptive language is beyond the normal limits of variation. In the early years, the child has difficulty in responding appropriately to verbal stimuli or in identifying familiar objects by name, or has difficulties in following simple instructions. Later, additional features include difficulties with grammar.
- **Acquired aphasia** consists of a loss of acquired language functions.[4] A birth-injured child cannot be described as having lost language functions, but more accurately as showing retardation of development of speech and language. In a 2- or 3-year-old child, acquired aphasia will initially be reflected in a language impediment and thereafter slowing of speech. When associated with epilepsy it is called the Landau–Kleffner syndrome,[8] in which there will be paroxysmal electroencephalographic abnormalities usually from the temporal lobes; the onset is between 3 and 7 years of age. Skills may be rapidly lost and impairment of receptive language becomes severe. It is thought to have a basis in an encephalitic process. Only one-third of children eventually make a complete recovery.

The categories of speech and language disorders are broadly similar in the ICD-10 and DSM-IV classificatory systems. Both give clear descriptions of diagnostic categories. However, ICD-10 provides a helpful definition of the concept of a developmental disorder which will be appreciated by both clinicians and researchers.

Neurolinguistic profiling

A classification has been developed based on neurolinguistic criteria.[2] This is commonly used by speech experts, and in their hands is a useful complementary assessment system. Research examining the profile of language impairment suggests that some of the subgroups correspond to different points in development rather than distinct disorders.

Prevalence

Specific retardation of speech and language

The rates reported in population studies are a function of the definition employed. Fundudis *et al.*[1] reported that 4 per cent of 3300 3-year-old children failed to use three or more words strung together to make some sort of sense. However, the rate reduced to 2.3 per cent when allowance was made for those children who have wide milestone delays.[3] Stevenson and Richman[9] reported expressive language delays of 3.1 per cent in 705 3-year-olds.

Other conditions

It is impossible to provide accurate estimates of the prevalence of rarer conditions when studying a relatively small population of children.[1] The following is a rough guide to the rates of the different types of conditions associated with speech and language disorders.

The single most common cause of slow speech development in paediatric clinics is **mental retardation** (learning disorder).[4] If this is defined as an IQ of less than 70, then over 2.5 per cent of the child population are likely to be affected, with over half of these children showing articulation defects or severe language disorders, or both.[10] **Deafness** is one of the major causes of delay in speech and language development—about 2 in 1000 children have deafness severe enough to merit the use of hearing aids. When **autism** is narrowly defined, a rate of 4 in 10 000 children has been reported.[11] Serious and persisting language disorders (**severe dysphasia**) have been estimated at 0.4 in 1000 children[1]—the condition seems to be as rare as infantile autism. The rate of **dysarthria** is approximately 1 in 1000 births.[1]

Prognosis

Epidemiological research reveals that the rate of problem behaviour in 3-year-old children with language delay is four times that seen in a random sample of children.[12] However, the predictive power of such early assessments is not great and the crucial question that remains is: How many children with language delay at 3 years of age will catch up in their cognitive and language ability and lose their behavioural disturbance?

Of the cohort of 3300 investigated by Fundudis *et al.*,[1] 4 per cent were previously speech retarded at the age of 3 years; one in five of these were found at school age to have serious language, intellectual, or physical handicaps and were labelled a 'pathological deviant' group. If this extreme group is then set aside and the residual group of 'specific speech-retarded children' with no other major impairments is compared with controls, the cohort is found to contain a subgroup with wider delays of milestones (28 per cent) and a subgroup with a circumscribed specific developmental delay of speech and language (72 per cent). There are firm grounds for believing that this latter group of children is very similar in speech and language development to those children with language delay described by Richman *et al.*[12] (and both consist mainly of children with moderate to severe developmental speech and language disorders.

