The Inferiorly Based Pharyngoplasty: Effects on Chronic Otitis Media with Effusion

PAUL H. M. SPAUWEN, M.D.
ROEL J. RITSMA, PH.D.
BOB J. C. HUFFSTADT, M.D.
HARM K. SCHUTTE, M.D.
ISABEL F. BROWN, F.R.C.S.(Ed).

Conflicting reports in the literature on the relationship between the inferiorly based pharyngeal flap and otitis media with effusion (OME) motivated a prospective study on 51 children between 3 and 12 years of age suffering from velopharyngeal incompetence. The patient group consisted of 34 children with cleft palate and 17 children with congenitally short palate. The male-female ratio was 37 to 14, showing no differences regarding physical disability. Each child underwent an inferiorly based pharyngoplasty. Repeated otoscopic, tympanometric, and audiometric examinations revealed that prior to pharyngoplasty half of the patients had bilateral chronic OME related to a conductive hearing loss of 30 to 35 dB. The disease was most frequently found in children with cleft palate ($p < 0.001$) and in children less than 6 years of age ($p < 0.05$). Bilateral chronic OME was independent of the type and side of cleft. Within 3 months of pharyngoplasty, a 60-percent decrease in the frequency of bilateral chronic OME was found. Children older than 6 years of age had most benefit with respect to OME ($p < 0.05$).

The biomechanical basis of the relationship between the pharyngeal flap and eustachian tube is discussed, focused on retropositioning of the levator muscle sling and reduced oronasal air leakage.

KEY WORDS: *otitis media with effusion, velopharyngeal incompetence, inferiorly based pharyngoplasty*

Patients with velopharyngeal incompetence (VPI) because of cleft palate or congenitally short palate frequently have bilateral hearing problems. Defective function of the eustachian tube followed by otitis media with effusion (OME) is the major cause of these hearing losses (Nassy, 1952; Bluestone et al, 1972; Holborow, 1975; Soudijn and Huffstadt, 1975; Beery, 1979; Rood and Stool, 1981). Surgical procedures such as pharyngeal flap may initiate an improvement in eustachian tube function, also seen following primary palatal repair. However, at present, little agreement has been found in the literature concerning the effects of pharyngeal flap on hearing. Generally, no disadvantages regarding hearing acuity are reported, whereas the percentages of improved hearing after pharyngoplasty vary from 0 to 65 percent (Graham and Lierle, 1962; Aschan, 1966; Bierens de Haan, 1971; Bennett, 1972; Le Worthy and Schliesser, 1975; Yules, 1975; Heller et al, 1978; Lendrum and Dhar, 1984) (Table 1).

The reasons for the inconsistency in the data are the variations among the patient groups tested with respect to age, basic pathology, type of pharyngoplasty, and definition of hearing pathology as well as hearing loss. To increase understanding of the relationship between pharyngoplasty and OME, a prospective investigation was performed in children requiring pharyngeal flap to treat their VPI. In this study, attention is focused on the presence or absence of OME before and after pharyngoplasty. Results are related

The authors are members of the Cleft Palate Team at the University Hospital Groningen in The Netherlands. Drs. Spauwen, Huffstadt, and Brown are affiliated with the Department of Plastic and Reconstructive Surgery. Within the ENT Department, Dr. Ritsma is Audiologist and Dr. Schutte is Phoniatrician.

TABLE 1 Changes in Hearing After a Pharyngeal Flap Operation

Studies	*Patients (N)*	*Hearing (percent of patients)*		
		Improved	*Constant*	*Impaired*
Graham and Lierle, 1962	43	0	100	0
Aschan, 1966	82	7	93	0
Bierens de Haan, 1971	9	65	29	6
Bennett, 1972	23	0	100	0
LeWorthy and Schliesser, 1975	53	26	70	4
Yules, 1975	69	12	88	0
Heller et al, 1978	51	some cases	—	—
Lendrum and Dhar, 1984	53	40	58	2

to physical disability (i.e., cleft palate or congenitally short palate) and age of these patients. Finally, the biomechanical basis of the relationship between a pharyngeal flap and the eustachian tube–middle ear system is discussed.

