1983

Bacterial meningitis and hearing loss in children.

Joan M. Gerharter
The University of Montana

Recommended Citation

Gerharter, Joan M., "Bacterial meningitis and hearing loss in children." (1983). *Graduate Student Theses, Dissertations, & Professional Papers*. 7300.
https://scholarworks.umt.edu/etd/7300

COPYRIGHT ACT OF 1976

BACTERIAL MENINGITIS

AND

HEARING LOSS IN CHILDREN

by

Joan M. Gerharter

B.A., University of Nevada, 1975

Presented in partial fulfillment of the requirements for the degree of

Master of Communication Sciences and Disorders

UNIVERSITY OF MONTANA

1983

Approved by:

Franucis M. Jueleci uH
Chairman, Board of Examiners

[signature]
Dean, Graduate School

_4-25-83_
Date

UMI Number: EP38101

UMI

Dissertation Publishing

ProQuest

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

I. BACKGROUND AND RATIONALE

During my externship at the Denver Children's Hospital I had the opportunity to participate in the evaluation of children with a variety of hearing problems. One of the most challenging tasks was the audiological evaluation of pediatric meningitis patients. The age of the children -- a "difficult to test population"[22] -- and their decreased responsiveness due to the critical nature of their illness posed unique clinical problems.

In view of the spectrum of possible sequelae to meningitis, hearing loss is considered minor,[11] and identifying patients at-risk for meningitic hearing loss has not been highly successful.[9,24] Thus, attending physicians' decisions regarding chemotherapeutic regimes to avert or minimize hearing loss are frequently made on the basis of results of hearing testing during this period of critical illness. Denver Children's Hospital protocol calls for audiological evaluation of all meningitis patients early in the course of treatment; obtaining reliable audiological data is imperative.

Recently, the suggestion has been made in the literature[?] that patients who suffer hearing loss due to bacterial meningitis may present a typical clinical profile. This paper will examine similarities in laboratory and clinical

data for those pediatric patients treated for Haemophilus influenzae meningitis at Denver Children's Hospital.

II. BACTERIAL MENINGITIS

1. Introduction

The coverings of the brain and the spinal cord -- the
dura mater, the pia mater, and the arachnoid -- are, collec-
tively, the meninges. Any inflammation of these tissues is
defined as meningitis. A wide variety of pathogenic and/or
chemical agents may be responsible for such inflammation.

Chemically-induced meningitis may arise from an imbal-
ance of a specific agent, for example, insulin. In these
cases, control of the imbalance results in immediate decrease
in inflammation. However, when the meninges are invaded by
disease-producing pathogens, the course of the disease, as
well as the treatment plan, are much different. Cases of
meningitis have been attributed to a startling spectrum of
pathogens, however, the micro-organisms primarily responsible
for meningeal inflammation are either viral or bacterial.
This paper will examine the course and effects of bacterial
meningitis due to a specific micro-organism, Haemophilus
influenzae.

2. Disease Processes

Aside from chemically-induced meningitis, the pathogens
responsible for meningeal infection may reach that site by
several routes. Following prolonged or particularly virulent
infection, inflammation of the sub-arachnoid space may result

-1-

from the transmission of the disease-producing micro-organisms from the middle-ear space or sinuses. Additionally, pathogens are circulated throughout the body by the bloodstream; an infection may, in this manner, reach the meninges.

According to Adams[1] and Keane,[14] once the pathogens have reached the meninges, a picture of the course of the disease can be drawn. The purulent exudate quickly envelopes the brain, surrounding the brainstem and the internal auditory meatus. This infected material may also reach out along the cranial and spinal nerves, although inflammation of the nerve sheaths occurs only after the infection has persisted for several days. During this time, pathogens irritate the blood-carrying vessels, interfering with circulatory processes and resulting in damage to cortical tissues. As the infection progresses, exudate continues to collect at the base of the brain, disrupting the passage of cerebrospinal fluid into the spinal cord. This leads to hydrocephalus, increased cranial pressure, and further cortical damage.