The research carried out by Fundudis *et al.*[1] can be used to answer some questions about outcome at school age. A high percentage of the residual specific speech-retarded group still had significant cognitive and educational impairment, poor language development (including poor expressive language skills), and a more restricted type of language expression. In addition, the prognosis for behaviour was likewise poor. Speech delay was found to be a better predictor of impaired verbal intelligence than of performance intelligence. There was a better outcome for specific developmental expressive language delays, with most children showing substantial improvement by the age of five. In contrast, children with a receptive language disorder followed into adulthood show a relatively poorer outcome for conversational skills and fluency.[13]

Social influences on speech and language development

While some workers emphasize the biological determinants of speech and language, others stress the importance of psychosocial influences. Chomsky[14] postulates the presence of an innate language acquisition device which determines the deep-seated properties of organization and structure of all human languages. The emergence of speech is most easily accounted for by maturational changes, so that children of deaf parents are found to babble appropriately and to develop speech adequately, despite being brought up in a grossly abnormal linguistic environment.[15]

Environmental stimulation and social interchange, particularly with adults, facilitates progress in vocalization and language development in the early years. In contrast, even serious impoverishment of the social environment, for instance where children are institutionalized, usually may only give rise to moderate degrees of impaired vocalization, speech delay, and language retardation—with the retardation being confined mainly to language expression rather than to comprehension.[16]

Language and psychiatric disorder

Population studies with children with specific language delays show that about 50 per cent have significant behaviour problems, four times more common than the rate in the control group.[12,17] Baker and Cantwell[18] found that 300 children attending community speech and hearing clinics showed a stepwise increase in the rate of psychiatric disorder—with rates of 29 per cent for those with speech disorders and 45 per cent for those with speech and language disorders. Thus they concluded that children with communication disorders are at risk for an increased rate of psychiatric disorder. There are no identifiable behaviour patterns—except that those children with widespread language disorders are more likely to have a psychiatric disorder—and those with severe language disorders are more likely to have an associated autistic-like impairment of social relationships.[19]

Speech and language disorders and behavioural abnormality: directionality of associations

Rutter and Lord[20] analysed the hypothesized direction of the association between speech and language impairment and psychiatric disorders in childhood. They addressed the previous common assumption that the usual relationship is that psychological disorder gives rise to language problems and offered five main patterns of probable directionality.

1. A psychiatric disorder gives rise to secondary speech and language problems, as exemplified in selective mutism and traumatic mutism.
2. There is a reverse causal process whereby a primary specific developmental language disorder gives rise to a psychiatric disorder.

The mechanisms probably include a sense of frustration with poor communication and socioemotional problems, which in turn give rise to the psychiatric disorder. A similar explanatory mechanism has been suggested by Bishop.[3]

3. In autism there is a probable common underlying problem, which 'forms part of a broader cognitive impairment and results in the psychiatric disorder and the language problem including autism'.
4. Severe environmental privation may give rise to both psychiatric disorder and language problems, with the mechanisms for each disorder being rather different. Rutter and Lord[20] suggest that while language delay is a consequence of inadequate stimulation and learning experiences from adults including communication and interaction, socio-emotional difficulties are probably a consequence of impaired attachment experiences.
5. In mental retardation the children often present with delays in language development and a psychiatric disorder. Here, the directionality and relationships are complex and may have a basis in the different forms of organic brain damage that have given rise to the mental retardation. Additionally, there may be an interaction between the language problem and psychiatric disorder in both directions.

Hearing impairment

Both because of the rarity of moderate to severe hearing impairment and because the hearing-impaired infant sporadically babbles, deafness may remain unsuspected in early infancy. Often a mother will suspect that there is something unusual about her child's hearing, but she may not be taken seriously until there is concern about a poor expressional response to sudden meaningful noises. Such suspicions should be checked using reliable screening tests, which are available for use from about 6 months of age.[21] Other accurate non-invasive assessment techniques are now available, such as evoked-response audiometry. The possibility of progressive hearing loss may need to be excluded by repeat audiometric examination. Probable causes of profound hearing impairment can be identified by taking a careful history, enquiring about conditions such as serious postnatal middle-ear infections, neonatal hyperbilirubinaemia, congenital rubella syndromes, etc.

Severe organic brain damage may cause deafness and also contribute to the poorer intellectual and educational performance of such children. About 16 per cent of deaf children have central nervous system dysfunction[22] and 30 per cent have one or more additional disabilities.[23]

Usually, epidemiological studies do not reveal an excess of social and family pathology in families of deaf children.[1] Surprisingly, deaf children of deaf parents often show better linguistic development than deaf children of hearing parent, which seems to favour early complementation of verbal and non-verbal methods of communication.

