Med. J. Cairo Univ., Vol. 62, No. 1, March: 113 - 122, 1994

Hearing Impairment Among Saudi Children with Past History of Health Problems During Infancy

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Abstract

The main goal of this study is to investigate the prevalence of hearing impairment among infants and children with history of health problems during infancy. A random sample of 6,421 Saudi children aged between 2 months and 12 years was screened and clinically examined for hearing impairment; 325 children had hearing impairment among 2,5468 children with health problems during early infancy or childhood. Ninety six (10%) children had hearing impairment among 962 children who had problems in early infancy, which include jaundice, incubator management, general infection, tube feeding, cyanosis and hemorrhage. Out of 1603 children with past history of tonsillitis, measles, meningitis, chickenpox, mumps, discharging ear and otitis media, 229 (14%) had hearing impairment. Hearing impairment consequent upon health problems during infancy has been highlighted and discussed. It can be prevented by attending to these problems.

Introduction

DEAFNESS causes significant disability in children. Many studies emphasized the early diagnosis of deafness and its management by effective amplification. Delay in the detection of significant hearing impairment will influence the development of speech, language and social interaction. The growing knowledge and understanding of the various pathological processes involved has helped in the reduction of the number of the common causes of hearing impairment in the past. however, there is still a large gap in our knowledge of the pathological processes involved in cases with abnormal genetic factors and with various embryopathies especially those caused by viral infection.

The primary reason for the early detection of hearing impairment in infants is to optimize the acquisition of the maturation processes, since early auditory stimulation is necessary for their full development - an attractive but not rigorously proven fact. The early detection of hearing impairment can be reached by a careful anamnesis of history, family pregnancy, birth and infancy history, careful physical examination and by the necessary clinical investigations. Over the years, many investigations have grappled with this task. The current North American guidelines which were

proposed in 1982 by the Joint Committee on Infant Hearing, include the following points: identification of sub-population with increased prevalence of hearing impairment by means of high-risk registers. The factors that identify infants who are at risk for hearing impairment include the following:

1- Family history of childhood hearing impairment (hereditary).
2- Perinatal infections (e.g. rubella, syphilis, cytomegalovirus, herpes simplex).
3- Anatomical malformation of the ear, head and neck.
4- Birth weight less than 1500 g.
5- Hyperbilirubinaemia at levels exceeding the indication for exchange transfusion.
6- Bacterial meningitis.
7- Severe neonatal asphyxia.

In Saudi Arabia, scanty information is available on this problem and neither its magnitude nor its etiological factors has been studied in detail. In this paper, we present the results of a large epidemiological study on hearing impairment in Riyadh. It is to assess the prevalence of hearing impairment among Saudi children who had history of health problems during their perinatal period and their first few years of life.

Material and Methods

A random survey of 6,321 Saudi infants and children below the age of 12 years, was carried out in Riyadh, the Capital City of Saudi Arabia, from May 1988 to September 1990, to determine the rate of hearing impairment. The sample was designed to represent all the socio-economic and demographic groups of Saudis living in Riyadh.

The sampling design was essentially based on quota sampling using two interlocked quotes: age and sex. An element of randomness was introduced in the actual sample selection process by a three stage random sampling frame of Riyadh. The city was divided into 93 administrative areas and these areas were distributed into six strata according to socio-economic homogeneity. One-fifth of the areas in each stratum were chosen randomly. Each area was further subdivided into roads and the latter were subsequently divided into smaller blocks of approximately equal size and a sample of each block was randomly selected. Within each block selected, a random starting point was chosen and a predetermined zigzag route followed calling at every other household encountered.

Each child attendant or close relative was asked to answer a questionnaire which included among other variables: age, sex, birthweight, parents-relationship, family history of possible hereditary deafness, any complaint of hearing or speech defect, history of exposure to the known factors of childhood deafness and perinatal history with special emphasis on the presence or absence of the high risk factors for hearing impairment. A Series of routine investigations was undertaken. All of the children were subjected to both a pediatric and an otological examination. Hospital records were obtained on all the babies to determine if any significant abnormality had occurred in pregnancy, during or after birth and if a history of meningitis or other infection had been reported. Pure tone audiometry or auditory evoked response was undertaken to determine the extent and type of any hearing impairment. Auditory evoked response has been used exclusively in children who were unable to cooperate.