MATERIAL AND METHODS

The patient group consisted of 51 children with VPI between 3 and 12 years of age. Thirty-four children had previously received palatal repair including levator sling reconstruction, and 17 children had congenitally short palate. The male-female ratio was 37 to 14 showing no differences with regard to physical disability. The patient group was subdivided into two equal age groups from 3 to 6 years and 6 to 12 years of age in order to discover relevant differences in the preschool and school periods. The ratio of cleft palate to congenitally short palate in the younger age group (N1 = 25) was 19 to six and in the older age group (N2 = 26) 15 to 11.

The patients involved were repeatedly tested for speech and language development. Velopharyngeal function was tested by means of listener judgments, peroral observation, mirror test, and stethoscopic examinations. On the same occasion, otoscopic and audiologic examinations were performed. In all cases, a modified inferiorly based pharyngeal flap was used to correct the VPI (Huffstadt et al, 1970).

The protocol of the investigation was influenced by the need for strict criteria for defining hearing pathology. Recently, leading investigators (Fiellau-Nikolajsen, 1979; Poulsen and Tos, 1980; Felding, 1983; Gimsing and Bergholtz, 1983) concluded from studies on normal children that screening on one occasion only shows the incidence of OME at a given time and in a given age group. On another occasion, changes may be seen from OME to normal or the reverse. In order to trace incidental and chronic cases of OME, repeated screenings of the same patient group are necessary. In addition, bilateral OME more than unilateral OME accentuates the chronic nature of the disease.

Therefore, the criterion chosen in order to detect patients with chronic ear pathology was bilateral OME found at two separate occasions before or after pharyngoplasty. Thus, there were two examinations conducted within a 3-month period both prior to and following pharyngoplasty.

Otoscopy

Otoscopy was performed at every occasion on which hearing was tested. The procedure was employed by the medical speech pathologist and by one experienced otologist using the microscope. Cerumen was removed and the eardrum was described including the presence of calcifications, retraction, perforations, and tympanostomy tubes. Presence or absence of fluid in the middle ear cavity was explicitly mentioned.

Tympanometry

Tympanometry, performed with the aid of a Madsen Electronics ZO 73, was used as the most reliable and objective method for determining the status of the tympanic membrane and the middle ear. Four basic types of tympanograms were distinguished as classified by Jerger (1970) and as modified by Poulsen and Tos (1980) for achieving better segregation of chronic cases of OME: type A (normal curve; pressure +50/−90 mm H_2O; air-containing middle ear), type C_1 (normal curve; negative pressure −100/−199 mm H_2O), type C_2 (normal curve; negative pressure −200/−349 mm H_2O), and type B (flat curve; negative pressure greater than −350 mm H_2O: fluid-containing middle ear).

The diagnosis of OME was related to the finding of C_2- and B tympanograms, since these types are far more specific for chronic OME than C_1- tympanograms (Gimsing and Bergholtz, 1983).

Audiometry

Pure tone audiometry was employed using a clinical audiometer model AC 4 (Interacoustics).