Speech and language disorder in hearing-impaired children

In a short review it is possible only to touch on some of the complex theoretical issues concerning speech, language, and the intellectual development of hearing-impaired children.[24] There is some evidence that the rules of language learned by most deaf children in their early years are similar to those learned by hearing children.[1] In profoundly deaf children the development and acquisition of speech and language is different from that of children with normal hearing. This is reflected in their relatively superficial language skills, such as spoken language, but it also includes poorer inner-language abilities.[24]

Childhood deafness hampers the development of language and verbal abilities, but not necessarily that of non-verbal abilities.[1] However, this comparatively poorer performance on cognitive tasks appears to reduce with age and appropriate stimulation. In addition, there is evidence that the academic achievements of deaf children, particularly progress in reading, are poorer than those of hearing children.[25] Some consider that these cognitive and educational impairments are more an indictment of the educational system than an inevitable consequence of deafness.

Psychiatric problems in hearing-impaired children

Usually hearing children are better adjusted socially and behaviourally than hearing-impaired children.[22] Further, hearing-impaired children raised in families where other members are deaf often prove better adjusted than those raised in families where other members are not deaf.[26] However, 50 per cent of profoundly deaf children present with psychiatric disturbance in school; this is mostly antisocial behaviour,[1] but anxiety disorders are also reported.[27]

Assessment

In the assessment of children with speech and language problems a preliminary formulation and differential diagnosis can usually be made on the basis of biographical enquiry and careful clinical assessment. This should include observation of the child during interview, informal free-field assessment of hearing, speech, and language and capacity for imitation during play, cognitive ability, social and behavioural functioning, and neurological assessment.

Biographical enquiry is intended to provide information about psychosocial influences and relevant physical factors, such as cerebral insults, history of clumsiness, etc.

Observation constitutes an integral part of all forms of assessment. While gathering biographical information, clinicians should note whether the child uses non-verbal clues from its parents to understand comments or questions; or whether there is evidence of clumsiness of gait typical of the child with cerebral palsy. Expert assessment is indicated where there are doubts, which may include audiometry, evoked-response audiometry, and other specialized techniques.

Speech and language in its various forms

Speech may be delayed or articulation skills deviant. For instance, articulation may be defective in deafness, immature in mental retardation and developmental speech and language disorders, deviant and immature in autism, and variably disordered in selective mutism. The articulation defect in dysarthria is characteristic.

Language can be assessed on a number of modalities; for example, spoken, sign (manual), or written. Comprehension can be tested by assessing the child's ability to understand simple commands both with and without the provision of visual and gestural clues. Evidence of language may be obtained from the way the child communicates through gesture, mime, and conversation.[28,29] 'Inner language', which reflects an understanding of a symbolic code, can be assessed indirectly

by observing whether there is a meaningful use of objects and by constructiveness and creativity in play. More formal tests of comprehension, which do not include speech, are available for a child who presents with speech difficulty; standardized tests are available which allow a quantification of the extent of the language impairment.

In profound deafness the child does not attend to, or respond to, auditory stimuli, but will extensively use gestures, attempt conversation when older, and may be constructive in play. The assessment of deaf children with psychiatric disorder is intimately related to the individual child's communication, which in turn is influenced by medical, social, and cultural factors.[30] The use of an interpreter can clarify communication.

The younger autistic child may not respond meaningfully to auditory stimuli, and, indeed, may tend not to respond to any form of verbal or non-verbal communication. There is little indirect evidence of language either in terms of gesture, mime, imitation, or play. In mental retardation there is usually no evidence of deafness, but rather of limited language abilities and usually a delay in the development of articulation, together with other evidence of slowness of other developmental milestones. Evidence of constructive play reflects the presence of inner language and augurs well for the growth of language in general. Gestural imitation and play constitute a form of communication, which is impaired in infantile autism and in severe language retardation.

Cognitive ability

Clinical impressions can be deceptive and need to be validated by careful psychometric assessment of both non-verbal intellectual skills and language performance. A wide range of tests are now available, which measure performance rather than verbal abilities and also language skills.

Intervention in speech and language disorders

The severity spectrum[4] outlined above provides a rough guide as to when to intervene. Mild disorders are likely to improve spontaneously provided that there are no associated psychological and relationship problems. The course suggests spontaneous improvement by the ability to use meaningful three-word phrases by 36 months, a pattern of normal development in all other aspects of language, communication, and general milestones, and no evidence of medical conditions such as hearing impairment, learning disorder, etc.[29] In these circumstances, treatment may be deferred to school age. However, irrespective of the mildness of the disorder, parents will wish to have specific information about prognosis and may prefer an earlier intervention. There is no evidence that such action in itself has negative effects, provided that pressure and coercion are avoided.