In addressing meningitic hearing loss, Ravio[24] has noted three processes which can adversely affect hearing during the course of a meningeal inflammation: "1) purulent labrynthitis, 2) perineuritis of the eighth cranial ·nerve, and 3) direct lesion to the brainstem or even higher hearing pathways." Thus, reports of hearing loss following meningitis may not form a consistent pattern as the impairment may be

attributable to one of several destructive processes. This seems to be a characteristic of meningitis: a constellation of life-long residua is possible following a relatively short duration infection. The difficulty of predicting and responding therapeutically to prevent loss of function is complicated by multiple, simultaneous infarctions.

3. Pathogens

Three micro-organisms are most frequently responsible in bacterial meningitis: Haemophilus influenzae, Diplococcus pneumoniae, and Neisseria meningiditus. Of these Haemophilus influenzae has been shown to be more prevalent in children under five years of age, accounting for two-thirds of meningitis in pediatric patients in one study.[14] Presence of this micro-organism does not necessarily indicate meningeal infection, as the majority of healthy adults and children have been shown to have Haemophilus influenzae bacteria in the mucosal lining of the nasopharynx. Thus, once the body has developed antibodies for Haemophilus influenzae, an individual can remain healthy despite the presence of this pathogen. For children under the age of two, the incidence of influenzal infection increases, which is likely due to the lack of the necessary antibody.[23] Neisseria meningiditus and Diplococcus pneumoniae have been noted to affect primarily infants and older adults.[14]

4. Diagnosis

Description of symptoms of influenzal meningitis are strikingly similar. Most commonly, the symptoms include lethargy, vomiting, "spiking" fever, nuchal rigidity, headache, and irritability.[1,7,15] Prompt diagnosis by symptomology is more difficult in younger children because they tend to respond to infection in an undifferentiated fashion.[14] Mothers of children being treated for meningitis have reported that intially, their children refused to cuddle, probably due to neck tenderness.[13] From a review of medical charts at Denver Children's Hospital, a typical clinical history of a child with meningitis can be drawn. This composite includes a history of a cold, upper respiratory infection, otitis media, and even one incidence of a urinary infection. This state of infection is followed by a sudden change in status such that the child became lethargic, feverish, irritable, and began vomiting. In cases where the illness was protracted, seizure activity -- episodes of staring, twitching, or generalized convulsions -- were reported. On hospital admission, a tender or stiff neck was generally noted.

As part of the diagnostic work-up, a lumbar puncture is performed in cases where meningitis is suspected. Critical diagnostic values include both cerebrospinal fluid protein and glucose levels, which are 20 to 45mg/100 ml and 50 to

100mg/100 ml respectively in the healthy individual.[7,19]

In the case of bacterial meningitis, cerebrospinal fluid protein levels will rise and glucose levels will fall. Viral meningitis does not cause change in the cerebrospinal fluid glucose level. Specific pathogens are identified by blood and/or cerebrospinal fluid cultures, which are then treated with antibiotics to determine resistance and suscep-tibility. Based on this laboratory information, a thera-peutic plan is established to combat the micro-organism responsible for the infection.

5. Sequelae

With the development of antibiotics, the mortality rate for meningitis has decreased from 90% in the early part of this century to approximately 10% and lower in the past ten years.[3,4,11,14,17,19] The continued critical nature of this illness is illustrated by the type and severity of the sequelae reported.[4,6,8,11,29,30,31] Disruptions of neurological processes can take the form of motoric diffi-culties as seen in paralysis, paresis, tremors, and seizures. It can also involve cranial nerves and/or end organs, resulting in impairment of vision or hearing. Potential for intellectual growth may also be reduced, yielding develop-mental delays.

Recently, investigators have suggested that the post-meningitic child who exhibits difficulties in perception or integration of sensory input indicated subtle neurological damage due to the disease. Reports of behavior or attention problems are common, and at least one investigator has identified emotional difficulties as a result of meningitis.[25,32] Typical of the surveys of menigitic sequelae is the study by Kresky,[17] which indicated that 34% of the children studied suffered some sort of neurological impairment as a result of meningitis. Of the 28 children exhibiting difficulties, three were classed as severe and 20 were judged to have more subtle evidence of neurological disorder.