Serological tests for the detection of anti-bodies against syphilis, toxoplasma, cytomegalic virus, rubella and herpes sim-

plex were carried out in all children with high risk factors and in children with hearing impairment.

The families of the children were also invited to attend the department for a routine audiometric examination in order to substantiate or eliminate the possibility of any other member of the family suffering from sensorineural deafness; such information was important in establishing a family pattern of hearing loss which might bear upon the nature of the hearing loss in the child. Based on the general and the E.N.T. examination, laboratory investigations and the presence of risk factors, all the sampled children were examined by an E.N.T. Specialist to identify the rate of deafness among the children at risk.

Results

I- The total number of children who participated in this study was 6.421; 55 percent (3532) were boys and 45 percent (2889) were girls. The age ranged from two months to 12 years with a mean age of 56.67 month (table 1). In only 0.3% of the sampled children we could not reveal the accurate age at the time of the interview. Out of 6,421 children, 19.58% (1256) were at risk of hearing impairment.

II- Medical history of the sampled children:

a) 15 percent (962) of the sampled children had health problems during early infancy. Table (2) details those with hearing impairment among this group. Children with the aforementioned health problems had slightly higher rate of hearing impairment than children with no health problems (Fig. 1).

b) In addition to the health problems during infancy mentioned in Table (2), 25 percent (1603) of the sampled children gave a history of other illnesses as shown in Table (3). Children with history of meningitis had the highest rate of hearing impairment (66.7%) Figs. 2 & 3, while the rates in those with past history of discharging ears and scarlet fever were 31.4% and 29.5% respectively. The rates of hearing impairment in children with severe diarrhea (19.5%), tonsillitis (16.7%) and runny nose (16%) were still relatively high.

c) Past history of otitis media:

- About 60% (3853) of the sampled children gave past history of otitis media.
- Ninety four (94/6.421) children constituting 1.5% were found with chronic ear disease, nine (9) children with cholesteatoma and 85 with central tympanic membrane perforation.
- Distribution of the different types of hearing impairment is summarized in Fig. (4).
- Degrees of hearing loss can be seen in Table (3).

Table (1): The Age Distribution of the 6421 Children who Entered the Study.

Age	No.	%
12 months	1004	15.6
13 - 24 months	851	13.2
25 - 36 months	683	10.6
37 - 48 months	676	10.5
49 - 60 months	638	9.9
61 - 72 months	651	10.1
73 - 84 months	374	5.8
85 - 96 months	347	5.4
97 - 144 months	1178	18.3
No data	19	0.3
Total	6421	100.0

Table (2): Health Problems During Early Life of the Sampled Children.

Health Problem	No.	Children with hearing impairment
Jaundice	520	46 (8.1%)
Incubator	205	24 (3.2%)
General infection	128	13 (2.0%)
Tube feeding	77	9 (1.2%)
Cyanosis	26	3 (0.4%)
Haemorrhage	6	1 (0.1%)
Total	962	96

Table (3)

History of illness	Children with hearing impairment	No.	%
Tonsillitis	62	372	5.8
Measles	23	340	5.3
Runny nose	39	244	3.8
Chickenpox	26	218	3.4
Mumps	8	135	2.1
Discharging ear	26	83	1.3
Severe diarrhea	16	83	1.3
Scarlet fever	13	45	0.7
Whooping cough	2	19	0.3
Head injuries	2	19	0.3
Tuberculosis	2	19	0.3
Meningitis	9	13	0.2
Epilepsy	1	13	0.2
Total	229	1603	25

Table (4): Degrees of Hearing Loss.

		20 - 40 dB	40 - 60	60 - 80
Conductive	184	265	19	
Bil. S.N.H.L.	67	3	17	47
Unilateral S.N.H.L.	13		2	11
Mixed hearing loss	55	37	15	3

