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review

Sensory impairments, intellectual disability and psychiatry

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Abstract

The present review looks at: (1) prevalence studies of sensory impairments in people with intellectual disability (ID); (2) studies looking at psychological and psychiatric disorders in people with sensory impairments; and (3) studies that have examined the association of sensory impairments with autism. Research has indicated that sensory impairments are more common in people with ID. Psychiatric disorders are believed to be more common in children with visual impairment (VI) when associated with other handicaps. Some authors believe that hearing impairment (HI) can result in personality disorders. Studies have also shown a higher prevalence of psychiatric disorders in children with HI and a higher incidence of deaf people in psychiatric hospitals than in the general population. Psychiatric disorders in children with HI are particularly associated with low IQ and low communication ability, especially in those with multiple handicaps. There is little evidence for a higher incidence of schizophrenia in people with HI. Blind people demonstrate many autistic-like features and there has been discussion in the literature as to their cause. Deaf people also demonstrate some similar features to those in autism, but an association with autism has not been conclusively made. Deaf-blind people commonly demonstrate problem behaviour (e.g. self-injury). Usher syndrome, which is the most common cause of deaf-blindness, is associated with psychiatric disorders, particularly psychosis. The need for assessment of sensory functioning in people with ID, the difficulties inherent in this and the need for specialist services is stressed.

Keywords autism, mental illness, sensory impairment

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Introduction

People with intellectual disability (ID) can be defined as those who have an IQ of 70 or below, concurrent deficits or impairments in adaptive behaviour, and the onset of their impairment before 18 years of age (American Psychiatric Association (APA) 1994).

Intellectual disability can coexist with other handicaps; for example, cerebral palsy, epilepsy, psychiatric illness and sensory impairment. The latter can occur to varying degrees and may involve multiple senses. Sensory impairment may be

overlooked, particularly in those with a more severe ID and inadequate communication skills. Carers and professionals may interpret behavioural symptoms as part of the individual's ID rather than recognizing an additional disability (diagnostic overshadowing). Training carers and staff to recognize possible signs of a sensory impairment is valuable (Yeates 1991). This should be followed by accurate assessment by specialist services. However, the latter is not always available (Yeates 1991).

The combination of sensory impairment and ID can result in the individual being more vulnerable to developing behavioural problems and psychiatric illness. Children with such multiple handicaps can also be affected by the reaction of their parents and colleagues to their impairments. A carer needs to feel confident in their own ability to look after a child with multiple handicaps. In addition, their expectations for the child's development and growth need to be realistic for a healthy relationship.

The following paper is a review of studies that have looked at the subject of people with ID who have associated sensory impairments. The review particularly looks at the prevalence of such disabilities, and the associations found with behavioural and psychiatric disorders including autism.

Method

An electronic search was made for all relevant papers. The keywords used were: mental retardation, sensory impairment, visual impairment, hearing impairment, deaf, blind, deaf-blind, mental illness and psychiatry. Medline, Embase and Psychlit were searched. In addition, a manual search of appropriate journals was also made. Relevant references cited in papers were also included.

Results

Epidemiology

Visual impairments

Various studies over the years have looked at the prevalence of visual impairment (VI) in people with ID (Table 1). The prevalences found partly depend upon how VI is measured, i.e. whether by testing or

Table 1 Prevalence studies of visual impairment (VI): (ID) intellectual disability

Reference	Population	Sample number	Method	Prevalence	Refractive errors
Warburg (1970)	Two ID institutions	706	Visual testing	5% blind; 7–8% partially sighted	–
Cullinan (1977, 1978)*	General UK population	–	Questionnaire survey	0.52% visual disability	–
Warburg *et al.* (1979)*	Children with ID in Denmark	7700		4% severe VI	–
Ellis (1982)	Residents of ID institutions in England and Wales	30020	Questionnaire survey	3.6% blind; 4.4% partially sighted	–
Tielsch *et al.* (1990)	General USA population		Questionnaire	0.1% severe; 1.5% moderate	34%
Wilson & Haire (1990)	Day centre in Nottinghamshire	75	Visual testing	50.8% moderate VI; 13.8% blind	–
Warburg (1994)	Residents' workshops and day centres (ID) in Copenhagen	778	Visual screening	1.6% severe VI; 10% moderate VI	–
Evenhuis (1995)	Old age residents' ID institutions in the Netherlands	70	10-year prospective study†	27.9% moderate–severe VI	63.5%

*Cited in Ellis (1986).

†Measures of visual function and intra-ocular pressure.

questionnaire. In addition, the variation in definitions used for visual disability confounds the picture. Questionnaire studies can be flawed in that they rely largely on subjective staff reports rather than actual measurements. In one study (Warburg 1994), estimates of vision by caregivers were compared to clinical assessments and concordance was found in only 32%. Despite this, questionnaires are effective in screening large populations to identify prevalence. Testing, although more accurate, can only be done on small numbers at a time. Prevalence rates will also vary depending on how the visual function was tested.

Survey rates can be compared to Cullinan's (1977, 1978; cited in Ellis 1986) results of VI in the general population in the UK, where a prevalence of 0.52% was found. Looking at the extremes of prevalence found in populations with ID (Ellis 1982; Wilson & Haire 1990), the rates found are between 8.5 and 200 times that of the general population.

