Endnotes

Profound Bilateral Sensorineural Hearing Loss in Nigerian Children: Any Shift in Etiology?

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Deafness, profound hearing loss, is a global problem. However, the causes of, attitudes toward, and management options for deafness differ considerably from region to region. This study seeks to identify the present causes of profound sensorineural hearing loss in Nigeria, which in our environment is almost synonymous to a life sentence of silence and isolation. This is a retrospective survey of children 15 years and below ($M = 6.7$ years, $SD = 3.2$). Of the 115 children included in this study, 64 (55.7%) were males, giving a male:female ratio of 5:4. Age group 1–3 years had the highest proportion of hearing loss, 33 (28.7%), and there was a progressive decline in frequency with advancing age. In about a third (34.8%) of patients, causes were unknown, probably congenital. The main acquired causes were febrile illness (18.3%), measles (13.9%), meningitis (8.7%), mumps (6.9%), or severe birth asphyxia (4.3%). Compared to the findings of two decades ago, we conclude that there is no significant shift yet in the etiology of profound sensorineural hearing loss in our environment.

Deafness, profound hearing loss, is a global problem. However, the causes of, attitudes toward, and management options for deafness differ considerably from region to region. In developing countries, preventable causes of hearing loss such as infections and obstetric mishap remain important (Brobby, 1988; Holborow, Martinson, & Anger, 1982; Ijaduola, 1982; Mc Pherson & Holborrow, 1985; Sellars, Beighton, Horan, & Beighton, 1977; Wright, 1991). In highly industrialized nations, these causes are receding, but noise-induced hearing loss is an important cause (World Health Organization, 1997). Although industrialization is a global index of development, it has been a mixed blessing to mankind. On one hand, it enhances the quality of life and on the other hand poses serious threats to public health. In our own context, excessive uncontrolled noise associated with industrialization is one of the most common causes of hearing loss in the world (Alberti, 1997). Genetic causes of hearing loss are important worldwide, particularly in areas where consanguineous marriages are common (David, Edoo, Mustafah, & Hinchcliffe, 1971; Kapur, 1970).

Holborow et al. (1982), working in western region of Nigeria, reported 60.8% of 803 profoundly deaf school children as having acquired sensorineural hearing loss. The remaining were congenital, unknown (36%), and familial (3.2%). In their series, the main acquired causes of sensorineural hearing loss were measles (20.5%), meningitis (18%), febrile illness (11.4%), birth trauma, including birth asphyxia (4.3%), and mumps and rubella with 1.5% each.

In the same year, Ijaduola (1982), working in Lagos, the area of the western region of Nigeria excluded from Holborow et al. (1982) study, found among 298 profoundly deaf children drawn from the school of the deaf and ENT clinic attendants 49.3% congenital, 36.2% unknown, and 13.1% familial causes. Of the remaining 50.7% with acquired profound sensorineural hearing loss, the main causative factors were measles (13%), meningitis (11%), ototoxicity (9%), neonatal jaundice (5.7%), febrile illness (5%), mumps (3%), rubella (2%), and birth trauma (1.7%).

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In a retrospective review of 105 patients with profound sensorineural hearing loss, aged 1.5–5 years attending otorhinolaryngology clinic in Ghana between 1979 and 1984, Brobby (1988) observed 21% had congenital unknown causes. Of the 79% with acquired causes, the main factors were measles (30%), febrile illness (19%), birth trauma (9.5%), meningitis (8.5%), rubella (3.8%), mumps (3.5%), and neonatal jaundice (1.9%).

Mc Pherson and Holborrow (1985) in a 2-year (1981–1983) fieldwork undertaken throughout the Gambia observed 41.3% congenital causes of severe to profound sensorineural hearing loss among 259 children aged 2–10 years, 33.2% unknown, and 8.1% familial. Of the acquired causes, meningitis and meningitic symptoms (31.7%), febrile illness, including "sickness" (21.2%), measles (1.9%), and rubella (1.5%) were the main factors. The Gambia is within the *meningitis belt* of Africa; this was thought to be responsible for the high prevalence of meningitis.