Threshold values were determined for air and bone conduction at five frequencies (i.e., 250, 500, 1,000, 2,000, and 4,000 Hz). The threshold values were related to the international standardized zero (ISO 1964 standard). The accuracy of conductive hearing loss testing was in the order of 5 dB. For the present investigation average air-bone gaps within the speech range at 500, 1,000, and 2,000 Hz were calculated. Conductive hearing loss was defined as an average air-bone gap of 20 dB or more. At seven of the 204 examinations, since the child was too young to cooperate in pure tone audiometry, a paedo-audiometric test was performed by presenting sounds within octave bands around the frequencies of 500 and 1,000 Hz and sounds with frequencies over 3,000 Hz, in order to determine the presence of bilateral hearing loss of more than 30 dB as a result of decreased air conduction. The tympanometric, audiometric, and otoscopic results in individual patients were not entirely congruent, as is also known from studies on normal children (Fiellau-Nikolajsen, 1979; Liden and Renvall, 1980). Particularly, otoscopy showed a high discongruence with the tympanometric and audiometric results as far as bilateral chronic OME was concerned. The importance of tympanometry and audiometry has to be stressed, whereas it has to be admitted that otoscopy alone may be insufficient for detecting bilateral chronic OME. In this study only tympanometric data were used in diagnosing OME.

Results

Bilateral Chronic OME Prior to Pharyngoplasty

The data resulting from this investigation showed that bilateral chronic OME occurred in 26 of the 51 patients, which agrees with data from the literature (Graham and Lierle, 1962; Gabka, 1964; Soudijn, 1972; Le Worthy and Schliesser, 1975). The average bilateral conductive hearing loss amounted to 33 $\pm$ 8 dB, both ears having a loss of at least 20 dB.

Some specific features were recognized:

1. Bilateral chronic OME was found in 24 of our 34 patients with cleft palate as opposed to two out of 17 patients with congenitally short palate. This was statistically significant (X^2 test : $p < 0.001$).
2. Bilateral chronic OME occurred as frequently in patients with cleft lip and palate as in patients with isolated cleft palate, indicating that the cleft of the soft palate is the determining factor in the pathogenesis of bilateral ear disease. For the same reason, no relationship could be found between the side of the cleft and the side of OME. In patients with unilateral cleft, bilateral chronic OME was the usual type of pathology.
3. Children less than 6 years of age showed a significantly higher incidence of bilateral chronic OME than children 6 years of age and older (X^2 test : $p < 0.05$): 17 of 25 children (68 percent) in the younger age group had bilateral chronic OME as opposed to 5 percent in normal children of that age (Poulsen and Tos, 1980). In the older age group, bilateral chronic OME occurred in nine out of 26 children (35 percent) as opposed to 3 percent in the normal population (Gimsing and Bergholtz, 1983). The apparent decrease in the incidence of bilateral chronic OME with age did not depend on previous ENT treatment or on physical disability. Probably, spontaneous improvement during the school years may be responsible for this phenomenon, as is also seen in the normal population (Fiellau-Nikolajsen, 1979; Poulsen and Tos, 1980; Felding, 1983; Gimsing and Bergholtz, 1983).
4. Adenoidectomy preceding pharyngoplasty did not seem to have a favorable influence on bilateral chronic OME. Of 23 patients who had an adenoidectomy within 6 months before pharyngoplasty, 14 had bilateral OME; 13 of these patients continued to have bilateral chronic OME at the time of pharyngoplasty. This finding raises serious questions concerning the indication for removing a hypertrophic adenoid in order to treat chronic OME in VPI patients.

Bilateral Chronic OME After Pharyngoplasty

Ten patients with tympanostomy tubes or perforations were excluded from the calculations because they could not show changes after pharyngoplasty. Of the remaining 41 patients, 17 had bilateral chronic OME. Bilateral chronic OME reverted to normal in 10 of these 17 patients. This means that in 60 percent of patients with bilateral chronic OME, the continuance of chronic ear disease was broken within 3 months after pharyngoplasty. The audiometric data paralleled the tympanometric results.

The gross decrease in the frequency of OME contrasts with the expectation that without pharyngoplasty immediate improvement in OME would not occur.

In three of 24 patients with normal hearing

before pharyngoplasty, a transient deterioration in the hearing was noticed postoperatively, most likely due to reactive edema of the orifices of the eustachian tube. In these cases, recovery to normal was found within 6 to 9 months after pharyngoplasty. Seven children with bilateral chronic OME both before and after pharyngoplasty were adequately treated by inserting tympanostomy tubes.