Authors agree that the incidence of meningitic hearing loss is on the order of 20%.[2,8,9,11,14,15,21,23] The degree, severity, and configuration of the hearing loss can vary enormously. Further, there are reports of changes in hearing status -- improvements as well as progressive deterioration -- following treatment for meningitis.[27]

6. Prediction of Sequelae

Due to the spectrum of possible residua, several authors have attempted to predict which children will suffer long-range consequences of meningeal infection. Wright[33] proposed a checklist identifying the factors which correlated with meningitis-related sequelae: seizures, duration of

hospitalization, age at onset of meningitis, and presence of bacteria in the cerebrospinal fluid. These considerations, however, would seem to coincide with the severity of the infection, rather than identifying which children are at-risk for specific sequelae. Nadol[21] determined that evidence of neurological disorder was positively correlated with hearing loss. The work of both these individuals is based on data gathered after the infection has been resolved, when the neurological insult, if any, has already taken place. Herson and Todd[11] developed a scoring system based on evaluation of physical condition at the time of hospital admission (Table I). The admission checklist is currently in use at Denver Children's Hospital. According to Herson and Todd[11] and Gary,[9] this method is valuable in identifying children at risk for major and/or minor sequela before the infection has been resolved. (See Talbe II) In an unpublished paper by O'Leary,[24] it was pointed out that this method, although valuable in predicting major sequelae, was not effective in identifying children at risk for hearing loss.

Recently, Berlow[2] has indicated that, of the group studied, a significant number of individuals suffering sensorineural hearing loss as a result of Haemophilus influenzae meningitis presented cerebrospinal fluid glucose levels below 20mg/100ml on hospital admission. Her impli-cation appeared to be that cerebrospinal fluid glucose of

20mg/100ml or lower represented a critical value which might correlate with hearing loss. Based on these data, a method for predicting which pediatric patients may be at risk for meningitic hearing loss could be available.

TABLE I

Herson-Todd[11]	Admission Factors	Points
Coma.		.3
Hypothermia		.2
Seizures.		.2
Shock		.1
Age (less than 1 year).		.1
CSF white blood cell count, 1,000/cu mm		.1
CSF glucose, 25 mg.		.0.5
Symptoms more than 3 days		.0.5

TABLE II

Herson-Todd[11]: Possible Meningitic Sequelae

Major sequelae	Minor sequelae
Blindness	Ataxia
Hydrocephalus	Hearing loss
Institutionalization	Hemiparesis
Microcephaly	Hyperactivity
Quadriplegia	Peripheral facial palsy
Retardation	Subdural effusions
Seizures	

III. BACTERIAL MENINGITIS AND HEARING LOSS

1. Method

From February, 1979, to December 1981, a total of 42 children were treated for <u>Haemophilus influenzae</u> meningitis at Denver Children's Hospital. In every case, the diagnosis of <u>Haemophilus influenzae</u> meningitis was made on the basis of blood and/or cerebrospinal fluid cultures. These children ranged in age from three months to three and a half years of age. Figure I depicts the distribution of age at the onset of meningitis for these children. During the course of their hospital stay, each child received at least one audiological evaluation by the same ASHA-certified pediatric audiologist on staff at Denver Children's Hospital. Hearing loss was defined as PTA 20dB for children older than six months. Due to developmental differences in ability to respond to auditory stimuli, expectations for infants six months of age or younger were adjusted. (See Figure II.) The differences in the ages of the children and their concommitant ability to respond required a variety of test paradigms. For children six months to approximately eighteen months of age, COR[34] (Conditioned Orienting Response) audiometry was used. Stimuli included both warbled pure tones and speech. Further, if the child's physical condition allowed participation, body part identification was included in the evaluation. For children

six months to approximately eighteen months of age, play
audiometry provided the vehicle for audiological evaluation.
In all cases, speech or music were presented in an attempt
to confirm responses to pure tone stimuli. As noted on
Table III, very young children were expected to respond to
speech at slightly softer levels of presentation than warbled
pure tones because speech is generally felt to be "more
interesting."[22]

Frequently, the child's state of physical discomfort
required that testing paradigms fit themselves to the patient's
level of function. For example, head-turn responses to music
were accepted from a critically ill two-year-old despite the
general rule that children that age can participate in play
audiometry. Indeed, occasionally, these pediatric patients
were unable to localize left or right due to nuchal rigidity
accompanying the meningitis. These situations required obser-
vation of behavioral responses -- eye-widening, change of
facial expresssion -- similar to those noted during the
testing of infants.