If the disorder is less mild, joint home and school intervention programmes are indicated, including counselling the parents about structuring the child's communication environment, combined with classroom teaching which focuses on the linguistic, social, and educational needs of the child.[3]

Severe disorders merit early attention by speech therapists and teachers. Routine and didactic teaching of language skills in a structured manner is now less fashionable.[3] It is considered preferable to complement structured teaching with a range of methods to stimulate language, including focusing on teaching the child communication skills within a naturalistic setting.[3] At the severe end of the spectrum, in children with significant auditory comprehension difficulties, there is a debate as to whether the child should be given preferential oral language training alone. This is because of concerns that supplementary signed language may become the preferred and only communication modality.[3] However, the evidence for this is scant and the current wisdom is that there should be a preparedness to employ multimodal approaches in intervention with such children, i.e. using oral and sign-language methods and, where necessary, appropriate behavioural techniques.

Psychological mutism

There are two forms of psychological mutism—traumatic and selective.[31,32] Both are dramatic and rare.

Traumatic mutism has an acute onset following a psychological shock or injury. Some consider it to be a hysterical phenomenon as it is not associated with any disorder of the structures subserving speech functioning (lips, tongue, palate, or vocal cords). Furthermore, the patient is able to cough normally. The literature suggests that it is common, but a wide clinical survey has attested to its considerable rarity.[32] **Selective mutism** is a fascinating disorder, where talking is confined to familiar situations, usually the home, and to a small group of intimates. The earliest manifestations are in the preschool years, with the parents being unaware of significant abnormality because there has been a period of relative competence in spoken language. Commonly, recognizable shyness is present from the early years of life and only in a small percentage are there indications that it emerges for the first time at a later stage in development.[31] It needs to be distinguished from inordinate shyness, which occurs relatively frequently in school reception classes, by its severity and persistence.[33]

Assessment

Enlisting the co-operation of the child for the purpose of psychological and psychiatric assessment usually constitutes a challenge, the bases of which are the complex emotional and attitudinal expressions accompanying the refusal to talk. Co-operation is best facilitated by adopting a style of communication that avoids expectation of the child to talk. In contrast, pressure is likely to intensify the child's resistance. Unobtrusive observations are invaluable sources of information about behavioural interactions and communication within the family, and can be achieved using one-way screens or family videotaped vignettes. For younger children, the provision of drawing and play materials as a medium for preliminary interaction is often helpful. Subsequently, standardized non-verbal measures are the main source of assessment, particularly among the older children.

Epidemiology

A rate of 0.8 per 1000 'core' selective-mute children was reported[1] in a total city cohort of 3300 7-year-olds, which is as common as broadly defined autism. In contrast, a rate of 7.2 per 1000 of children who do not speak at school soon after entry at the age of 5 was reported,[32] but a year later the rate had fallen to 0.7 per 1000. This 'persistent shyness' of the latter group is closely comparable to core selective mutism.[32] Even higher rates are recorded when broader criteria are used and where there is no associated clinical assessment.[34]

Diagnosis and differential diagnosis

Inordinate shyness and the reluctance to talk at school entry have proved to be transient phenomena,[33] which are likely to reflect normal separation anxiety compounded by transient adaptation reactions to the usual stresses and unfamiliarity of the new school situations. With exposure this anxiety diminishes and the child may begin to talk.[19] Thus a distinction needs to be made between such transient states of inordinate shyness and pathological behaviours by applying more rigorous criteria when defining selective mutism as being persistent, severe, and showing pathological shyness beyond the home situation.[32] ICD-10 captures the essential criteria well: 'characterised by a marked emotionally determined selectively in speaking...With language competence demonstrated in some situations but not in others...with a normal or near normal language comprehension-...with demonstrable evidence that the child can speak normally in some situations...the failure to speak is persistent over time'. A further issue concerns children who speak rather little in all circumstances, with the view that they should not be assigned the diagnosis of selective mutism, nor should those children who display 'reluctant speech', i.e. they do not speak spontaneously but may answer questions.

Developmental and biological factors

There is a consistent pattern of an excess of girls to boys, which is unusual for childhood disorders.[32,35] Kolvin and Fundudis[32] included a control group drawn from a general population sample. They reported an excess of birth complications and also found evidence of slow or uneven development compared with the controls, which included delayed onset of speech, excessive developmental mispronunciations, and associated problems of speech. Underlying speech or language delay or problem are reported to range from 20 per cent[35] to 50 per cent.[32] Recently Steinhausen and Judi[36] reported similar features, namely being more common in girls and seen in all social strata. Early developmental risk factors were also quite common as was a background of migration. School and unfamiliar people were common social concerns and comorbid psychiatric diagnoses were common.