Improvement in bilateral chronic OME after pharyngoplasty appeared to be confined almost exclusively to the cleft palate group, since bilateral chronic OME was primarily a problem of these patients as opposed to patients with congenitally short palate. Children older than 6 years of age had most benefit (X^2 – test : $p < 0.05$) with respect to their bilateral chronic OME, whereas younger children, preoperatively more frequently affected, appeared to be more resistant to postoperative improvement. The immediate effects of pharyngoplasty seem to accelerate the natural course of bilateral chronic OME.

DISCUSSION

The influences of a pharyngeal flap on the eustachian tube can be based upon the pathophysiology of the tubal and paratubal structures in VPI. Defective functioning of the eustachian tube can occur from many reasons (Fig. 1). They can be condensed into three main problems:

1. Reduction in middle ear ventilation by dysfunction of the tensor and levator veli palatini muscles as well as retrograde obstruction of the eustachian tube (Bluestone et al, 1972) caused by abnormal muscle paths, altered anatomy and quality of the eustachian tube, and adenoidal hypertrophy (Fara and Dvorak 1970; Dickson, 1975; Maue-Dickson et al, 1976; Rood and Stool, 1981; Shprintzen and Croft 1981; Beery, 1983)
2. Abnormal oronasal reflux leading to irritation of the orifices of the eustachian tube (Masters, et al 1960; Yules, 1975)
3. Susceptibility to infection, caused by adenoidal tissue or pharyngeal contents

According to recent research, tubal opening is probably achieved by coordinated contraction of both the tensor (Honjo et al, 1979; Cantekin et al, 1979; Honjo et al, 1980) and levator veli palatini muscles (Dickson, 1975; Kriens, 1975; Seif and Dellon, 1978; Honjo et al, 1980; Croft et al, 1981). The tensor muscle and particularly the medial part (called the dilator tubae muscle) (Dickson, 1975; Rood and Doyle, 1978) is directly related to the outward displacement of the membranous portion of the eustachian tube, whereas the levator muscle causes a lifting action on the medial cartilage of the tube causing an inward displacement related to opening of the eustachian tube orifices as confirmed by nasopharyngoscopic studies (Seif and Dellon, 1978; Honjo et al, 1980). This phenomenon could be observed in some of our patients (Fig. 2). In patients with VPI a forwardly directed levator muscle may be present (Holborow, 1962; Kriens, 1975). This may be responsible for the absence of a lifting action on the cartilaginous portion of the eustachian tube, thus closing the tube rather than assisting in its opening (Doyle et al, 1983). The functional obstruction is followed by a negative pressure in the middle ear, swelling of the mucosal lining of the eustachian tube, and fluid accumulation in the tympanic cavity (Bluestone et al, 1972, Bluestone et al, 1975, Dickson, 1975, Beery et al, 1979). Fluid accumulation, based on a secretory mechanism causes an acoustical impedance change of the

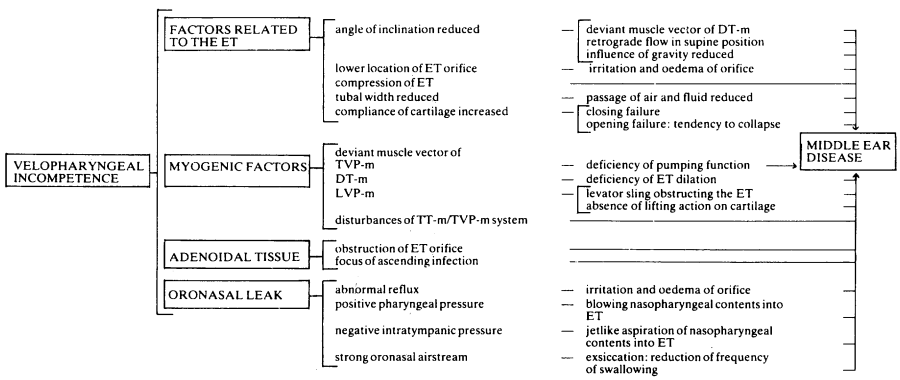

FIGURE 1 The causes of OME in VPI patients: ET = eustachian tube; TVP-m = tensor veli palatini muscle; Dt-m = dilator tubae muscle; LVP-m = levator veli palatini muscle; TT-m = tensor tympani muscle.