Audiometric data for the 42 subjects were reviewed.
Figure 2 presents the incidence of hearing loss by age in the
post-meningitic patients. In addition, hospital records for
each child were reviewed to determine cerebrospinal fluid
glucose level at admission, length of hospital stay, and duration
and type of symptoms prior to hospital admission.

FIGURE 1

Distribution of Occurance of Meningitis
by Age at Onset

Girls (N=17)

Boys (N=25)

Subjects

Age in Months
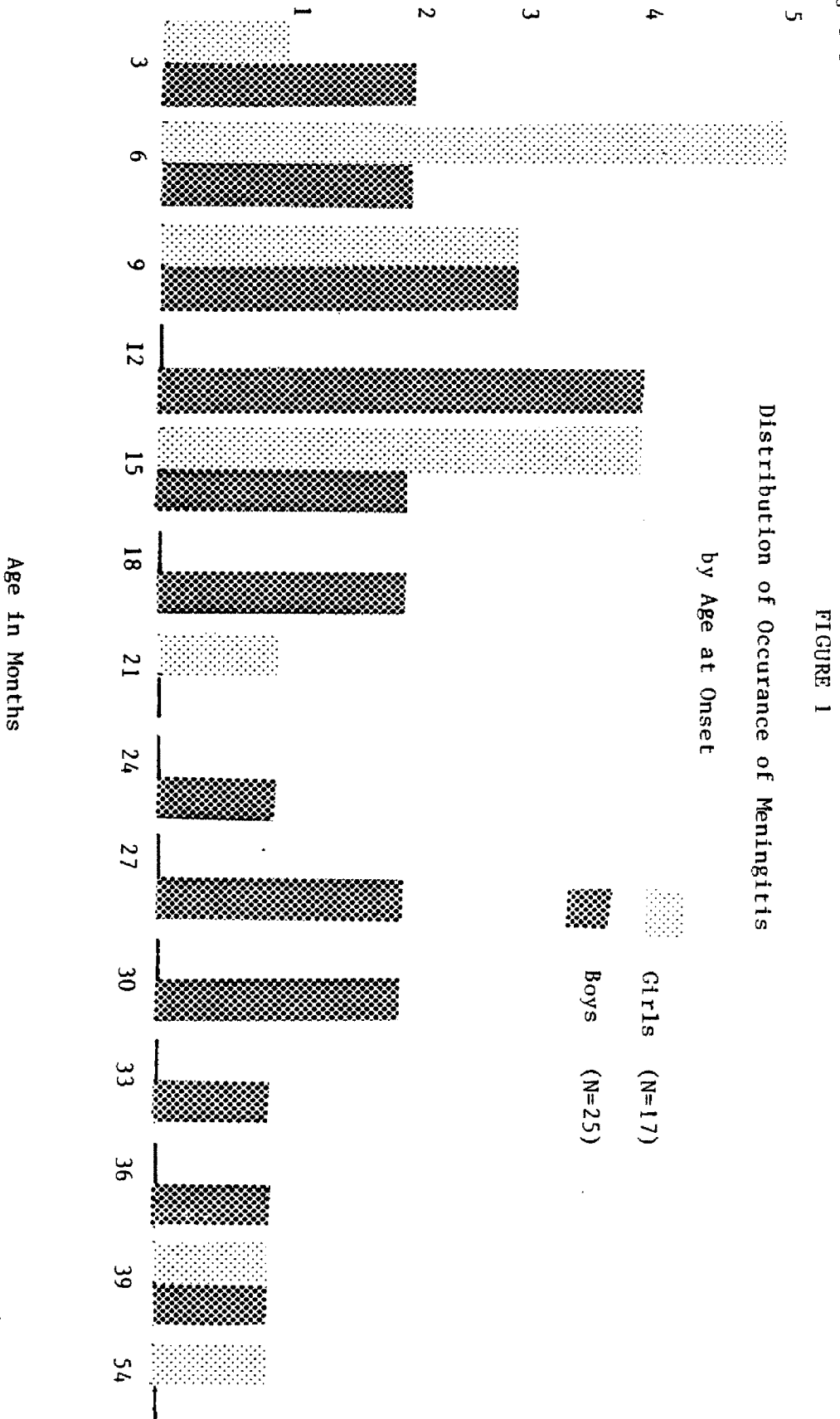
TABLE III.