Evenhuis (1995) completed a 10-year prospective study looking at elderly patients. She found that visual function varied according to the level of ID in that those with mild ID had a better mean visual function than those with moderate or severe ID. She also found that 13.1% of the group deteriorated in their visual function over the 10 years as a result of cataracts, macular degeneration and glaucoma.

In Warburg's (1994) study, the most frequent causes of VI were excessive myopia, cortical VI, optic atrophy, cataract and keratoconus. Optic atrophy was the most common cause amongst children. Evenhuis (1995) found that cataracts and hyperopia were the most common conditions causing the VI.

Hearing impairments

Deafness has been defined as, 'that which with the best hearing aids available does not allow understanding of speech through the ears well enough to take part in a brisk conversation' (Basilier 1972). Deafness or hearing impairment (HI) can further be categorized by a number of factors such as the age of onset (especially whether pre- or post-lingual), whether bilateral or unilateral, the frequency of tones affected (i.e. high tone, low tone, flat or U-shaped), and the severity of the impairment.

Both the incidence and the prevalence of deafness in the general population have been found to be about one per 1000 (Kitson & Fry 1990). For people with ID, there has been much research studying rates of recognized and unrecognized HI (Table 2). As with VI, frequencies partly depend upon how the HI is measured, i.e. whether by testing or questionnaire. A study by Lavis *et al.*

Table 2 Prevalence studies of hearing impairments: (ESN) educationally subnormal; and (HI) hearing impaired

Reference	Population	Sample number	Method	Prevalence
Kropka *et al.* (1984)*	ID institutions in England and Wales	18 657	Questionnaire survey	3.4% deaf; 7.3% partially deaf
Denmark & Adams (1982)*	ID institutions in England, Scotland and Wales	–	Postal survey	3.6% deaf; 4.2% partially deaf
Murphy (1978)*	ESN schools in England and Wales	–	Postal questionnaire	8–13% HI
Yeates (1989, 1992, 1995)	ID population in Lewisham and North Southwark	500	Hearing tests	40% HI (80% over 70 years old)
Evenhuis (1995)	ID institutions in the Netherlands	70	10-year prospective study	50.9% HI

*Cited in Ellis (1986).

(1997) found widely differing rates for HI of 38.9% and 9.4% for testing and questionnaire, respectively.

Services. Hearing tests for people with ID are known to be problematic (Wilson & Haire 1990; Yeates 2000). Adult assessments are usually the responsibility of the local ear, nose and throat service. However, busy generic audiological services are often not geared to allow the extra time required for people with an ID. Yeates & Moorey (1996; cited in Yeates 2000) have proposed that the optimal method would be to establish a special service for people with ID. This would comprise a team of people skilled in both audiology and ID using performance tests and audiometry. This would then avoid more extensive testing such as the use of brain stem evoked responses which require a general anaesthetic in people with ID (Yeates 1991).

Yeates' (1989, 1992, 1995) survey of the ID population in Lewisham and North Southwark found that only 20% had normal hearing. It is interesting to note also that 70% were able to cooperate with subjective testing. The types of hearing loss identified were sensori-neural in 30%, mixed in 29%, conductive in 10% and unknown in 31%. The most common diagnosis in those found to have a HI was Down's syndrome (DS). Nearly half of the group with DS (47%) had a HI. It appeared from the results that people with DS are prone to developing a high-frequency hearing loss in early middle life, similar to that seen in presbyacusis. Yeates (1989, 1992, 1995) concluded from her study that screening for HI is essential in all people with ID. Ideally, this should be carried out on admission to the adult service and in old age, in addition to middle life testing for people with DS.

Diagnostic 'overshadowing'. It is believed that HI and ID together are multiplicative rather than additive, i.e. a HI may impede learning which will then increase the level of ID which, in turn, lowers the ability to cognitively process auditory input and so on. Trying to tease these diagnoses out may prove challenging, but is essential in order not to miss a HI in people with ID or to misdiagnose ID in a person with a HI and a normal IQ (Table 3). Denmark's (1966, 1969, 1985) study of deaf patients revealed that their poor language skills had been the factor accounting for the misdiagnosis.

For many people with profound deafness, sign language will become their means of communication, either solely or in conjunction with lip reading and/or spoken speech. In the UK, there are currently 500 000 users of British Sign Language (BSL). Sign language has been described as, 'highly expressive; can express essentially anything that spoken language can' (Sachs 1990). It has also been shown that, despite its dependence on spatial awareness, it is treated by the brain as a language, being processed by the left hemisphere as opposed to the right (Sachs 1989). This distinctive language has served to give deaf people a sense of their own community and culture. This is marked by using a small 'd' for audiological deafness and a capital 'D' for Deafness as the term for their community. However, membership of the community has been described as being achieved rather than ascribed, and involves identifying with the community and acting within the norms (Meadow-Orlans & Erting 2000). Users of American Sign Language have a sign to designate themselves which in written English equates to 'Deaf-world' (Lane *et al.* 1996).

People with both ID and HI frequently do not have an opportunity to identify and participate in Deaf culture. Acknowledgement of and response to

Table 3 'Diagnostic overshadowing' in the populations with intellectual disability (ID) and deafness: (HI) hearing impairment

Reference	Population	Sample number	Those misdiagnosed as 'subnormal'
Denmark (1966)	Deaf residents of psychiatric hospitals	–	7
Kropka (1979)	Residents with HI in Devon ID hospitals	100	15%
Kropka *et al.* (1984)	ID institutions in England and Wales	18 657	5% deaf (no ID); 3% partial hearing

their HI may not be made. As a result, such individuals are often subsumed under the ID service culture largely because of a lack of community sensory impairment workers.