Personality temperament and behaviour

Insidious development of shyness has been reported in over 80 per cent of cases, and even where it appeared acute the essential abnormality became dramatically obvious only when the child started school.[32] In addition, a wide variety of complex personality patterns often occur, such as oppositional behaviour and poor manageability both at school and at home.[32] A common personality pattern was of sulkiness combined with aggressiveness, with a child presenting as sulky to strangers and aggressive within the home. Many of these children are described as having powerful personalities, with 'wills of steel'. About a quarter showed a combination of shyness in social situations with submissiveness at home. Another quarter seemed to be rather sensitive children. In addition, there was an important trend for such children to be more withdrawn in relation to peers than to adults. Problem behaviours were described in over two-thirds of the children.[32]

Black and Uhde[37] studied 30 5- to 12-year-olds using parent- and teacher-rating scales and structured diagnostic interviews. They report that mutism varied according to the psychological setting. The majority of their subjects were diagnosed as social phobic or avoidant disorders (97 per cent) and simple phobic disorders (30 per cent). The severity of social anxiety correlated with the severity of the mutism, and they argue that selective mutism may be a symptom of social anxiety.

Cognitive aspects

The evidence available shows that although selectively mute children as a group cover most ranges of non-verbal intellectual ability, there is a distinct shift to the left. In general terms, when selectively mute children do speak—within the confines of their own homes—their vocabulary, verbal conceptualization of ideas, and grammatical structure of sentences are commonly normal.

Family and social factors

The families of selective mutes are represented at all socio-economic levels.[32] Of importance is the nature of the operant psychological dynamics. The literature abounds with examples of parents with unusual personalities and psychiatric problems, which are often offered as explanations for the selective mutism of their children.[58] The confidence placed in these findings must be limited by the lack of controls and small sample size of the studies upon which the theories are based. Some of the factors which were presumed causative include maternal rejection, maternal anxiety, fearfulness, and overprotectiveness, the influence of 'family secrets' and the child's fear of parental retaliation, and the effects of a symbiotic relationship between a parent and the child. Most of these notions derive from older publications.

In one controlled study,[32] one-third of the parents of selectively mute children were reported to have personalities characterized by serious or marked reserve and shyness or oddness; neurotic disorder was present in one of the parents in about one-sixth of the families, and depression was present in one of the parents, again in one-sixth of the families. Considerations of psychiatric disorder and major personality problems, in combination with serious marital disharmony, occurred in about 60 per cent of the families. However the disturbance in families is defined, the available evidence points to an excess of psychological morbidity compared with families of normal control children.[32] On balance, the origins appear multifactorial.

Another study reported a 75 per cent rate of parental psychological disturbance.[36] This higher rate was probably due to the use of a much broader definition of disturbance. Shyness in parents of selectively mute children appears to be fairly common,[32] and raises the interesting possibility of a familial or even a genetic link between shyness of the parents and selective mutism of the child. The latter possibility is enhanced by the finding of a number of affected siblings[35] or twins[39] in different series of selectively mute children. On the other hand, the influence of learning and modelling cannot be discounted.

More recent research exploring the parental perceptions of selective mutes as adults suggests important negative parental influences, with the compounding of poor affection and care with overcontrolling behaviour (S. Baharaki *et al.*, personal communication). Others imply that it is a learned pattern of behaviour. Yet others implicate temperamental or personality factors which have a familial basis. Or it 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; others implicate an important maturational component.

Finally, Kolvin *et al.*,[40] following the conceptualization of Stevenson-Hinde and Shouldice,[41] refer to the mother's poor sensitivity followed by child dysfunction in each of three behavioural systems—reflecting wary and fearful behaviour; inhibition in social situations; and insecure attachments. Another theme is stranger anxiety[42] represented by either avoidant freezing reactions in social situations or oppositional, silent, and quiet reactions. The notion of severe anxiety has been taken up by Dummit *et al.*[43] and by J. Morgan *et al.* (personal communication) who argue for an early onset of a social phobic condition. This diversity of presumed aetiological factors suggests that the origins are multiple and the condition heterogeneous.