Level of Presentation of Auditory Stimuli

Expected to Elicit Responses in Normally-Hearing Infants

AGE	DENVER CHILDREN'S HOSPITAL[13]		WILBER[*]	
	Warbled Pure Tones	Speech	250 Hz, 500 Hz	1000 Hz, 2000 Hz
1 month	85 dB[†]	75 dB	60-80 dB	65-85 dB
2 months	75 dB	65 dB	60 dB	70 dB
3 months	65 dB	55 dB	60 dB	70 dB
4 months	55 dB	45 dB	40 dB	50 dB
5 months	45 dB	35 dB	40 dB	50 dB
6 months	35 dB	25 dB	40 dB	50 dB

*For the purposes of comparison, data from Wilber[33] on the subject of threshold measurement is presented here.

†dB HTL re:ANSI, 1969

Subjects

FIGURE 2

Distribution of Meningitic Hearing Loss

by Age at Onset of Meningitis

Girls (N=17)

Boys (N=25)

Hearing Loss (N=14)

Age in Months

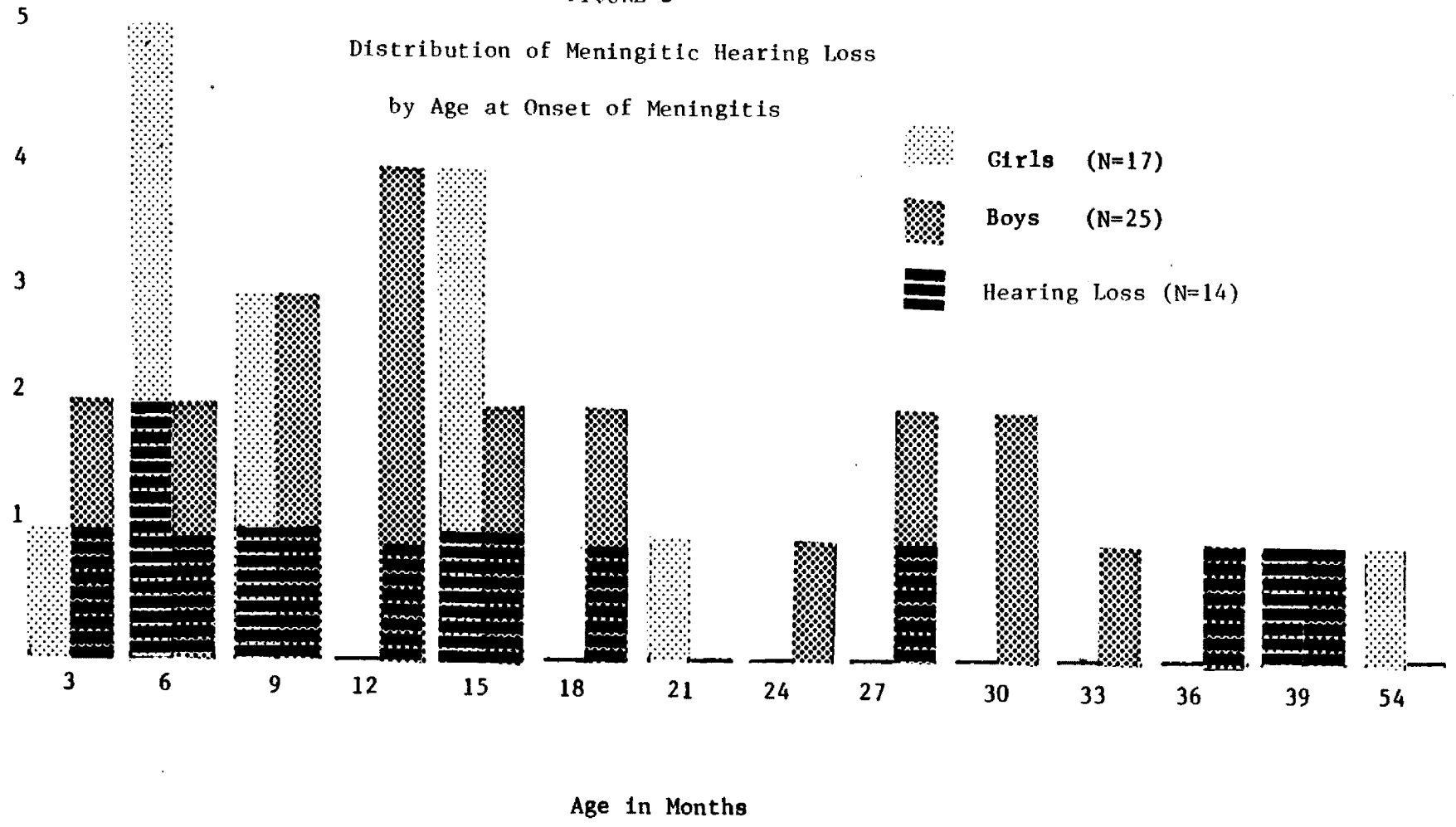Based on laboratory and audiological data, a contingency table was constructed.