Deaf-blind

The incidence of people affected by both HI and VI is far greater than would be expected if the relationship was coincidental. This is because a number of conditions will cause extensive neurological damage resulting in a number of disabilities, including ID. Causes include genetic anomalies (e.g. Usher syndrome), anoxia at birth, prematurity, low birth weight and infections (e.g. cytomegalovirus and rubella).

Usher syndrome is the most common cause of deaf-blindness. This is an inherited condition with an autosomal recessive pattern. The deafness, which is congenital, is usually severe but remains stable. This is associated with a progressive loss of vision. The initial symptoms of the latter are usually night-blindness, extreme sensitivity to light and/or tunnel vision. There is a large variability in the progression of the visual deterioration. Some children may be registered as blind, whereas others retain reading vision all their lives. In the USA, estimates of prevalence rates have indicated that about three per 100 000 of the population suffers from the disorder. About 25% have an ID.

Congenital rubella is another common cause of deaf-blindness. Rubella (meaning 'red') is a virus which normally affects children between the ages of 4 and 9 years. It causes an indistinctive rash, fever, malaise, sore eyes and enlarged cervical lymph glands. Although trivial in a child, rubella in a pregnant woman can be disastrous as a result of the infection passing to the foetus. The greatest damage is done in the first 9 weeks of pregnancy with multiple defects in over 90% of infants. These include cataracts, cardiac defects, inner ear damage, retardation of growth, muscular hypotonia or spastic paresis, ID, inflammatory lesions of brain, lungs and bone marrow, and possibly death. In a child born with congenital rubella syndrome, the virus remains and will be excreted for up to 2 years. In the UK, rubella vaccination of schoolgirls began in 1970. In 1986–1988, over 500 women were found to have rubella infections in pregnancy and out of those who went to term, 46 children had congenital rubella syndrome (Miller & Bradley 1990). Visual impairments in congenital rubella usually result from cataracts, but in addition, glaucoma, microphthalmia, retinopathy and nystagmus can develop as well (Van Dijk 1982). Some evidence suggests that further visual deterioration can occur as the child grows. Hearing loss is reported in over 70% (Miller & Bradley 1990). Both visual and hearing impairments are found in 20–25% of rubella children (Van Dijk 1982). A review by Trybus (1985) found congenital rubella to be the most common cause of deaf-blindness.

Ellis (1982) looked at the UK ID registers. He found details of 40 deaf-blind subjects. Up to one-third of those with hearing handicaps also had visual handicap. A nation-wide survey in the USA (Jensema 1980) looking at the deaf-blind population found that over 60% had IQs below 50. The 'SENSE' survey (Best 1983) identified 288 children with multiple sensory impairments being educated in schools for those with severe ID More recently, it has been estimated that one in 10 000 school children in the UK are deaf-blind, a large proportion of whom will also have ID (Bond 2000).

Psychiatric disorders

Visual impairment

Most studies agree that behavioural and developmental problems are commoner among blind and visually impaired children than those with normal sight (Jan *et al.* 1977). Psychiatric disorder is significantly more common in children with multiple handicaps and in children in whose central nervous system (CNS) is damaged. The behaviour problems are thought to arise as a result of a number of interactions including psychological, developmental, attitudinal and social factors (Jan *et al.* 1977).

As infants, the block to exploring and discovering the world around them presents a major developmental disadvantage. Unlike their sighted peers, infants with VI fail to begin to reach out for objects (Warren 1986). Blind children with ID are more likely to have motor handicaps (Warburg 1982). This further impedes their ability to explore. Fraiberg (1977) has observed that the concept of distinction between oneself and an object or

another person (the recognition of 'I'), which is normally present by the age of 2 years, is delayed in blind children until as much as 5 years. This equates to the sensorimotor period in Piaget's stages of cognitive development where sight is a critical factor. The sensory deprivation and possible social isolation which blind children experience can lead to self-stimulatory behaviours (Warren 1986). These are similar to those seen in people with a profound ID. They are often referred to as 'blindisms' and include eye pressing, light gazing and flicking fingers in front of lights, and motor stereotypies (e.g. rocking, spinning, tapping and twirling). The presence of the various mannerisms can give an indication of the type of visual loss (Jan & Groenveld 1993). Eye pressing occurs in young children with severe ocular visual loss, especially retinal disorders, but not in cortical visual impairment (CVI). IN rare cases children with multiple handicaps, including CVI, poke their eyes from the side. The reason for this is unknown, but they are recognized to exhibit other self-abusive behaviours (Jan & Groenveld 1993). Eye pressing occurs in those with bilateral severe ocular loss and is self-stimulatory in nature (Jan & Groenveld 1993). Prolonged light gazing is usually seen in those with cortical visual disturbance and is seen in about 60% of those with CVI although it does not reflect its severity (Jan *et al.* 1990). Fascination with lights and hands before eyes is also a recognized feature in rubella children (Van Dijk 1982). Rocking and other motor mannerisms are seen in those children who are believed to require more activities and movement (Jan & Groenveld 1993). Stereotypies can delay more functional use of vision and can interfere with learning new skills so are important to tackle early (Miller & Bradley 1990).