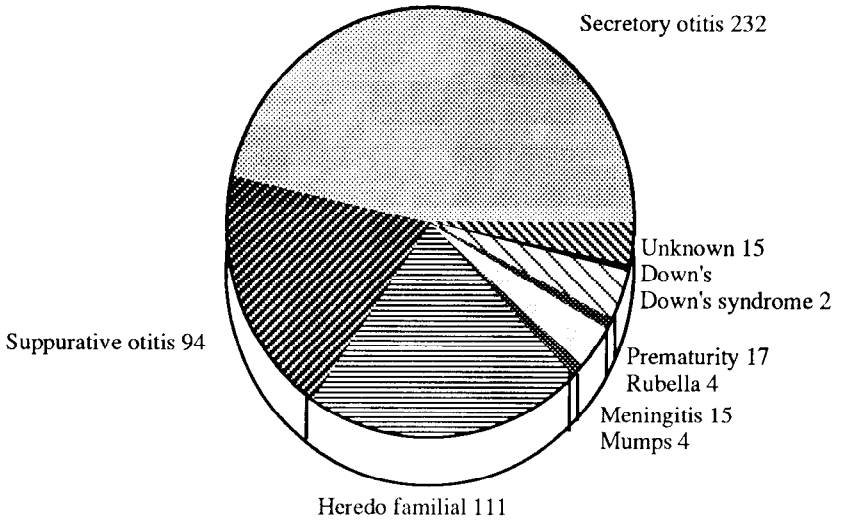

Fig. (1): Aetiology of hearing impairment.

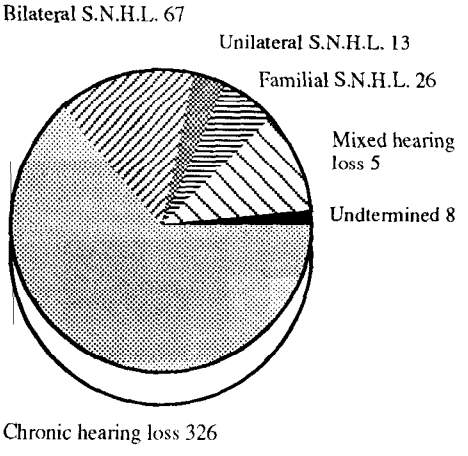

Fig. (2): Distribution of the different types of hearing impairment.

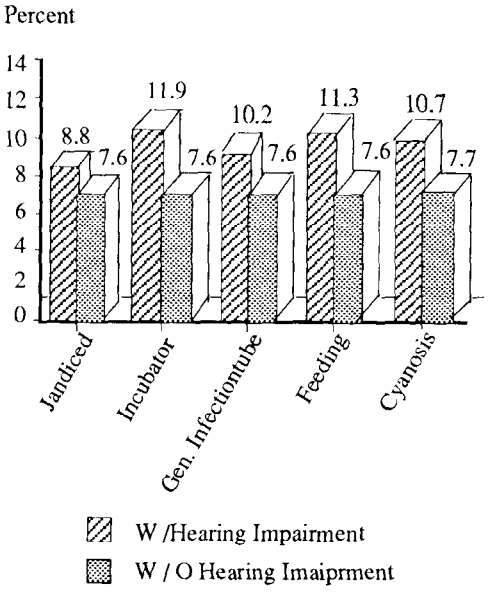

Fig. (3): History of possible health and hearing loss.