Intervention and outcome

Selective mutism continues to be a challenge to psychodynamically and behaviourally oriented psychotherapists. Success rates are variable. It is difficult to offer general conclusions from major studies because of the differences in diagnostic criteria used, the severity of the mutism, and the criteria of improvement. For example, Wright[35] reported that almost 80 per cent of children achieve an 'excellent' or 'good' adjustment, whereas Kolvin and Fundudis[32] report an adjustment rate of 46 per cent. The literature emphasizes the intractability of selective mutism to psychotherapy, in that, although degrees of improvement are usually reported, the spontaneity of interpersonal communication of the selectively mute child is seldom fully shifted. Some now suggest that psychodynamic approaches have to vary according to the mutism subtype under scrutiny. For example, those who are more compliant will respond to longer psychodynamically oriented approaches; and those who are non-compliant are more likely to respond to group therapy.[42]

The emphasis of psychodynamic approaches has given way to behavioural strategies. In keeping with the more pragmatic stance of the behaviourists it has been argued that intervention should be directed not only at 'mutism' but more broadly at 'social skills.'[44] Consistent with the above is the proposal of a multidimensional management approach[45] consisting of the following:

- avoidance of strategies likely to put pressure on the child to talk
- inclusion of the child in small peer-group activities
- use of reading, story-telling, and other verbal activities which do not make the child feel especially uncomfortable
- parental encouragement for relatives and peers to visit the selectively mute child at home to create a natural social context of conversation without putting pressure on the child to talk
- encouragement of the child, within the classroom situation, to engage in non-verbal and non-threatening interpersonal relationships
- a gradual process of encouragement and involvement of the child in one-to-one situations, where appropriate stimuli are used as a means of creating the potential for verbal communication and then drawing one or two other children into the activity
- encouragement to engage in joint activities outside the home.

This broad-based behavioural approach seems attractive, but it merits more careful evaluation.

Inevitably, some clinicians have turned to pharmacotherapy spurred by notions of origins in anxiety and social phobia. Most studies are single case or have small sample sizes, without controls, with fluoxetine being favoured. One open trial, using a repeated measure design, treated 21 selectively mute children with a combination of behaviour therapy and fluoxetine.[46] There was a reduction of anxiety and improved ability to speak in 13, no change in 4, and the fluoxetine was discontinued in 4 because of an increase in oppositional behaviour. However, there was no control group. The efficacy of fluoxetine has been evaluated in a double-blind placebo-controlled study.[47] Here, 15 placebo non-responders were assigned to double-blind treatment with fluoxetine—significant improvement was reported in mutism, anxiety, and social anxiety. However, most subjects remained very symptomatic at the end of the study. Overall, the fluoxetine findings must be viewed as interesting rather than definitive, with improvement linked to the duration of the treatment and where the selective mutism is more widely defined.

References

1. Fundudis, T., Kolvin, I., and Garside, R. (1979). *Speech retarded and deaf children: their psychological development.* Academic Press, London.
2. Rapin, I. and Allen, D. (1983). Developmental language disorders: nosologic considerations. In *Neuropsychology of language, reading and spelling* (ed. U. Kirk), pp. 155–84. Academic Press, New York.
3. Bishop, D.V.M. (1994). Developmental disorders of speech and language. In *Child and adolescent psychiatry* (ed. M. Rutter, E. Taylor, and L. Hersov), pp. 546–68. Blackwell Science, Oxford.
4. Ingram, T.T.S. (1972). The classification of speech and language disorders in young children. In *The child with delayed speech (Clinics in developmental medicine no. 43)*, pp. 13–32. SIMP/Heinemann, London.
5. Lewis, M. (1968). Language and mental development. In *Development in human learning* (ed. A.E. Lunzer and J.F. Morris), p. 68. Staples Press, London.
6. Rutter, M. (1972). Clinical assessment of language disorder in the young child. In *The child with delayed speech (Clinics in developmental medicine no. 43)*, pp. 33–47. SIMP/Heinemann, London.
7. World Health Organization (1992). *International statistical classification of diseases and related health problems, 10th revision.* WHO, Geneva.
8. Bishop, D.V.M. (1985). Age of onset and outcome in 'acquired aphasia with convulsive disorder' (Landau–Kleffner syndrome). *Developmental Medicine and Child Neurology*, **27**, 705–12.
9. Stevenson, J. and Richman, N. (1978). Behaviour, language and development in three-year-old children. *Journal of Autism and Child Schizophrenia*, **8**, 299–314.
10. Rutter, M. and Mittler, P. (1972). Environmental influences on language development. In *The child with delayed speech (Clinics in developmental medicine no. 43)*, pp. 52–67. SIMP/Heinemann, London.
11. Lotter, V. (1966). Epidemiology of autistic conditions in young children. I—Prevalence. *Social Psychiatry*, **1**, 124–37.
12. Richman, N., Stevenson, J., and Graham, P. (1982). *Preschool to school: a behavioural study.* Academic Press, London.
13. Rutter, M., Mawhood, L., and Howlin, P. (1992). Language delay and social development. In *Specific speech and language disorders in children* (ed. P. Fletcher and D. Hall), pp. 63–78. Whurr, London.
14. Chomsky, N. (1969). *The acquisition of syntax in children from 5 to 10.* MIT Press, Cambridge, MA.
15. Lenneberg, E.H. (1967). *Biological foundations of language.* Wiley, New York.
16. Klaus, R.A. and Gray, S.W. (1968). The early training project for disadvantaged children: a report after five years. *Monographs of the Society for Research in Child Development*, **120**.
17. Beitchman, J.H., Nair, R., Clegg, M., Ferguson, B., and Patel, P.G. (1986). Prevalence of psychiatric disorders in children with speech and language disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, **25**, 528–35.
18. Baker, L. and Cantwell, D.P. (1982). Psychiatric disorder in children