Prescott (1976) has argued that these stereotypies reflect the amount of somatosensory stimulation. He believed that a visual defect can result in the child being deprived of somatosensory experience. This deprivation could result in the cerebral and cerebellar cortexes becoming 'supersensitive' or 'hyperactive', leading to behavioural consequences. Adelson & Fraiberg (1976) found that stereotypies in blind children were reduced through sufficient somatosensory stimulation.

As well as being delayed in their concept of self, blind children may develop imaginative play at a later stage than sighted children (Kitson & Thacker 2000). This, combined with a preference to be left alone, may lead them in adult life to appearing unmotivated and 'schizoid'. Therefore, assessing the mental state of a blind person may present with difficulties (Kitson & Thacker 2000).

Hearing impairment

The impact of deafness appears to depend on the age of onset and degree of impairment so that in those born with profound deafness the impact will be the greatest. Sachs (1989) has commented that, in the infant, motor skills are developed naturally by oneself, whereas language 'is impossible to acquire . . . without some essential innate ability . . . [which] . . . is only activated by another person who already possesses linguistic power and competence.' The idea of an 'innate' ability is derived from Chomsky (1968). He proposed that all new-borns have a latent neural structure or Language Acquisition Device that is kindled by exposure to language from around 21 months until it diminishes in capacity at around 12 years of age. Sachs (1989) also added 'that if communication goes awry, it will affect intellectual growth, social intercourse, language development, and emotional attitudes'. For a deaf child born to hearing parents (90% of cases), oral communication will, initially at least, be their only means of communicating with their child. Comparisons between deaf children born to deaf parents as opposed to hearing parents have shown the former to be superior in their educational achievements (Fundudis *et al.* 1979).

Personality. Various authors have looked at personality attributes of deaf people in the past. Findings such as social and emotional immaturity, egocentricity, lack of tact and empathy for others, as well as labile and explosive natures have been described (Denmark 1966). Basilier (1964) went on to name this personality type as 'surdophrenia' which literally means 'deaf mind'. Meadow (1981) believed that the incidence of behaviour disorders including personality disorders is at least three times that found in the hearing population. This stereotype of deaf people has been criticized and a review has been made of the literature on the psychology of the deaf (Lane *et al.* 1996). It was found that studies

consistently emerged with negative attributions for the deaf. In addition, many serious flaws in the studies were found, related to test administration, language, scoring, subjectivity, reliability, validity, content, norms and population characteristics. Bond (2000) has stressed that any problem behaviours seen in those with HI are not a result of the HI *per se*, but a consequence of other factors leading from the HI such as the environment and the reaction and interaction of others.

Children. For the deaf, the child–parent communication ability appears to be a critical factor in their early development and psychological health (Meadow *et al.* 1983). For deaf children born to deaf parents, communication is less likely to be a problem. Such parents appear better at interacting with their children (Hindley 2000). However, in the deaf child born to hearing parents, attachment behaviour can be delayed and threatened altogether. This is believed to be as a result of a number of factors including inequalities in communication, unresolved grief and the practicalities of dealing with a deaf child (Hindley 2000). Deaf children born to hearing parents have also been described as having more behaviour problems (Stokoe & Battison 1975), being less happy, enjoying interaction with their mother less, and being less compliant and less creative than the more communicatively able deaf child (Schlesinger & Meadow 1972). In addition, it has been observed that behaviour problems can enhance communicative difficulties, creating a vicious circle (Schlesinger & Meadow 1972).

Some studies have suggested that behaviour disorders are more of the conduct type rather than emotional (Fundudis *et al.* 1979). When looking at other risk factors for developing psychiatric disorders, the degree of deafness does not appear to be related (Hindley 2000). However, findings have been inconsistent when looking at schooling (Hindley 2000), but major problems have been described as occurring sometimes on leaving school and this is a common time for referral to the psychiatric service (Meadow 1975). Interaction and abuse within families is also an important factor for mental health. Problems such as marginalization, scapegoating, and physical and sexual abuse have been described (Hindley 2000).

Behaviour and emotional problems are particularly common in children with multiple handicaps (Vernon 1969; Jensema & Trybus 1975). Low IQ has been found to correlate with behaviour problems in deaf children (Schlesinger & Meadow 1972), and as with less handicapped children, low communication ability has been found to be significantly associated with psychiatric disorder (Sinkkonen 1994). These findings are to be expected since other handicaps such as impairments of learning or vision, or other CNS damage, are risk factors for psychiatric disorder in their own right (Graham & Rutter 1968; Hindley 2000). In addition, deaf children are more likely to experience emotional, physical and sexual abuse than hearing children (Hindley 2000).

Hindley (2000) has reviewed the studies that have looked at psychiatric disorders in deaf children.

Adults. Some conflicting reports have emerged as to whether mental illness is more prevalent in the deaf population or not (Lane *et al.* 1996). Mahapatra (1974) looked at 89 patients with post-lingual deafness and found a higher prevalence of psychiatric illness among these individuals, with depression being the most common diagnosis. Rainer & Altshuler (1972) found low rates of depressive illness and obsessional disorders, whereas Kitson & Fry (1990) found obsessional and ritualistic behaviour to be common. However, the latter authors found no significant increase in mental illness. In their survey of state hospitals in the USA, Altshuler & Sarlin (1963) found that schizophrenia was no more common in the deaf. Similarly, in a review of deafness and psychiatric illness in the elderly, Cooper *et al.* (1976) found that the prevalence of schizophrenia in the pre-lingual deaf was not raised.