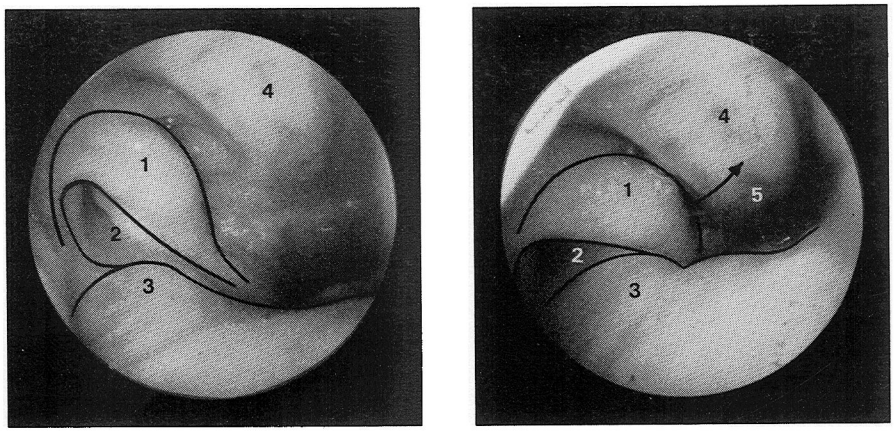

FIGURE 2 The eustachian tube orifice observed by nasopharyngoscopy in a girl aged 14 years (VPI after palatal repair); *A*, at rest, *B*, during contraction of the levator veli palatini muscle causing posteromedial displacement of the cartilage. 1-medial cartilage; 2-eustachian tube orifice; 3-levator muscle; 4-posterior pharyngeal wall; 5-velopharyngeal aperture.

middle ear system resulting in a range of conductive hearing losses that varies with frequency and duration of the presence of middle ear fluid. In addition, the accumulated viscid fluid may become a growth medium for micro-organisms resulting in suppurative otitis media.

An inferiorly based pharyngeal flap may influence the function of the eustachian tube at several levels:

1. The forward directed vector of the levator muscle may be corrected by backward traction of the soft palate because of the pulling force of the pharyngeal flap (Fig. 3). The muscular strength of the pharyngeal flap (Fara and Dvorak, 1970; Owsley et al, 1972; Mulder, 1976) may retroposition the levator sling. It is possible that the pharyngeal flap pulls the levator sling across a mechanical dead-point, realizing a better point of impact on the eustachian tube cartilage. This may also facilitate the effect of