with different types of communication disorder. *Journal of Communication Disorders*, **15**, 113–26.
19. Cantwell, D.P. and Baker, L. (1985). Speech and language: development and disorders. In *Child and adolescent psychiatry: modern approaches* (ed. M. Rutter and L. Hersov), pp. 526–44. Blackwell, London.
20. Rutter, M. and Lord, C. (1987). Language disorder associated with psychiatric disturbance. In *Language development and disorder* (ed. W. Yule and M. Rutter), pp. 206–33. Blackwell Science, Oxford.
21. McCormick, B. (1988). *Screening for hearing-impairment in young children*. Croom Helm, London.
22. Freeman, R.D., Malkins, S.E., and Hastings, J.O. (1975). Psychosocial problems of deaf children and their families: a comparative study. *American Annals of the Deaf*, **120**, 275–304.
23. Schildroth, A.N. and Hotto, S. (1993). Annual survey of hearing impaired children and youth 1991–1992. *American Annals of the Deaf*, **138**, 163–71.
24. Meadow, K.P. (1980). *Deafness and child development*. University of California Press, Berkeley, CA; Edward Arnold, London.
25. Conrad, R. (1977). The reading ability of deaf school-leavers. *British Journal of Educational Psychology*, **47**, 138–48.
26. Myklebust, H.R. (1964). *The psychology of deafness* (2nd edn). Grune and Stratton, New York.
27. Hindley, P.A., Hill, P.D., McGuigan, S., and Kitson, N. (1994). Psychiatric disorder in deaf and hearing impaired children and young people: a prevalence study. *Journal of Child Psychology and Psychiatry*, **35**, 917–34.
28. Rutter, M. (1977). Delayed speech. In *Child psychiatry: modern approaches* (ed. M. Rutter and L. Hersov), pp. 698–716. Blackwell Science, Oxford.
29. Rutter, M. (1977). Assessment of language disorders. In *Language development and disorder* (ed. W. Yale and M. Rutter), pp. 295–311. Blackwell Science, Oxford.
30. Roberts, C. and Hindley, P.C. (1999). Practitioner review: the assessment and treatment of deaf children with psychiatric disorders. *Journal of Child Psychology and Psychiatry*, **40**, 151–67.
31. Tramer, M. (1934). Electiver Mutismus bei Kindern. *Zeitschrift fur Kinderpsychiatrie*, **1**, 30–5.
32. Kolvin, I. and Fundudis, T. (1981). Elective mute children: psychological development and background factors. *Journal of Child Psychology and Psychiatry*, **22**, 219–32.
33. Brown, J.B. and Lloyd, H. (1975). A controlled study of children not speaking at school. *Journal of the Association of Workers with Maladjusted Children*, **3**, 49–63.
34. Kumpulainen, K., Raesaenen, E., Raaska, H., and Somppi, V. (1988). Selective mutism among second-graders in elementary school. *European Child and Adolescent Psychiatry*, **7**, 24–9.
35. Wright, H.L. (1968). A clinical study of children who refuse to talk. *Journal of the American Academy of Child Psychiatry*, **7**, 603–17.
36. Steinhausen, H.C. and Judi, C. (1996). Elective mutism: an analysis of 100 cases. *Journal of the American Academy of Child and Adolescent Psychiatry*, **35**, 606–14.
37. Black, B. and Uhde, T.W. (1995). Psychiatric characteristics of children with selective mutism: a pilot study. *Journal of the American Academy of Child and Adolescent Psychiatry*, **34**, 847–56.
38. Wergeland, H. (1979). Elective mutism. *Acta Psychiatrica Scandinavica*, **59**, 218–23.
39. Halpern, W.I., Hammond, J., and Cohen, R.A. (1971). A therapeutic approach to speech phobia: elective mutism re-examined. *Journal of the American Academy of Child Psychiatry*, **10**, 94–107.
40. Kolvin, I., Trowell, J., Lecouteur, A., Baharaki, S., and Morgan, J. (1997). The origins of selective mutism: some strategies in attachment and bonding research. *ACPP Occasional Papers*, **14**, 17–25.
41. Stevenson-Hinde, J. and Shouldice, A. (1993). Wariness to strangers: a behaviour systems perspective revisited. In *Social withdrawal, inhibition and shyness in childhood* (ed. K. Rubin and J. Asendorpf), pp. 101–16. Erlbaum, Hillsdale, NJ.
42. Lesser-Katz, M. (1988). The treatment of elective mutism as stranger reaction. *Psychotherapy*, **25**, 305–13.
43. Dummit, E.S., Klein, R.G., Tancer, N.K., Asche, B., Martin, J, and Fairbanks, J.A. (1997). Systematic assessment of 50 children with selective mutism. *Journal of the American Academy of Child and Adolescent Psychiatry*, **36**, 653–60.
44. Kratochwill, T.R., Brody, G.H., and Piersel, W.C. (1979). Elective mutism in children. In *Advances in clinical child psychology* (ed. G.B. Lahey and A.E. Kazdin), pp. 193–239. Plenum Press, New York.
45. Friedman, R. and Karagan, N. (1973). Characteristics and management of elective mutism in children. *Psychology in Schools*, **10**, 249–52.
46. Dummit, E., Klein, R.G., Tancer, N.K., Asche, B., and Martin, J. (1996). Fluoxetine treatment of children with selective mutism: an open trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, **35**, 615–21.
47. Black, B. and Uhde, T.W. (1994). Treatment of elective mutism with fluoxetine: a double-blind, placebo-controlled study. *Journal of the American Academy of Child and Adolescent Psychiatry*, **33**, 1000–6.