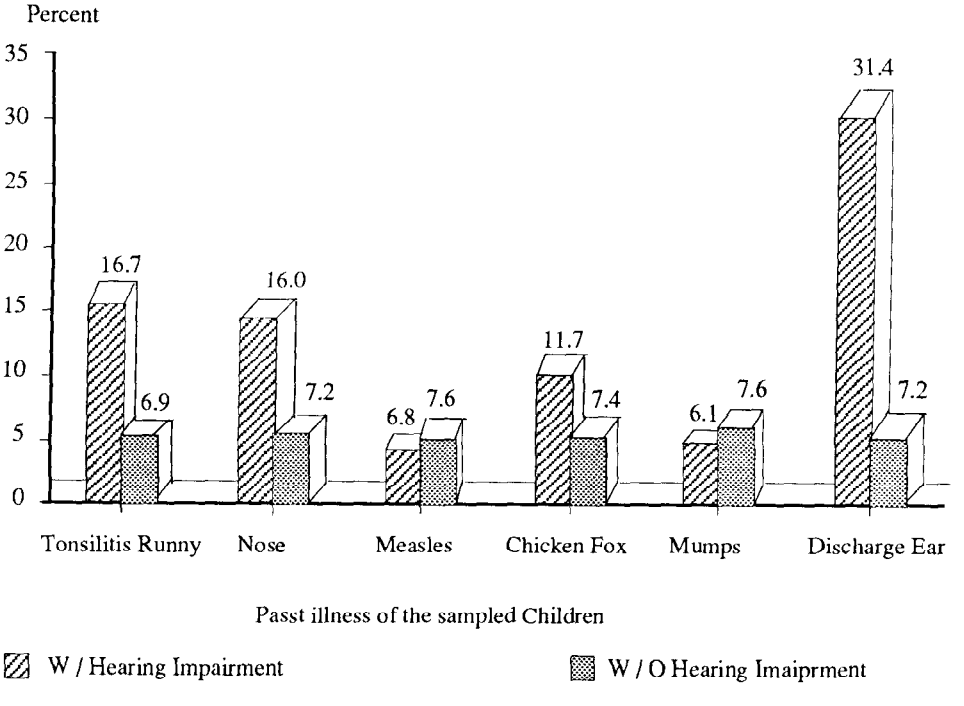

Fig. (4): History of illness and rate of hearing impairment (1).

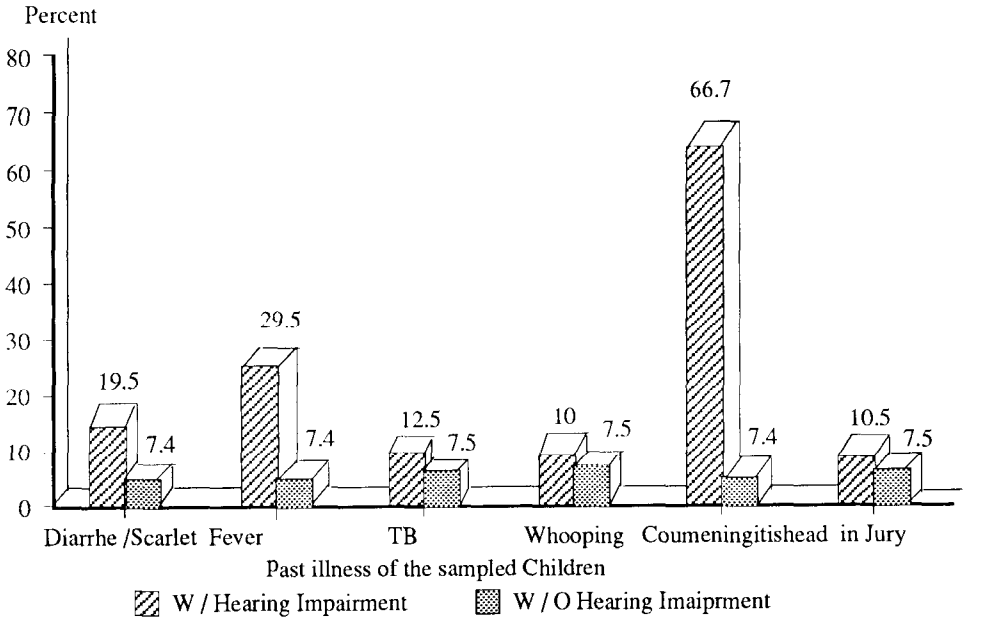

Fig. (5): History of illness and rate of hearing impairment (2).

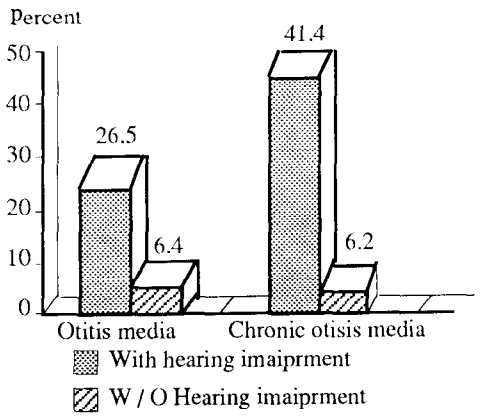

Fig. (6): History of otitis media and rate of hearing impairment.

Discussion

This study showed that children with the aforementioned health problems (jaundiced, incubator management, general infection, tube feeding, asphyxia, haemorrhage, etc.) during early infancy had slightly higher rate of hearing impairment than other children without such problem.

In this study, forty six (46) children had developed sensorineural hearing loss, out of 520 children who had Jaundice during early infancy. The Joint Committee on Infant Hearing recommended the reliance on clinical judgement regarding the degree of severity of hyperbilirubinaemia as a cause of congenital deafness especially when the level of bilirubin exceeded the indication for exchange transfusion [1]. Furthermore, the prevalence of hearing loss among children with cerebral palsy is greater than in the general population, probably because of the aetiological significance of kernicterus resulting from Rhesus incompatibility [2].