Despite this, there have been reports of deaf people having longer lengths of stay in psychiatric hospitals and receiving less treatment than other patients (Lane *et al.* 1996). Denmark (1966, 1969, 1985) found a higher incidence of deaf people compared to the general population in psychiatric hospitals (0.52% versus 0.1%, respectively). It is felt that this is likely to be a result of staff knowing too little about Deaf language and culture with misdiagnosis a consequence (Lane *et al.* 1996). Kitson & Fry (1990) believed that grammatical differences

between BSL and English may lead inexperienced clinicians to come to false conclusions about the written thoughts of deaf patients. Thought disorder can be wrongly diagnosed. However, it is acknowledged that the diagnosis is very difficult in pre-lingual profoundly deaf people even when they are examined by a psychiatric team skilled in manual communication (Critchley *et al.* 1981).

For deaf people who do develop psychosis, it has been found that 'mental deficiency' is often also present in those with organically caused deafness (Rainer & Altshuler 1972). In addition, for deaf people with schizophrenia, it is believed that the illness is less responsive to antipsychotics, may require higher doses and often needs combining with mood-stabilizing drugs (Kitson & Fry 1990).

Post-lingual deafness. The onset of post-lingual deafness may affect people in different ways psychologically. Factors that can influence the reaction include the age of onset, the speed at which the hearing loss progresses, the degree of impairment, personality, life situation and interests, and the possibility of alleviation and support. Goldberg (1975) noted that the presence of deafness at 16 years creates a higher risk for emotional disturbance. Mykleburst (1964) reviewed 127 autobiographical accounts of people who had become deaf. Social isolation was markedly apparent in the accounts.

Some groups of people with ID are at risk of developing HI later in life, such as those with DS. In the latter, their susceptibility is related to abnormalities of the outer, middle and inner ear as well as a predisposition to ear infections and otitis media (Holland 1986). Others who have had HI from birth may experience deterioration in their hearing later in life. For those lacking the communicative skills to highlight their distress, behavioural problems may result. The importance of hearing checks at critical life stages in these people has already been stressed.

Deaf-blind

The combination of deaf-blindness and an ID means that language and communication skills are often severely limited or absent. In addition, self-help skills and ability for abstract thinking may be lacking (Hindley & Brown 1994). Bond (2000) has commented that children with these multiple handicaps may become unresponsive to traditional reinforcers such as food, warmth, love, communication and interaction. Their ability to interpret the erratic and somewhat unrelated information being presented to them may give rise to an unfavourable perception of the environment. Carers may find the child unrewarding from their apparent lack of responsiveness. As a result, attempts by the child to communicate may be missed and only behaviours indicating distress may be attended to, thus reinforcing them (Bond 2000). The development of problem behaviours may then lead to a breakdown in relationships and placements, for example. This then compounds the problem as the individual will feel rejected, anxious and may possibly become depressed. In addition, parents may find it difficult to accept their child with multiple handicaps, leading to rejection and a failure to bond. Children with multiple handicaps will often have a number of specialists involved and their condition may necessitate numerous hospital admissions and procedures (Hindley & Brown 1994; Bond 2000). As for those who are deaf or blind, the environment is very important. It has been noted that those who are under-stimulated may become passive and disinterested, whilst those who are over-stimulated may become overactive (Hindley & Brown 1994).

In Usher syndrome, the most common cause of deaf-blindness, psychiatric disorders have been found to be common. It has been found associated with 5% of psychosis among the deaf (Rainer *et al.* 1963). Hallgren (1959) found that, out of 114 cases diagnosed with Usher syndrome, 23.3% ($n=26$) were psychotic. Sixteen presented with a schizophrenia, three suffered depressive reactions, two had psychotic episodes and three had been hospitalized because of aggression and agitation. Many reported auditory hallucinations. On average, the age of onset of the psychosis was later than is the case for the onset of psychosis in the general population. Twenty-three out of the 26 subjects also had ID. In the total cohort of 114, 23.8% had ID. Another study looked at 117 individuals with Usher syndrome and found 35 suffered from 'psychological disabilities'. Eleven of these subjects had ID.

Congenital rubella syndrome, another common cause of deaf-blindness, also has behaviours and problems associated with it. Very young rubella

babies have been reported as not enjoying cuddles or being held, and may arch themselves away from their mother. This can lead to frustration and resentment on the mothers' part (Van Dijk 1982). Eating and sleeping problems have been reported in young children. In later life, their vision and hearing may deteriorate, and this may lead to behavioural problems which can be misdiagnosed. Young adults with congenital rubella have been found to have a high incidence of diabetes mellitus and thyroid problems (Miller & Bradley 1990). Diabetes in itself can cause further visual deterioration. In addition, both of these conditions can give rise to psychiatric disorders or symptoms that may mimic them.