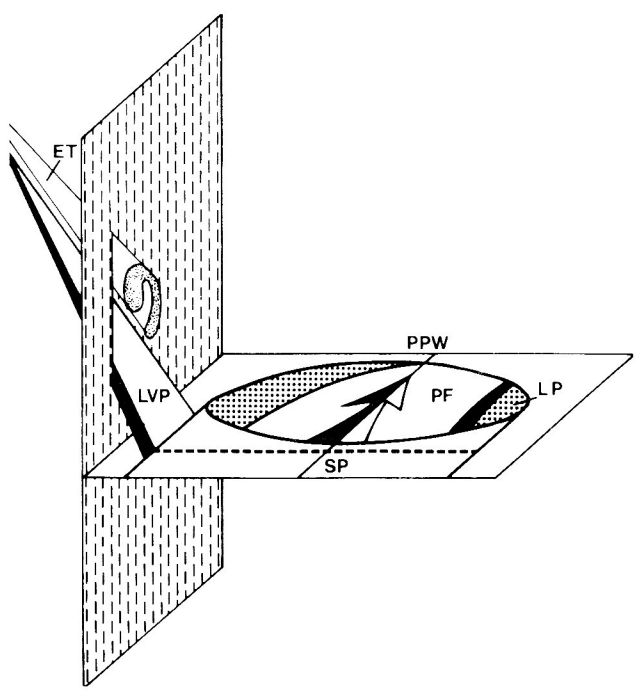

FIGURE 3 Dorsal traction of the posterior border of the soft palate, consequently followed by posterior displacement of the levator sling. Frontal view superior to the soft palate. ET-eustachian tube; PF-pharyngeal flap; LP-lateral port; LVP-levator muscle; SP-soft palate; PPW-posterior pharyngeal wall.

the contraction of the dilator tubae muscle on the membranous wall of the eustachian tube.

2. After achieving velopharyngeal competence by means of a pharyngeal flap, the oronasal reflux becomes diminished. Subsequently, the irritation of the eustachian tube orifices is eliminated.
3. After pharyngoplasty, the stimulus for adenoidal hypertrophy may be decreased, thus minimizing the chance of eustachian tube obstruction and of ascending infection. Further testing of this inference requires nasopharyngoscopic observation.

Finally, some remarks on the influence of the physical disability on OME have to be made. As opposed to data from Heller et al (1978) and Caldarelli (1978), the children with cleft palate in our study more frequently had bilateral chronic OME than the children with congenitally short palate. Although both cleft palate and congenitally short palate may be based upon the same causative pathology, the effects appear to be different. Both the basic anatomical-functional defect and the hearing pathology are less severe in patients with congenitally short palate.

In the literature, no consensus exists concerning the relationship between hearing and the type of cleft. Although some authors suggest that in children with a complete cleft the larger defect accounts for more hearing problems (Nassy, 1952; Masters et al, 1960; Bennett et al 1968; Komatsu et al, 1982), no differences between complete clefts and clefts confined to the palate were found in this study. The action of the levator sling per se on the eustachian tube appears to be the determining factor in causing hearing problems, independently of the type of cleft (Graham, 1964; Soudijn and Huffstadt, 1975; Webster and Eldis, 1978; Swigart, 1979).

CONCLUSIONS

1. The definition of hearing pathology in VPI should be based upon standardized criteria. As a proposal, bilateral chronic OME should be related to the repeated findings of bilateral C_2- and B tympanograms, whereas hearing loss should be related to average air-bone gaps of 20 dB or more measured at 500, 1,000 and 2,000 Hz. Otoscopy alone appears to be insufficient for detecting bilateral chronic OME.
2. Half of the patients with VPI had bilateral chronic OME as opposed to 5 percent of normal children.
3. Bilateral chronic OME occurred most frequently in children with cleft palate. Children less than 6 years of age showed a high incidence of bilateral chronic OME whereas during the school years a change in the persistance of bilateral chronic OME occurred, as is also seen in the normal population.
4. The likely reason for the high occurrence of bilateral chronic OME in VPI patients is the forward insertion of the levator muscle sling rather than a hypertrophic adenoid. In addition, adenoidectomy on otological grounds appears to be questionable in VPI patients.
5. A 60-percent decrease in bilateral chronic OME was demonstrated within 3 months of performing pharyngoplasty. Children older than 6 years of age had most benefit with respect to OME. The immediate effects of pharyngoplasty seem to accelerate the natural course of the disease.
6. The most obvious explanations for the decreased incidence of bilateral chronic OME after pharyngoplasty appears to be: (a) retropositioning of the levator muscle sling, facilitating the lifting action of this sling on the eustachian tube cartilages as well as dilation of the membranous wall by the dilator tubae muscle, and (b) reduced oronasal leakage.

