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ORIGINAL ARTICLES

ECoG results in perilymphatic fistula: Clinical and experimental studies

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Patients with perilymphatic fistula have been described as having symptoms similar to Meniere's disease and endolymphatic hydrops. Direct clinical or experimental evidence linking the two inner ear disorders has been lacking. An enhancement of the summating potential observed with electrocochleography suggests a diagnosis of ELH in both of these inner ear disorders. In this study, ECoG results of 27 patients with surgically confirmed PLF are reported. Fourteen patients with surgically confirmed spontaneous PLF had abnormal ECoG. Six of these 14 patients had normal hearing. The ECoG changes in patients with Meniere's disease and those with surgically confirmed PLF are identical, indicating the underlying pathologic change in both is hydrops. But there is no specific diagnostic abnormality on ECoG that differentiates these two inner ear disorders. Also, an experimental model of PLF was developed and studied in guinea pigs. "Inactive" PLF is defined as "an opening was made into the cochlea, but if no perilymph moved out through the fistula, it was defined as inactive" An "active" PLF occurs when perilymph actually moves from the inner ear out to the middle ear. ECoGs were recorded before and after creation of an "active" PLF. ECoG abnormalities were seen in "active" PLF *and* correlated with histologic data demonstrating ELH. An abnormally enhanced summating potential was demonstrated after active removal of perilymph through the experimentally created fistula. Cochlear duct histology showed hydropic distention of Reissner's membrane in the experimental ears and no changes in the membranous labyrinths of the unoperated, control ears. Experimental PLF, with histologically confirmed secondary ELH, produced changes similar to those observed in guinea pigs with ELH experimentally produced on a primary basis. Preliminary experimental data suggest ECoG may be helpful in differentiating (acute) PLF and (secondary) ELH. ECoG obtained in intensity series showed greatest intensity effect at 40 dB and no effect at 60 dB. Further studies using similar intensity series information will be elucidated to better determine the significance of these observations. The underlying common denominator for the two disorders of inner ear fluid dynamics is ELH. ELH, either primary or secondary, is indicated by the same ECoG changes of enhancement of the summating potential. Since the abnormal ECoG can occur in patients with vertigo but no clinical hearing loss, it is presumed ELH can occur in the vestibular labyrinth as well as in the cochlear duct. (OTOLARYNGOL HEAD NECK SURG 1988;99:435.)

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Clinical awareness of perilymph fistula (PLF) arising from longstanding chronic ear disease—specifically erosive cholesteatoma—goes back to the nineteenth century.[1] A clinical awareness of PLF as a result of severe head and/or temporal bone trauma also goes back to the nineteenth century.[1] Awareness that less obvious head trauma can cause labyrinthine concussion with acute, active PLF is of more recent vintage.[2] A clinical and theoretic awareness of post-stapedectomy PLF was first noted less than 30 years ago,[3-5] after John Shea[6] introduced stapedectomy. Lewis[3] described the phenomenon in 1961. The incidence of PLF as a surgical complication of stapes surgery was reported to be as high as 7%.[7] In 1967, Howard House[4] noted the symptom similarities shared by post-stapedectomy PLF patients and patients with Meniere's disease. House also proposed a cochlear hydrodynamic explanation for the similar symptom complex between these two apparently distinct inner ear pathologic groups. House[4] stated in 1967:

> "the endolymphatic system can function normally only if the pressure of the endolymphatic and perilymphatic fluid is the same. Any imbalance in this equalization over a period of time will result in dilation of the endolymphatic system (into the perilymphatic system), thereby producing "artificial" (or secondary) hydrops."*

Stroud and Calcaterra[9] first noted spontaneous PLF. Our clinical and theoretic awareness of traumatic PLF was first noted in 1968 by Fee.[8] In 1968, Fee described the first reported cases of spontaneous PLF, which was discovered inadvertently at the time of labyrinthectomy for intractible vertigo in a patient with Meniere's disease. The fistula repair eliminated the symptoms of vertigo, precluding the need for a destructive procedure. A more detailed review of the theoretic, experimental, and clinical aspects of perilymphatic fistula has been reported by the authors elsewhere.[10]

Also in 1968, Simmons[12] presented a theory of intracochlear membrane ruptures, along with tears and defects of the round and/or oval window. This double membrane rupture theory helps to explain the variation in symptoms between oval window fistulas and round window fistulas. Simmons expanded on this concept, which suggests that a leak of perilymph from a window rupture could cause intralabyrinthine membrane tears or vice versa. Simmons[13] demonstrated in 1962 that rupture of the round window in cats did not necessarily cause any significant hearing loss.

In 1974, Harker et al.[14] showed that by increasing cerebrospinal fluid (CSF) pressure as little as 120 mm of water, the round window membrane could rupture, demonstrating a route for explosive rupture of the round window membrane. Goodhill's theory[15] of explosive route for spontaneous fistula via the cochlear aqueduct and internal auditory canal was substantiated in laboratory animals. Some early reports showing that patients with Meniere's syndrome had surgically confirmed fistula were reported in the early 1970s, further documenting the connection between the symptoms of Meniere's disease and PLF.[16] In 1982, Allen[17] described that an active fistula with secondary endolymphatic hydrops are interrelated. The interrelated mechanism is the result of changes in intralabyrinthine fluid pressures.