9.2.12 Gender identity disorder in children and adolescents

Richard Green

Variance in psychosexual development

Psychosexual development of sex-typed behaviours spans a broad mix of the elements that comprise 'masculinity' and 'femininity'. The possibility for permutations and combinations is rich. Thus contemporary discussions talk of masculinities and femininities throughout the lifespan, rather than masculinity and femininity. Among males, there are boys and men whose stereotypical masculinity may pose problems in mental health and criminality. They are not the focus here. Rather, here it is the marked deviation from the mean towards the 'non-masculine' or 'feminine' extreme. That pattern can also cause clinical concern and constitutes gender identity disorder (**GID**) as manifested in childhood. Further, conventional 'tomboyism' in females is not the focus here, but rather the extreme that can cause clinical concern and constitute GID.

Epidemiology

No epidemiological studies exist of GID in children. Prevalence can be estimated only roughly from indirect sources. Two items on the Child Behavior Checklist[1] are consistent with components of the diagnosis. They are 'behaves like opposite sex' and 'wishes to be of opposite sex'. Among 4- to 5-year old boys, not clinically referred for behavioural problems, about 1 per cent of parents answer in the affirmative that their child 'wishes to be the opposite sex'. For ages 6 to 7 it drops to near zero, but rises to 2 per cent at age 11. For girls, the highest rate was 5 per cent at ages 4 to 5, but less than 3 per cent for other ages. With respect to 'behaves like opposite sex', among the boys the rate was about 5 per cent and among girls about 11 per cent for all ages. However, these data do not indicate any longitudinal aspect of the reported behaviour, and do not detail the behaviour.[2]