A study conducted in the western area of Sierra Leone from 1975 to 1985 showed that 28% of sensorineural hearing loss among children was of unknown etiology. Measles was responsible for 45% of cases, meningitis 15%, head injuries 4%, rubella 3%, mumps 2%, and febrile illness mainly lassa fever 3% (Wright, 1991).

All these studies, including the present one, had some difficulties with the term "fever" and/or "sickness." These made etiology sometimes imprecise. These terms are common responses in West Africa to an inquiry of the cause of hearing loss. This could vary from malaria, upper respiratory tract infection, sinusitis, meningitis, typhoid, lassa fever, and so forth. Generally, febrile illness that could not be specifically assigned to a disease entity came under this heading. The offending agent of the sensorineural hearing loss could be the disease itself or the medications given for the treatment.

Although a deaf culture (deafness perceived as a trait binding individuals together: sociocultural concept) is evolving in many developed countries (Anstey, 2002; Levy, 2002), in Nigeria, and indeed Africa, misconceptions and superstitions still persist about deafness as punishment from the gods (Ijaduola, 1982; Lasisi & Ajuwon, 2002). There are reports in some developed countries in which some parents show indifference and occasionally preference for a deaf child (Anstey, 2002; Levy, 2002). In our society, the attitude of the hearing and seeing members of the community toward the deaf is partially discriminatory and suspicious (Lasisi & Ajuwon, 2002; Owoeye, Ologe, & Akande, in press).

There is better awareness, availability, and use of hearing aids in Nigeria today. In fact, an impressive local technology has resulted in small-scale production of much more affordable individual and group hearing aid, which is being used with some benefit. An otorhinolaryngologist, with interest in audiology, is carrying out this project. Local electronics-oriented technologists assist him. The individual hearing aid is body worn type, using regular AAA batteries. Recently (2005) also, the first cochlear implantation and Internet stimulation was performed in Jos, Nigeria. This was made possible through a collaborative work between otorhinolaryngologists at University of Jos Teaching Hospital (Nigerians); University College Hospital, Ibadan, Nigeria (Nigerian); ECWA-Evangel Hospital, Jos (American head and neck surgeon); and House Institute, United States (American neuro-otologist). Although these are encouraging signs of improvement, the deaf in Nigeria still face tremendous odds (Ologe & Akande, 2003).

More than two decades ago, it was noted that for a Nigerian deaf child to achieve speech, there is a very difficult path to follow (Ijaduola, 1982). The problems of the profoundly deaf Nigerian child are complicated by ignorance, poverty, superstition, late diagnosis, and inadequate rehabilitation facilities (Chukuezi, 1991; Ijaduola, 1982; Mc Pherson & Holborrow, 1985). One reason for late diagnosis is because mothers delay seeking help; they wait until their children have failed to acquire speech (Ijaduola, 1982). And even when speech has failed to develop, they delay further with various excuses ranging from having late starters in their family or the child having "only tongue tie" (Ijaduola, 1982). Generally, diagnosis is made between the age of 3 and 5 years (Ijaduola, 1982); where it is believed that the deafness is due to an evil spell, parents do not believe that Western medicine is of any value (Ijaduola, 1982; Mc Pherson & Holborrow, 1985). So they waste precious time, which could have

been spent for educational training and rehabilitation, in search of traditional healers (Ijaduola, 1982; Mc Pherson & Holborrow, 1985).

Situation may be only slightly better now, especially for those who have means. There is probably no kind of hearing aid, irrespective of the sophistication, that is not found among wealthy Nigerians. To this date, National Health Service does not distribute hearing aids. Hearing aids are excluded from areas covered by the newly established (2005) National Health Insurance Scheme.

Results of work done in West Africa showed that much of the sensorineural deafness could be prevented (Brobby, 1988; Holborow et al., 1982; Ijaduola, 1982; Mc Pherson & Holborrow, 1985; Wright, 1991). Thus, the best and most practical way to help the deaf is to identify the causative factors and press for preventive measures (Holborow et al., 1982; Ologe & Akande, 2003). It is for this reason that this study seeks to identify the present causes of profound sensorineural hearing loss, which in our environment is almost synonymous to a life sentence of silence and isolation.

Methods

This is a retrospective survey of children 15 years and below, presenting with severe to profound sensorineural hearing loss (pure tone average >71 dBA) (Campbell, 1998), at the University of Ilorin Teaching Hospital, Ilorin, Nigeria, between July 1999 and June 2002.