TABLE IV.

Contingency Table for $\underline{X}^2$

Hearing Loss as a Function of

Cerebrospinal Fluid Glucose at Hospital Admission

	Hearing Loss		No Hearing Loss		Total	
	No.	(%)	No.	(%)	No.	(%)
CSF glucose ≥20 mg/100 ml	0	(0%)	16	(38%)	16	(38%)
CSF glucose <20 mg/100 ml	14	(33%)	12	(29%)	26	(62%)
Total	14	(33%)	28	(67%)	42	(100%)

$\underline{X}^2 = 12.9231$; the probability of $\underline{X}^2 > 12.9231 = 0.0003$; $p < 0.05$.

The association between cerebrospinal fluid glucose at hospital admission and hearing loss was not statistically significant. (See Appendix 1.)

2. Results

The incidence of hearing loss in this population is 33%, which is slightly higher than, but still within the range reported in the literature.

A $\underline{x}^2$ test for independence indicated no statistically significant relationship between cerebrospinal fluid glucose level at hospital admission and hearing loss. Additionally, relationships between hearing loss and cerebrospinal fluid glucose level at hospital admission, while not subjected to statistical analysis, were clearly not significant.

3. Discussion

The rather high incidence of hearing loss in this subject population may be attributable to the precise, capable audiological evaluation these children received. Dependable, repeatable responses were obtained, frequently for each ear independently. This is particularly note-wothy in light of the physical status of the children, who were desperately ill youngsters. Furthermore, normal healthy children at this age have been labelled "difficult to test."[22] Therefore, clinical expertise may account for identification of unilateral, high-frequency, or mild hearing losses in infants which might have gone undetected in another setting.

Another aspect of the audiological evaluation of these pediatric patients concerns evaluation of auditory attention. As Kresky[17] and others[25,30] have pointed out, post-meningitic children have demonstrated difficulty processing or integrating information. Of the 28 children who were determined to have hearing within normal limits, three, or approximately 7% showed significant deficits in auditory attention. These deficits might include ability during testing to respond to music, speech, or warbled tones at expected levels, but significantly decreased or delayed responses to pure tones. In light of these findings, children who have recovered from meningitis with their hearing acuity intact, may still suffer from difficulties and/or delays in processing and integration of auditory stimuli.