REFERENCES

ASCHAN G. (1966). Hearing and nasal function correlated to postoperative speech in cleft palate patients with velopharyngoplasty. Acta Otolaryngol 6:371-379.

BEERY QC, DOYLE WJ, CANTEKIN EI. (1979). Longitudinal assessment of Eustachian tube function in children. Laryngoscope 89:1446-1456.

BEERY QC. (1983). Pharyngeal wall motion in prosthetically managed cleft palate adults. Cleft Palate J 20:7-17.

BENNETT M, WARD RH, TAIT CA. (1968). Otologic-audiologic study of cleft palate children. Laryngoscope 78:1011-1019.

BENNETT M. (1972). The older cleft palate patient. Laryngoscope 82:1217-1225.

BIERENS DE HAAN M. (1971). De invloed van een farynxplastiek volgens Rosenthal op het gehoor. Personal Communication.

BLUESTONE CD. (1971). Eustachian tube obstruction in the infant with cleft palate. Ann Otol Rhinol Laryngol 80:1-30.

BLUESTONE CD, WITTEL RA, PARADISE JL. (1972). Roentgenographic evaluation of Eustachian tube function in infants with cleft and normal palates. Cleft Palate J 9:93-99.

BLUESTONE CD, CANTEKIN EI, BEERY QC. (1975). Eustachian tube ventilatory function in relation to cleft palate. Ann Otol Rhinol Laryngol 84:333-338.

CALDARELLI DD. (1978). Incidence and type of otopathology associated with congenital palatopharyngeal incompetence. Laryngoscope 88:1970-1984.

CANTEKIN EI, DOYLE WJ, REICHERT TJ, PHILIPS DC, BLUESTONE CD. (1979). Dilation of the Eustachian tube by stimulation of the mandibular nerve. Ann Otol Rhinol Laryngol 88:40–51.

CROFT CB, SHPRINTZEN RJ, RAHOFF SJ. (1981). Patterns of velopharyngeal valving in normal and cleft palate subjects. A multiview videofluoroscopic and naso-endoscopic study. Laryngoscope 91:265–271.

DICKSON DR. (1975). Anatomy of the normal velopharyngeal mechanism. Clin Plastic Surg 2:235–247.

DOYLE WJ, KITAJIRI M, SANDO I. (1983). The anatomy of the auditory tube and paratubal musculature in a one month old cleft palate infant. Cleft Palate J 20:218–226.

FARA M, DVORAK J. (1970). Abnormal anatomy of the muscles of palatopharyngeal closure in cleft palate. Plast Reconstruct Surg 46:488–494.

FELDING JU. (1983). The longitudinal Hjorring-population study. Scand Audiol 17:55–59.

FIELLAU-NIKOLAJSEN M. (1979). Tympanometry in three-year-old children. Scand Audiol 8:181–185.

GABKA J. (1964). Hasenscharten und Wolfsrachen. Entstehung, Behandlung und Operationsverfahren. Zweite Auflage, Berlin: Walter de Gruyter & Co.

GIMSING S, BERGHOLTZ L. (1983). Audiologic screening of seven and ten-year-old children. Scand Audiol 12:171–177.

GRAHAM MD. (1964). Longitudinal study of ear disease and hearing loss in patients with cleft lips and palates. Ann Otol Rhinol Laryngol 73:34–46.

GRAHAM MD, LIERLE DM. (1962). Posterior pharyngeal flap palatoplasty and its relationship to ear disease and hearing loss. Laryngoscope 72:1750–1755.

HELLER JC, GENS GW, CROFT CB. (1978). Conductive hearing loss in patients with velopharyngeal insufficiency. Cleft Palate J 15:246–253.

HOLBOROW CA. (1962). Deafness associated with cleft palate. J Laryngol Otol 76:762–773.

HOLBOROW CA. (1975). Eustachian tube function. Changes throughout childhood and neuromuscular control. J Laryngol Otol 89:47–55.