The individual case records were identified and retrieved from the Central Medical Records after due permission. The information obtained includes age, gender, cause of deafness, and past medical records of patients. Records of ear examination and audiological measurements were also noted. Examination of the ear was done in the clinic using head mirror with light source and/or a dry cell-operated otoscope in accordance with standard procedure (O'Connor, 1997). Patients with impacted wax or debris had evacuation with Jobson Horne's probe where possible or syringing with warm normal saline. The patients' hearing acuity was crudely assessed with the Barany noise box. Afterward, they were referred to the audiology room for tympanometry using a tympanometer (Impedance audiometer AT 235) in accordance with standard procedure (O'Connor, 1997; Ologe, Okoro, & Oyejola, 2005). Patients less than 5 years old had free field hearing assessment by observing their response to various sounds (the loudness of the sounds were measured using a sound level meter, Testo 815 [Testo GmbH & Co., Lenzkirch, Germany]), duly calibrated with a sound level meter calibrator (Testo 0554.0009). Older children had pure tone audiometry using a clinical audiometer (Danplex AS 67), equipped with well-fitting standard headphones (TDH 39). Equipment was calibrated with sound level meter (Testo 815) initially and periodically to ensure accuracy of measurements. Testing was carried out in a double-walled soundproof booth in accordance with standard procedure (O'Connor, 1997; Ologe et al., 2005).

The degree of hearing loss was assessed using pure tone average of air conduction threshold at 0.5, 1, and 2 kHz, computed separately for each ear (Campbell, 1998). Hearing thresholds were thus categorized into within normal limits (−10 to 25 dB), mild hearing loss (26–40 dB), moderate hearing loss (41–55 dB), moderately severe hearing loss (56–70 dB), severe hearing loss (71–90 dB), and profound hearing loss (>91 dB) (Campbell, 1998). Hearing loss was categorized as conductive where air–bone gap was equal to or greater than 15 dB (Ologe et al., 2005).

Patients with inadequate data, those with mild or moderate sensorineural hearing loss, patients with conductive hearing loss, and those with unilateral hearing loss were excluded from the study.

Results

Table 1 shows the age and sex distribution of the children enrolled in the study. Of the 115 children, 64 (55.7%) were males, giving a male:female ratio of 5:4. The group of children aged 1–3 years had the highest proportion of hearing loss, 33 (28.7%), and there was a progressive decline in frequency with advancing age. The mean age was 6.7 years (±3.2 *SD*).

Table 2 shows the etiological factors of profound bilateral sensorineural hearing loss in the study population. In about a third (34.8%) of patients, causes were unknown, probably congenital. The main acquired causes were febrile illness (18.3%), measles

Table 1 Age and gender distribution of the children

Age (years)	Male (%)	Female (%)	Total (%)
1–3	15 (13.0)	18 (15.7)	33 (28.7)
4–6	16 (13.9)	10 (8.7)	26 (22.6)
7–9	16 (13.9)	9 (7.8)	25 (21.7)
10–12	9 (7.8)	9 (7.8)	18 (15.7)
13–15	8 (7.0)	5 (4.3)	13 (11.3)
Total	64 (55.6)	51 (44.3)	115 (100.0)

(13.9%), meningitis (8.7%), mumps (6.9%), and severe birth asphyxia (4.3%).

Discussion

About two thirds of the causes of deafness in this study were acquired. This, along with the main acquired causes, does not seem to be drastically different from the findings of studies done in West Africa about two decades ago (Brobby, 1988; Holborow et al., 1982; Ijaduola, 1982; Mc Pherson & Holborrow, 1985; Wright, 1991), summarized in Table 3.

It could mean that the long-term benefits of the global expanded program on immunization are late in coming or may be they are not as effective as anticipated. We are aware that recent evaluation of the program indicates success below expectation and a need to rejuvenate efforts in this regard (Centers for Disease Control and Prevention, 2006). Also, the maternal and child health indices do not appear to be significantly better (Ehiri, Oyo-Ita, Anyanwu, Meremikwu, & Ikpeme, 2005). In this study, obstetrics and perinatal etiology were responsible for 12 (10.4%), that is, severe birth asphyxia 5 (4.3%), neonatal jaundice 4 (3.5%), and cerebral palsy 3 (2.6%). This is comparable, in fact higher than the previous quoted studies except the study from Ghana (Brobby, 1988).