Although statistical analysis revealed no correlation between cerebrospinal fluid glucose level at admission and hearing loss, inspection of these data indicate a pattern. Of the 26 children with cerebrospinal fluid glucose level below 20 mg/100ml, fourteen children, or 54%, showed evidence of hearing loss. Therefore, although a significant correlation was not demonstrated, slightly more than half the children presenting cerebrospinal fluid glucose level below 20mg/100ml gave evidence of hearing loss. From these data, it can be inferred that 50% ore more of children treated for H. influenzae meningitis who present cerebrospinal fluid glucose less than

20mg/100ml may suffer hearing loss.

4. Summary and Conclusions

Meningitis was at one time a serious threat to the lives
of young children. Although the nature of the disease is
still critical, mortality due to this disease has decreased
in recent years. However, the type and severity of possible
sequelae to meningitis are overwhelming; successful prediction
of neurological residua is limited. Further recovery may be
lengthy and may involve fluctuation in ability or acuities
for months or even years following resolution of the initial
infection.[2,24,28]

In this study, hospital records were reviewed for 42
children treated for Haemophilus influenzae meningitis at
Denver Children's Hospital. Analysis of data gathered from
those records indicates that of the children with significantly
low cerebrospinal fluid glucose (20mg/100ml) on hospital
admission, slightly greather than 50% were shown to suffer
hearing loss. Therefore, children who meet these criteria
should be considered at-risk for hearing loss due to
Haemophilus influenzae meningitis.

Additional questions are raised with respect to the
material covered here:

    1) Is there a method of identifying different
        destructive mechanisms responsible for loss

of hearing? If so, would such information be of value in predicting with greater accuracy those patients likely to give evidence of hearing loss? Do these destructive processes respond to different treatment regimes, particularly steroid therapy?

2)   Is the lack of significant correlation noted due, at least in part, to different destructive processes? That is, if specific sites of infection were identified, would cerebrospinal fluid glucose level or some other diagnostic factor contribute to prediction of hearing loss?

3)   The data in this study may not be totally representative as a slightly higher percentage of hearing loss was identified in the subject population. Has this affected correlative studies?

The critical nature of meningitis and the swiftness of loss of neurological function is startling. Accurate, successful prediction of possible sequelae will be an important advance. Identification of probable sequelae and development of chemotherapeutic regimes to avert loss of function would result in tremendous differences in the lives of pediatric patients.

Contingency Table for $\underline{x}^2$

Hearing Loss as a Function of

Cerebrospinal Fluid Glucose at Hospital Addmission

	Hearing Loss		No Hearing Loss	
	Actual	Expected	Actual	Expected
CSF glucose 20 mg/100 ml	0	5.33	16	10.67
CSF glucose 20 mg/100 ml	14	8.67	12	17.33

BIBLIOGRAPHY

1. Adams, R.D., Kubik, C.S., Bonner, F.J. Clinical and pathological aspects of influenzal meningitis. Arch. Pediatr. 65:354-376, 408-441 (1948)

2. Berlow, S., Calderelli, D., Matz, G., Meyer, D., Harch,G. Bacterial Meningitis and sensori-neural hearing loss: a prospective investigation. Laryngoscope 9(9):1445-1452 (1980)

3. Bertolussi, R. Prognosis for survival in neonatal meningitis: A clinical and pathogenic review of 52 cases. Can. Med. Assoc. Jour. 165-168 (1968)

4. Dodge, P.R., Swatz, M. Bacterial meningitis--a review of selected problems, post-meningitic complications, and clinicopathologic corrections. N. Engl. Jour. Med. 272: 954-960, 1003-1009 (1965)

5. Downs, D.W., et al. Acoustic reflex hyperactivity in an infant with meningitis. Arch. Otoloayngol. 107(5):324-325 (1981)

6. Emmet, M. Sequelae of H. influenzae meningitis. Aust. Paediatr. Jour. 16(2):90-93 (1980)

7. Felgenhaur, K., et. al. The process dynamics of viral and bacterial diseases on the central nervous system. Jour. Neurol. Sci. 47(1):21-34 (1980)

8. Gamstarp, J., Klockhoff, E. Bilateral, severe, sensori-neural hearing loss after Haemophilus influenzae meningitis in childhood. Neuropaediatr. 5:2; 121-124 (1974)