Marcus in 1970 [3] found 10% of all cases of congenital deafness in the United States, Prior to 1960, were due to neonatal jaundice. His own data showed only two new cases of kemicteric deafness up to the

year 1965 and none thereafter. Marcus correctly ascribed this reduction to the greatly improved methods of clinical management of hyperbilirubinaemia. In recent years, this hazard was reduced markedly since haemolytic disease of the newborn was almost eliminated totally by the prophylactic administration of anti-D vaccine in addition to the general improvement in antenatal care. Significant succes has also been achieved in the reduction of congenital deafness from Rhesus incompatibility by Anti- D vaccination program and early exchange of blood in newborn infants with hyperbilirubinaemia [4].

Asphyxia and haemorrhage are known causes of hearing loss. In this study, hearing impairment was noted in 3 out of 26 children who had past history of asphyxia and in one of 6 who had haemorrhage. The Joint Committee identified asphyxia as a risk factor as follows: Severe asphyxia which include infants with apgar scores of 0.3 who fail to start spontaneous respiration by 10 min. and those with hypotonia persisting up to two hours of age.

Others added that asphyxia can be determined by measurement of arterial pH, which must be 7.3 to be included on the high risk register [5]. In the last two decades, the viability of premature infants increased markedly as a result the improved intensive care facilities and therefore infants with prematurity and dismaturity are at a low risk of developing hyperbilirubinaemia and kernicterus.

To what extent the degree of asphyxia can be the cause of deafness and hearing impairment is still not clear. Pappas [6] using pH as a marker, found that 6% of the 111 deaf babies had history of asphyxia, 40% of whom had low birth weights and were hyperbilirubinaemic. It should be emphasized that these clinical phenomena occur simultaneously. A positive medical history, together with sensorineural deafness associated with other neurological findings which give sufficient indication of the nature of the handicap. Whilst the exact mechanism of the deafness in premature babies is not clearly understood, there can be no doubt of the close association between these two factors [7].

Parving [8] found that 14% (16) of the total sampled children (117) in her study had natal or neonatal problems. Of these children, 38% (6/16) had neonatal jaundice requiring exchange transfusion; another 38% (6/16) had severe asphyxia requiring incubation with continuous oxygen inhalation for at least 24 hours; 6% (1/16) had neonatal sepsis which was treated with ototoxic drugs, while 18% (3/16) were born before term and had low birth weight (<2500 g). Three of the children with asphyxia and/or jaundice also had low birth weights.

In animal experiments, Myers in 1972 [9] has found that acute total asphyxia, in which there is sudden complete cessation of oxygen supply to the foetus, results in damage predominantly to the brain stem. The cerebral cortex and basal ganglia are either unaffected or damaged later. In the clinical practice a similar form of asphyxia could be associated with cardiac arrest. There is evidence that the resulting otological lesion was identified as haemorrhage into the cochlea [10]. In hypoxia there is a rise in arterial capillary rupture [11]. Buck [10] studied the temporal bones of 73 new born infants and suggested that traumatic procedures played an important role causing the heamorrhage into the inner ear.

In a report by Thomson et al. on the quality of survival after severe birth as-

phyxia in 31 babies, one of these children was found to have a sensorineural loss [12].

Abramovich et al. [13] investigating the hearing loss in very low birth weight babies treated with neonatal intensive care reported that of the 111 survivors of birth weights, 1500 g. or less, 10 (9%) had sensorineural deafness and one baby had congenital conductive hearing loss. Apnoeic attacks in the neonatal period being the most significant predictor of hearing loss while an indirect serum bilirubin level of at least 170 u mol/1 in the neonatal period had an additive effect [13]. They concluded that ambient noise from incubator had not affected the hearing of these low birth weight babies. Premature infants nursed in an incubator sometimes receive ototoxic drugs of the aminoglycoside group, which carry a risk of causing deafness [14]. Animal experiments have shown that the noise produced by an incubator in combination with Kanamycin is harmful to the hair cells of the inner ear, but under the same circumstances each of the two factors separately do not appear to be ototoxic [15].