Table 2 Etiological factors

Etiology	Frequency (%)
Unknown	40 (34.8)
Febrile illness	21 (18.3)
Measles	16 (13.9)
Meningitis	10 (8.7)
Mumps	8 (6.9)
Severe birth asphyxia	5 (4.3)
Ototoxicity	4 (3.5)
Neonatal jaundice	4 (3.5)
Cerebral palsy	3 (2.6)
Cerebral malaria	3 (2.6)
Congenital rubella syndrome	1 (0.9)
Total	115 (100)

A few years ago, there was a report from Sierra Leone demonstrating an inverse relationship between the incidence of measles-induced sensorineural hearing loss and the increased immunization coverage (Wright & Leigh, 1995). But authors add that the evidence linking the sharp decline in the incidence of measles-induced sensorineural hearing loss with the decline in the number of reported measles cases could be circumstantial. They suggest that more direct evidence of the linkage could accrue to a survey for sensorineural hearing loss in the relevant population (Wright & Leigh, 1995).

In previous studies done in West Africa, measles-induced hearing loss constituted 19.3% of the childhood hearing impairment in Nigeria (Holborow et al., 1982), 30% in Ghana (Brobby, 1988), and 46.1% in Sierra Leone (Wright, 1991). In the Gambia, the incidence of measles-linked hearing loss was 2% and was thought to be partly due to geographical factors but largely to the effective National Immunization Program (Mc Pherson & Holborrow, 1985). In the current study, measles as the cause of profound bilateral sensorineural hearing loss constitutes 13.9%.

An important aspect of the etiology to consider is the proportion for which no specific cause could be assigned. Again, there is no significant difference between the present data and those of two decades ago. Malaria may have been a significant etiological factor in the unknown group where fever and lassitude were the only significant symptoms highlighted in the history (Mc Pherson & Holborrow, 1985; Wright & Leigh, 1995). Chloroquine and other local herbs and plant extracts, widely used in the treatment of malaria in the West African subregion and often misused, may also have been a contributing factor among this group of children (Wright & Leigh, 1995). Observations show that cerebral malaria is an important cause of neurological deficits in Nigerian children (Bondi, 1992).

The most frequent cause of febrile illness in Nigerian, and indeed West African, children is malaria (Mc Pherson & Holborrow, 1985). Malaria, and drugs used in treating it, has been implicated as etiological

Table 3 Summary of reports of studies done two decades ago in West Africa

	Holborrow (1977–1979) (%)	Brobby (1979–1984) (%)	Mc Pherson (1981–1983) (%)	Ijaduola (1981) (%)	Wright (1975–1985) (%)	Ilorin (1977–1979) (%)[a]
Congenital unknown	36	21	33.2	36.2	28	59.7
Measles	20.5	30	1.9	13	45	8.1
Febrile illness	11.4	19	21.2	5	3	21.0
Meningitis	18	8.5	31.7	11	15	8.1
Mumps	1.5	3.5	—	3	2	—
Rubella	1.5	3.8	1.5	2	3	—
Malaria	0.4	—	—	—	—	—
Familial	3.2	—	8.1	13.1	—	4.8
Birth trauma	4.3	9.5	—	1.7	—	—
Jaundice	0.6	1.9	—	5.7	—	—
Trauma	0.6	—	—	—	4	—
Tetanus	—	2.5	—	—	—	—
Ototoxicity	—	—	—	9	—	—
Others	2	6.2	—	—	—	—

[a]Ilorin, the center of the current study was involved in the Holborrow study, 1977–1979. These data are extracted from that work.

factors in deafness in several studies in the West Africa subregion (Bondi, 1992; Chukuezi, 1991; Holborow et al., 1982; Wright & Leigh, 1995). Malaria causing deafness in children needs to be investigated further. It is thought that malaria infection may cause deafness either by local action with microvascular changes in the end arteries of the cochlea or in a general way by lowering resistance to disease and thus enhancing the adverse effects of other infections.