Summary

Psychiatric evaluation of patients with sensory impairments is not easy, especially when there is an added ID. For those with HI, it is important to have an interpreter of sign language or manual alphabet who is capable of translating the patient's feelings and thoughts. However, problems can arise in the translation from BSL to English. Evaluation of thought processes can be difficult and it is generally easier to evaluate disturbances of mood rather than thoughts. For those individuals with HI and severe to profound ID, all forms of communication must be considered, employing carers as interpreters. When no communication skills are present, careful and close observation of behavioural manifestations is the only means of examination. When evaluating appearance and general behaviour, the examiner should be aware of the mannerisms and stereotypies often seen in blind and deaf-blind people. Repeated examinations are necessary as well as reports from several observers and relevant background history (including family).

Autism

Visual impairment

Various studies have looked into the association of VI and autism (Table 4). Some have looked at specific diagnoses and others have examined all causes of blindness.

As part of a research programme looking at childhood schizophrenia, Keeler (1958) looked at referrals of children to the Hospital for Sick Children in Toronto, Canada. He found that the five children who were blind secondary to retrolental fibroplasia (RLF) had the most strikingly similar picture to infantile autism. Their features included self-isolation, delayed language, echolalia, a lack of use of language for interpersonal communication, self-referral in the third person, indiscriminate response to people, rhythmic rocking and preoccupation with music, preference for noisy or mechanical toys, smelling and tapping the environment, unusual gait, and 'blindisms' (i.e. hands/fingers over eyes and ears). To varying degrees, their mothers, had 'rejected' all of these children. Keeler (1958) then looked at 35 more children with RLF registered with the Canadian National Institute for the Blind and found very similar histories and behaviour, although the deviations were less marked in the second group. He compared these to 18 children who were congenitally blind from other causes and 17 children with postnatal blindness. The former of these groups showed some tendency to be withdrawn, but did not demonstrate the same degree of autistic behaviour. Abnormalities were least conspicuous in the postnatal group. He suggested that the autism resulted from a combination of brain damage, blindness and emotional deprivation, and believed that if such children received 'special type of favourable mothering care and psychological environment . . . this syndrome would not occur, at least not to such a degree'.

Another study looking specifically at RLF and autism was done by Chase (1972). She looked at 246 individuals with RLF. Chase (1972) found a gradient of autistic-like symptomatology in the group, but none with a clear diagnosis of infantile autism. She also found a strong relationship between autistic-like symptoms, and the presence of neurological findings and/or conditions.

Elk *et al.* (1998) looked at a group with retinopathy of prematurity (ROP, previously termed RLF) and compared them to a group with congenital blindness resulting from hereditary causes. Fifteen out of the 27 subjects in the former group were diagnosed as having an autistic spectrum disorder and four as having an autistic-like disorder (i.e. showing five or more of the DSM-IV criteria). Only two out of four subjects in the hereditary group were found to have autistic disorder. All of the children diagnosed as having autistic disorder also had

Table 4 Studies of autism in subjects with blindness: (RLF) retrolental fibroplasia; (CB) congenital blindness; (PNB) postnatal blindness; (LCA) Leber congenital amaurosis; (VI) visual impairment; (ROP) retinopathy of prematurity; and (HB) hereditary blindness

Reference	Subjects	Method	Findings
Keeler (1958)	5 children with RLF (referred) 35 children with RLF (from register) 18 children with CB 17 children with PNB	Developmental history and behaviour assessment	RLF: 5 with autism; 35 with partial autism CB: no autism PNB: no autism
Chess (1971)	243 pre-school children with rubella	Behavioural histories and direct examination	10 with autism 8 children with partial autism
Chase (1972)	246 children with RLF	Parent interview Professionals ratings Medical histories Rimland checklist E-2	Gradient of autistic-like symptoms None with autism
Fraiberg (1977)	27 children with blindness	Review of medical findings and birth histories Behaviour assessment and examination (in some)	7 with autism; remainder with 'autistic patterns'
Jan *et al.* (1977)	92 children with congenital VI		3 with 'psychosis'
Rogers & Newhart-Larson (1987)	5 children with LCA 5 children with CB from other causes	Reynell–Zinkin Scales of Development CARS* and ABC‡ DSM-III criteria for infantile autism	All 5 children with LCA had autism Comparisons had no autism
Brown *et al.* (1997)	24 children with CB	Teacher reports Direct behavioural observation Comparison of: (a) 15 blind and 10 sighted children (IQ > 70) (b) 9 blind and 9 autistic children (IQ < 70) CARS* and BCDP† DSM-III-R diagnosis	(a) Number of autistic features more common in blind group (b) Substantial overlap in presentation
Elk *et al.* (1998)	27 children with ROP 4 children with HB	IQ, behaviour and developmental assessment Developmental history review of medical records CARS* DSM-IV diagnosis	ROP: 15 with autism; 4 with autistic-like disorder HB: 2 with autism
Hobson *et al.* (1999)	9 children with CB 9 sighted children with autism	CARS* BCDP† DSM-III-R	None of the children with CB 'classically autistic'

*Childhood Autism Rating Scale.
†Behaviour Checklist for Disordered Preschoolers.
et al. Autism Behaviour Checklist.

ID. Nearly all those who had only two or three symptoms of autism demonstrated stereotyped behaviour, but showed relatively normal reciprocal interaction and communication. The above authors concluded that the autism was linked to the cerebral damage. They postulated that the blindness contributed to and intensified the autistic symptomatology. They considered blindisms as adaptive behaviour that could easily be falsely interpreted as autism.