The health problems during the first few years of life, 25% (1603) of our sampled children had history of past illness (tonsillitis, measles, mumps, meningitis, otitis media, etc.), 229 children among this group had hearing impairment. Thus, those children with past history of meningitis had the highest rate of hearing impairment (66.7%). The death rate of intracranial complications of otitis media is only in the preantibiotic area, the percentage of children who suffer from a hearing loss after meningitis seems to have fallen by 50% [14].

Bacterial meningitis is the most common postnatal cause of permanent hearing loss and is certainly is more dangerous than viral agents pneumococi, meningorocci and heamophilus influenzae, in decreasing order; these being the most important bacterial pathogens causing meningitis complicated by deafness [16]. The child who develops hearing impairment as a result of bacterial meningitis usually sustains cochlear destruction by the time he presents to hospital [17]. The length of prodromal illness prior to hospitalization or institution of antimicrobial therapy does not correlate with risk of hearing loss; [18] however, late improvement in sensorineural hearing loss has been reported, but it is quite rare in a child with severe or profound deafness to revert completely to normal.

Younger children are more susceptible to developing meningitis and the resulting deafness is usually due to labyrinthitis which is a complication of meningitis. This is sufficient reason for such children to have their hearing ability examined after recovery [16]. The prevalence of post-meningitis deafness persists over the years as evidenced by the unmber of survivors with this sequelae [18]. Flint [19] considered meningitis to be the most significant factor in acquired childhood deafness. It should be stressed that meningitis may be treated with potentially ototoxic drugs, in particular the aminoglycosides such as streptomycin, dihydrostreptomycin, kanamycin, gentamycin, tobramycin and amikacin [19] and therefore the choice of medication is a compromise between the spectrum of activity of the antibiotic or the combination of antibiotics and the possiblity of ototoxicity which is difficult to assess [14].

The mumps virus is felt to be the most common etiological agent for unilateral acquired sensorineural hearing loss in the pediatric age group [20]. It has been estimated that hearing loss associated with mumps

occurs in approximately 5 out of every 10.000 cases of mumps [21]. The loss is usually abrupt in onset, and may be associated with nausea, vomitting and vertigo. Serological diagnosis can be made on the basis of a fourfold or greater increase in specific antibody titer [20]. Temporal bone histology revealed damage mainly in the basal turn of the cochlea with atrophy of the stria vascularies and organ of corti [22].

Rubella virus may cause bilateral symmetric sensorineural hearing loss that affects the high tones more than the low, other clinical symptoms include Koplik's spots maculopapular rash, conjunctivitis, diarrhea and vomitting [20]. Diagnosis can be made by isolating the virus from throat culture or by noting an increasing measles and antibody titer. Regarding the other viral causes of sensorineural hearing loss include varicella and influenza, a new combination-vaccine has now been introduced against mumps, measles and rubella (the so called MMR-vaccine), which is to be administered to girls as well as boys at the age of 14 months. The second dose is usually given at the age of 9 years. This is consistent with a recent recommendation from the United States [19].

Sensorineural hearing loss has been well documented in association with clinically evident otitis media. In this study 60% (3853) of the sampled children had past history of A.O.M. 1.5% (94/642) children had chronic otitis media affecting the children's hearing. The mechanism suggested for sensorineural impairment in these patients has been the passage of inflammatory agents or toxins from the middle to the inner ear, with the round window membrane the most likely site of entry since it is permeable to various toxins and to antibiotics [23]. The local toxic agent might alter the mass or stiffness of the spiral ligament or the basilar membrane, thus resulting in altered displacement of the cochlear partition in response to sound [24]. Infants and children may have histological evidence of chronic otitis media without clinical manifestations [24]. Paparella [25] termed this disease "Silent Otitis Media" and felt that it could play a role in the pathogenesis of sensorineural hearing loss. It seems that many factors contribute to susceptibility to chronic ear disease in addition to environmental factors and repeated attacks of acute otitis media. Increased awareness in addition to early management of acute otitis media will no doubt stop its preogression to chronic ear disease.

Acknowledgements:

I would like to express my appreciation to the King Abdul Aziz City of Science and Technology (KACST) for financial support of the project; to Dr. Med. Fawziah Al-Kandari, Medical Labor, for her valuable advice throughout and acknowledgement must be given to all the parents and children who gave us all the assistance we needed to make the necessary possible and Prof. Siraj M. Zakzouk, for his continued interest in this work and for his invaluable advice throughout.

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