Hereditary and congenital causes of sensorineural hearing loss are poorly studied and hardly diagnosed early in our environment. In Gambia 8% and in South Africa 11% of childhood hearing loss has been attributed to heredity (Mc Pherson & Holborrow, 1985; Sellars & Beighton, 1983). Family tracing is difficult because of the associated stigma. We were unsuccessful in this study to identify specific familial hearing loss. This is probably due to the aforementioned reason. Genetic studies are close to nonexistent in most health care delivery centers. This is not peculiar to Nigeria (Brobby, 1988). The attitude of parents to prenatal diagnoses of deafness is yet to be studied. Because unknown causes accounts for a third of the etiological factors, there is need to be more rigorous concerning studies in this area.

In the current study, meningitis maintained its traditional pride of place as one of the top three causes of profound sensorineural hearing loss in children. Fifteen percent of patients who survive acute bacterial meningitis develop neurological sequelae, and permanent sensorineural hearing loss account for approximately 75% of these cases (Barton, Court, & Walker, 1962; Brobby, 1988; Holborow et al., 1982; Mc Pherson & Holborrow, 1985).

One child was suspected to have congenital rubella syndrome. There was a positive history of fever and rashes while the mother was pregnant. The child also had other associated congenital problems such as squint and cataract. However, no serological confirmation was done. Brobby (1988) was unable to validate the diagnosis of rubella in his series because there was no serological evidence. However, the clinical history was clear and typical, and the children had distinct clinical signs of the syndrome (Brobby, 1988). Rubella is an apparently minor disease with tragic sequelae. The diagnosis of rubella depends largely on the retrospective history from the mother, and in circumstances where minor fevers are common, the incidence of moderate cervical lymphatic glandular enlargement unremarkable, and a faint skin rash unnoticeable, it is no wonder that rubella occurring during pregnancy may go entirely unrecorded (Holborow et al., 1982).

The proportion of our patients with profound sensorineural hearing loss secondary to mumps was at least twice the proportion reported two decades ago in West Africa (see Table 3) (Brobby, 1988; Holborow

et al., 1982; Ijaduola, 1982; Mc Pherson & Holborrow, 1985; Wright, 1991). Mumps is known to commonly cause unilateral sensorineural hearing loss (Brobby, 1988). And becausechildren can cope well with one functioning ear, there may be underreporting of sensorineural hearing loss secondary to mumps (Brobby, 1988).

Rubella, measles, mumps, and viruses that produce a similar pathology are believed to infiltrate the stria vascularis of the cochlea during the viremic stage, and from there they either enter the endolymph directly or damage the metabolic functions of the stria vascularis in some way (Brobby, 1988).

The male preponderance in this study is consistent with previous similar studies in West Africa (Holborow et al., 1982; Ijaduola, 1982; Mc Pherson & Holborrow, 1985; Ologe & Akande, 2003; Wright & Leigh, 1995). Although it has been suggested that there is a true disease preponderance of males and that some of the factors causing deafness are sex linked, this finding may also be sociocultural (Holborow et al., 1982; Mc Pherson & Holborrow, 1985; Ologe & Akande, 2003; Sellars et al., 1977). Some of these studies are wholly or partly school based (Holborow et al., 1982; Ijaduola, 1982; Mc Pherson & Holborrow, 1985; Ologe & Akande, 2003). And there is significant male preponderance in school enrollment in West Africa because of misplaced high value on the male gender (Mc Pherson & Holborrow, 1985; Ologe & Akande, 2003). However, in recent times, increasing female population is seen in schools (Ologe & Akande, 2003). A few hospital-based studies have not found differences in gender distribution of profound sensorineural hearing loss (Chukuezi, 1991).

We conclude that there is no significant shift yet in the etiology of profound sensorineural hearing loss in our environment when compared to findings of two decades ago. This is sad because it follows a pattern in the larger society: high consumption of products from the western world and poor attention to fundamental issues at home. Thankfully, the current health sector reform, which is a subset of an ongoing reform agenda, seeks to address this kind of trend.

Going by the very difficult circumstances that patients with profound sensorineural hearing loss have to live in, more efforts should continue to be made toward prevention. Prevention is by far cheaper and better than rehabilitation. Prevention will also reduce the number of patients competing for the scarce resources required for rehabilitation.

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