Chess (1971) gathered behavioural data on 243 rubella children. Ten children were assessed as having Kanner autism and eight as having partial autism. Note was made of particular differences between the autistic and non-autistic groups. The autistic children tended to use other sensory modalities less and did not respond to contact in the same way. The latter also showed fewer gestures and initiatives to communicate. There was no evidence for a rejection by the families of the autistic group, and no differences between the mothers of this and the non-autistic group. Chess (1971) believed that the common component accounting for the autism was brain damage.

Fraiberg (1977) looked at a group of 27 blind children who had been referred for a 'guidance service'. She found that seven presented 'with a clinical picture that closely resembled autism'. Behaviours included stereotyped hand movements, rocking, swaying, mutism, echolalia and general withdrawal. Most of the remainder showed some form of stereotypy and mannerism, but were described as being different from the others in having attained a sense of 'self' and 'I'. In both groups, the mothers were described as 'depressed'. There was no evidence of neurological impairment in any of the subjects. Fraiberg (1977) believed that it was possible that either central impairment or 'gross impoverishment in the stimulus nutriments for early sensorimotor organisation' could produce autism.

Brown *et al.* (1997) compared three groups of children. Twenty-four congenitally blind children were identified from schools for the blind in England, 10 sighted children were selected from mainstream schools and nine sighted autistic children with an IQ below 70 were selected from an earlier screening process. Fifteen of the blind children with an IQ greater than 70 were matched in IQ to 10 of the sighted children. The above authors found more autistic features in the blind group. The latter were reported to manifest stereotypies, other abnormalities of body use and coordination, unusual ways of relating to and communicating with other people, unusual play, and echolalia. They also matched nine blind children with an IQ below 70 to the group of sighted children with autism and found a substantial overlap in their presentation. However, the author who acted as assessor described having misgivings as to whether the quality of the autistic-like blind childrens' social and communicative impairments were comparable to the sighted children with autism. She felt confident to make a diagnosis of Kanner autism in only two of the blind as opposed to all nine of the sighted children. In addition, the CARS scores for the 24 blind children showed a broad range and not a bimodal distribution as if the autistic syndrome had been distinct. Brown *et al.* (1997) postulated that blindness added something to and interacted with the social-affective impairment, and therefore, might be amenable to modification in some.

Rogers & Newhart-Larson (1989) identified five males with Leber congenital amaurosis and compared them to five congenitally blind children with other diagnoses who were matched developmentally. All five of the group with Leber congenital amaurosis fulfilled the criteria for a diagnosis of autism while none in the comparison group did. The developmental histories were described as very similar in the group with Leber congenital amaurosis and were reflected by a normal period of development for about 2 years. This was followed by the development of stereotypic, perseverative and repetitive behaviour with little appropriate play, abnormal patterns of communication, abnormal interpersonal relationships and a tendency to loneness. Rogers & Newhart-Larson (1987) proposed that the autistic symptoms were caused by neurological deficits and postulated a cerebellar deficit in particular.

Hobson *et al.* (1999) identified nine congenitally blind children from schools for the blind in England and compared them to an IQ matched group of nine sighted children with autism. The latter had been diagnosed according to DSM-III-R criteria. The author who rated the children found

that only two of the blind children displayed the quality of social-affective impairment that characterized the children in the sighted group and that none of the blind children were classically autistic. The results also indicated that the blind children who did manifest severe autistic features were more prone to show echolalia, but more likely to have a developmentally appropriate form of communication. Hobson *et al.* (1999) concluded that blindness together with other factors 'may predispose a child to suffer limitation in the experience of and identification with other people's psychological relatedness to a shared world . . . resulting deficiencies in psychological perspective taking are critical for the development of . . . autism'. They also suggested that these children could improve if the appropriate support was in place.

Jan *et al.* (1977), on the basis of their study, did not believe that autism was any more common in children who were blind. They looked at 92 children with congenital visual impairment (some perception of light at least) and found only three with autism. They concluded that a diagnosis of autism was more likely to be made by those unfamiliar with the child.

Cass *et al.* (1994) looked retrospectively at the development of 102 children from their initial presentation before 16 months up to 2.5 years of age. They found that those with the most severe VI were most likely to present with developmental setback of which social accessibility and behaviour were described as the central features. For those whose vision improved over the period, their development also improved. In four children who showed partial recovery, changes had been made to their supported environment prior to this. Interestingly, deterioration was also associated with increasing amounts of stereotypies which, it was noted, appeared to be blocking learning.

In summary, the studies which have looked at the relationship of blindness in autism have varied in their methodology from subjective diagnoses with no formal use of criteria (Keeler 1958; Chess 1971; Fraiberg 1977; Jan *et al.* 1977) to others which have employed diagnostic tools and DSM criteria (Rogers & Newhart-Larson 1987; Brown *et al.* 1997; Elk *et al.* 1998; Hobson *et al.* 1999). These studies appear relatively consistent in their finding that autistic-like symptoms are fairly common in blind children. Some blind children can develop a complete picture of autism. The above authors' theories suggest that this may be because of the related neurological impairment, the inability to experience a shared world, or the delayed cognitive development of the child with a resultant lack of a sense of 'I' or 'you'. Many authors suggest that appropriate early support for blind children could lessen the severity of the autistic symptoms.