9. Gary, N., Hartman, D., Little, M. Haemophilus influenzae memingitis: a follow-up study. Unpublished (1980)

10. Harris, H. Cerebrosphinal otorrhea and recurring meningitis: a report of three cases. Laryngoscope 88(10):1577-;585 (1978)

11. Herson, V., Todd,J. Prediction of morbidity in H. influenzae meningitis. Pediatrics 59:35 (1977)

12. Jahn, A.F., et al. Chronic middle-ear disease with intracranial extension. Jour. Otolaryngol. 8(1):77-84 (1979)

13. Johnson, Linda, Personal communication

14. Keane, W., Postic, W., Rowe, L., Konkle, D. Meningitis and hearing loss in children. Arch. Otolaryngol. 105:39 (1979)

15. Keim, R. J. Meningitis: the influence of routine otolaryngologic consultation on morbidity and mortality in 290 cases. Laryngoscope 88 (Part 2, Supplement 9): 1-30 (1978)

16. Koskiniemi, M. et al. H. influenzae meningitis: a comparison between chloramphenicol and ampicillim therapy with special reference to impaired hearing. Acta Pediatrica Scandinavia 67(1):17-24 (1978)

17. Kresky, B., et al. The incidence of neurological residua in children after recovery from bacterial meningitis. Arch. Ped. 79:63 (1962)

18. Meade, R. H. Complications of earache. Hospital Practitioner 14(8):79-81 (1979)

19. Merck, Sharp, and Dome Research. The Merck Manual of Diagnosis and Therapy. Merck and Company, Rahway, N.J. (1972)

20. Moxon, E. R., et al. Brain carbohydrate metabolism during experimental H. influenzae meningitis Ped. Res. 13(1):52-59 (1979)

21. Nadol, J. Hearing loss as a sequelae of meningitis. Laryngoscope 88:431 (1978)

22. Northern, J., Downs, M. Hearing in Children, Second Edition. Williams and Wilkins, Baltimore, MD (1979)

23. Nylen, G., et al. H. influenzae meningitis and hearing. International Jour Ped. Otorhinolaryngol. 1(1):97-101 (1979)

24. O'Leary, C. Bacterial meningitis in children. Unpublished (1981)

25. Paine, R., et al. Syndromes of "minimal brain dysfunction". Ped. Clin. N. Amer. 15:779 (1969)

26. Ravio, M. Hearing disorders after H. influenzae menigitis--a comparison of different drug regimes. Arch. Otolaryngol. 104(6):340-344 (1978)

27. Rosenhall, U., Nylen, O., Lindberg, J., Kakken, A. Auditory function after H. influenzae meningitis. Acta Otolaryngol. 85 (3-4):234-247 (1978)

28. Rosenhall, U. Hearing alteration following meningitis: hearing improvement. Ear and Hearing 1(4):185-190 (1980)

29. Sell, S., Merrill, R., Doyne, E., Zimshy, E. Psychological sequelae of bacterial meningitis: two controlled studies. Amer. Psych. 42:713 (1972)

30. Sell, S., Merrill, R., Doyne, E., Zimshy, E. Long-term sequelae of H. influenzae meningitis Pediatrics 49:206 (1974)

31. Sproles, E. Azerd, J., Williamson, C. Merrill, R. Meningitis due to H. influenzae: Long-term sequelae. Jour. Ped. 75:782 (1969)

32. Vernon, M. Meningitis and deafness: the problem, its physical, audiological, physiological, and educational manifestations in deaf children. Laryngoscope 77:1856 (1967)

33. Webb, W., Sell, S., Pate, T., Solomon, A. The sequelae of acute bacterial meningitis: a possible clue to early school problems. Jour. Spec. Ed. 2:327 (1969)

34. Wilber, L. Threshold measurement methods, in Hearing Assessment. W. Rintelmann, ed., University Park Press, Baltimore, MD (1979)

35. Wright, L. A method for predicting sequelae to meningitis. Amer. Psych. 33(11):1037-1039 (1978)