HONJO I, OKAZAKI N, KUMAZAWA T. (1979). Experimental study of the Eustachian tube function with regard to its muscles. Acta Otolaryngol (Stockh) 87:84–89.

HONJO I, OKAZAKI N, KUMAZAWA T. (1980). Opening mechanism in the Eustachian tube. Ann Otol Rhinol Laryngol 89:25–27.

HUFFSTADT AJA, BORGHOUTS JMHM, MOOLENAAR-BIJL AJ. (1970). Operative treatment of rhinolalia: a review of 139 pharyngoplasties. Br J Plast Surg 1970; 23:150–154.

JERGER J. (1970). Clinical experience with impedance audiometry. Arch Otolaryngol 92:311–315.

KOMATSU U, GENBA R, KOKAMA G. (1982). Morphological studies of the velopharyngeal orifice in cleft palate. Cleft Palate J 19:275–280.

KRIENS O. (1975). Anatomy of the velopharyngeal area in cleft palate. Clin Plast Surg 2:261–283.

LENDRUM J, DHAR BK. (1984). The Orticochea dynamic pharyngoplasty. Br J Plastic Surg 37:160–168.

LEWORTHY GW, SCHLIESSER H. (1975) Hearing acuity before and after pharyngeal flap procedure. Plast Reconstruct Surg 56:49–51.

LIDEN G, RENVALL U. (1980). Impedance and tone screening of school children. Scand Audiol 9:121–126.

MASTERS FW, BINGHAM HG, ROBINSON DW. (1960). The prevention and treatment of hearing loss in the cleft palate child. Plast Reconstr Surg 25:503–519.

MAUE-DICKSON W, DICKSON DR, ROOD SR. (1976). Anatomy of the Eustachian tube and related structures in age-matched human fetuses with and without cleft palate. Trans Am Acad Ophthalmol Otolaryngol 82:159–164.

MULDER JW. (1976). Velopharyngeal function and speech. Thesis, Groningen, The Netherlands.

NASSY EA. (1952). Gehoorsafwijkingen bij verhemeltespleet. Thesis, Amsterdam, The Netherlands.

OWSLEY JQ, CREECH BJ, DEDO HH. (1972). Poor speech following the pharyngeal flap operation: etiology and treatment. Cleft Palate J 9:312–318.

POULSEN G, TOS M. (1980). Repetitive tympanometric screenings of two-year-old children. Scand Audiol 9:21–28.

ROOD SR, DOYLE WT. (1978). Morphology of the tensor veli palatini, tensor tympani and dilator tubae muscles. Ann Otol Rhinol Laryngol 87:202–210.

Rood SR, Stool SE. (1981). Current concepts of the etiology, diagnosis and management of cleft palate related otopathologic disease. Otolaryngol Clin North Am 14:865–884.

SEIF S, DELLON AL. (1978). Anatomic relationships between the human levator and tensor veli palatini and the Eustachian tube. Cleft Palate J 15:329–336.

SHPRINTZEN RJ, CROFT CB. (1981). Abnormalities of the Eustachian tube orifice in individuals with cleft palate. Int J Pediatr Otorhinolaryngol 3:15–23.

SOUDIJN ER. (1972). Slechthorendheid bij patiënten met aangeboren afwijkingen van lip, bovenkaak en gehemelte. Ned Tijdschr Geneesk 33:1482.

SOUDIJN ER, HUFFSTADT AJC. (1975). Cleft palates and middle ear effusions in babies. Cleft Palate J 12:229–233.

SWIGART E. (1979). Hearing sensitivity of adults with cleft lip and/or palate. Cleft Palate J 16:72–80.

WEBSTER JC, ELDIS F. (1978). Ear disease in relation to age in the cleft palate child and adolescent. Clin Otolaryngol 3:455–461.

YULES RB. (1975). Current concepts of treatment of ear disease in cleft palate children and adults. Cleft Palate J 12:315–321.