Hearing impairment

There have been far fewer studies looking at autism and HI. The major study to address this was conducted by Jure *et al.* (1991) in New York, NY, USA (Table 5). They looked at a population of 1150 children with HI. The criteria for autism was met in 4% ($n=46$). The hearing loss was severe enough in 37 of the children to necessitate special education for language training. The above authors stressed how difficult it was to assess cognitive functioning in the children with both autism and HI. Only eight out of the 45 individuals on whom data was available were considered to have normal or near-normal intelligence, and none of these was judged to be severely autistic. In addition, the social skills of five of these improved significantly with intervention including the teaching of sign language. In 21 out of the 46 subjects, there was an average lag of 4 years before the diagnosis of autism. Deafness was overlooked in five individuals with autism for up to 6 years. Jure *et al.* (1991) found that the educational programme of nine children had been disastrous because of errors in diagnosis. These problems included frequent changes of educational establishment, admission to units for those with multiple disabilities where the HI was overlooked, and residence in programmes for children with autism where their was no experience of dealing with HI or *vice versa*. No clear relationship was found between sociability and the severity of the HI, but there was a relationship between the severity of the ID and the autism. The above authors concluded from their findings 'that autism and deafness coexist more often than by chance alone'. They emphasized that reliable hearing tests should be carried out on all children with language delay. They also added that education is a major problem for children with

Table 5 Studies of autism in subjects with hearing impairment (HI)

Reference	Subjects	Method	Results
Jure *et al.* (1991)	1150 children with HI	Review of medical records Autistic behaviour and cognitive level assessed by neurologist	46 (4%) with autism
Gillberg *et al.* (1990)	28 children with autism under 3 years of age	Behaviour observation Physical examination Parent interview and completion of questionnaire on autistic symptomatology ABC* Griffith's developmental scale Social history Extensive neurobiological investigations DSM-III-R criteria Follow-up after 6 months	20 with confirmed autism 6 (30%) with moderate to severe HI
Steffenburg (1991)	35 children with autism; 17 children with autistic-like conditions	DSM-III-R criteria Neuropsychiatric and neurobiological assessment IQ testing ABC*	1 totally deaf 1 with severe HI 1 with moderate HI 5 with slight to moderate HI
Rosenhall *et al.* (1999)	199 children and adolescents with autism	Assessed by two autism experts, a child psychiatrist/neurologist and a child psychologist DSM-III-R criteria IQ assessment Audiometry	10 with mild to moderate HI 2 with unilateral HI 7 with profound HI

*Autism Behaviour Checklist.

both autism and HI because of the lack of appropriate establishments. The ideal would be for a school for the deaf with special classes to provide both sign language and a programme of behavioural management.

Other studies have looked at the frequency of HI in children with autism (Gillberg *et al.* 1990; Steffenburg 1991) (Table 5). Gillberg *et al.* (1990) found that six out of 20 (30%) children with autism suffered from 'moderate to severe conductive hearing deficit'. Steffenburg (1991) found that eight out of 38 (21%) subjects with autism suffered from 'neurogenic hearing deficits'. However, no conclusions were drawn from these results. These authors stated in a later report (Gillberg & Steffenberg 1993) that, 'auditory problems are difficult to diagnose in autism . . . Only population studies can answer the question whether deafness occurs in autism and autism in deafness more often than suggested by chance.'

Rosenhall *et al.* (1999) also looked at the frequency of HI in those diagnosed as having autism. They looked at 199 children and adolescents and detected HI in 19 members of the group. Seven

(3.5%) of these had a profound HI. They also found that 18.3% were suffering from secretory-otitis-media-related conductive HI. The above authors postulated that anatomical anomalies such as low-set ears, which have been found in children with autism, could account for the high prevalence of this condition. They stressed how difficult it was to assess hearing function in children with autism. Most required more than one test, with 17 individuals needing three separate tests.

Deaf-blindness

There have been no studies looking specifically at autism in those who are deaf-blind. The combination of severe VI and HI appear more likely to produce autistic symptoms, in especially those affected by congenital rubella (Chess 1971). Again, the autism in these cases is likely to be caused by brain damage (Wing 1996). With such a combination of disabling conditions, early diagnosis and identification is essential to provide adequate treatment at the language-learning stage (Jure *et al.* 1991).

Conclusion

The prevalence of sensory impairment in people with ID is greater than in the general population. Visual impairment is 10 times more prevalent in people with ID and HI is anything from 40 to 100 times above the rate in the general population. The survey results show how many more people are found to have sensory impairments when formal testing methods are employed. This highlights the need for the development of specialist services to identify sensory impairments in people with ID. The behavioural and psychological sequelae of having a sensory impairment are compounded by the ID, especially if severe communication problems arise. There are no studies that have looked at the incidence and types of psychiatric illness in people with ID coupled with a sensory impairment. However, by extrapolating from studies in the general population, it can be deduced that there is likely to be an increased prevalence of emotional and behavioural problems in such people.

People with ID and sensory impairment may also present with autistic-like symptoms, and in some cases, autism itself. The need for accurate and early diagnosis of the ID, autism and sensory impairment in such cases is vital. Clearly, early recognition and use of appropriate teaching techniques in such children is vital to maximize their potential and minimize possible behavioural problems.

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