Electrocochleography (ECoG) became important in the clinical work of Gibson and colleagues[18,19] on Meniere's disease. ECoG abnormalities provided an objective assessment of endolymphatic hydrops (ELH). These abnormalities were further corroborated by other researchers who showed that an enhanced negative summating potential was indicative of distortion and distention within the cochlear duct and ELH. These ECoG changes were later corroborated in animal studies of ELH as well.[20,21] ECoG changes are most helpful in the objective diagnosis of presumed ELH in Meniere's disease.[22-25]

Use of ECoG in diagnosing secondary hydrops caused by PLF has not been reported. In this study, patients with surgically confirmed ("active") PLF who

Table 1. Symptoms

N = 27*	
Vertigo	n = 20
Hearing	n = 17
Hearing	n = 15
Pressure	n = 15
Tinnitus	n = 13
Imbalance	n = 6

*22 patients (82%) reported two or more symptoms.

Table 2. Cause

N = 27	
Physical trauma	n = 15
Barotrauma	n = 4
Exertion	n = 4
Drug overdose	n = 1
Unknown	n = 3

*(Parentheses added)

Table 3. Location of fistula

N = 27	
Right ear	n = 14
Left ear	n = 13
Round window	n = 9
Oval window	n = 9
Both windows	n = 2
Fluid accumulation/ No discrete defect	n = 7

Table 4. Test results

N = 27	
SRT > 25 dB	n = 6 (22%)
Abnormal ECoG	n = 14 (52%)
+Obj (ENG) PLF Test	n = 14 (52%)
+Subj PLF Test	n = 13 (48%)
+O/S PLF Test	n = 10 (37%)
−O/S PLF Test	n = 10 (37%)
N = 23	
RVR > 20%	n = 7 (30%)

Table 5. ECoG/hearing test results

N = 27	
Abnormal ECoG and normal SRT	n = 11 (41%)
Normal ECoG and normal SRT	n = 10 (37%)
Abnormal ECoG and Abnormal SRT	n = 3 (11%)
Normal ECoG and Abnormal SRT	n = 3 (11%)

had preoperative ECoG were studied to further evaluate the relationship between "active" PLF and ELH. The clinical findings and concepts were also studied and tested in guinea pig PLF models, using ECoG monitoring with electrophysiologic analysis of inner ear function. Morphologic analysis was made of the cochlear duct and ELH in these animals in which PLF was produced experimentally.

CLINICAL STUDY

Methods (Patients). Between 1980 and 1986, twenty-seven (27) patients operated on for suspected PLF had surgically confirmed PLF. Only patients with preoperative ECoG and no history of other otologic surgery were included in this study. Patients manifested and reported vertigo (n = 20), hearing loss (n = 17), aural pressure (n = 15), tinnitus (n = 13), and imbalance (n = 6). Most patients reported a combination of two or more symptoms (Table 1). The causes of PLF (Table 2) were: physical trauma (n = 15), barotrauma (n = 4), physical exertion (n = 4), drug overdose (n = 1), and other etiologies (n = 3).

All patients were given a baseline audiometric battery of tests, puretones (PT), speech reception threshold (SRT), and speech discrimination scores (SDS), ECoG, and fistula test (subjective and objective). Most patients (n = 23) were also tested with electronystagmography (ENG). ECoG was considered positive for fistula when summating potential (SP)/action potential (AP) amplitude ratio equalled or exceeded 0.50. SP amplitude was identified as the number of microvolts from prestimulus baseline to the SP deflection entry point on AP. AP amplitude was measured in microvolts from pre-stimulus baseline to maximum AP deflection. Subjective fistula test was considered positive when air-pressure change in the ear canal of the affected ear caused an observable response of disequilibrium or vertigo. Objective (ENG) fistula test[26] was considered positive when ear canal air-pressure change produced recordable nystagmus of at least seven degrees per second. ENG caloric test was deemed abnormal when a reduced vestibular response of 20% or greater was found for the affected ear.

Results (Patients). The average age of patients with surgically confirmed PLF in this study was 36 years old; the age range was from 11 to 57 years. PLF occurred in the right ear in fourteen (14) patients and in the left ear in thirteen (13) patients. Nine (9) round window (RW) and nine (9) oval window (OW) fistulas were observed, and in two patients both windows showed active fistulas. In seven (7) patients, clear fluid consistent with a PLF was observed, but no discrete defect was identified at the time of surgery (Table 3).

Audiometric results showed an abnormal speech reception threshold (SRT) of 25 dB or greater in only six of 27 patients, or 22%. SP/AP ratio of 0.50 or greater occurred in 14 of 27 patients (52%). Subjective fistula test was also positive in 13 patients (48%), and objective (ENG) fistula test was positive in 14 patients (52%). In ten patients (37%), both objective and subjective fistula tests were positive, and in ten patients both objective and subjective fistula tests were negative. Caloric irrigation produced a reduced vestibular response in nine of 23 patients (39%). Preoperative baseline test results are summarized in Table 4.

Abnormal ECoG occurred with normal SRT in 41% of cases, and normal ECoG occurred with normal SRT in 37% of cases. Abnormal ECoG was found when the SRT was also abnormal in only three cases, and ECoG was normal when the SRT was abnormal in only three cases. Comparisons of ECoG and audiometric test results are given in Table 5.

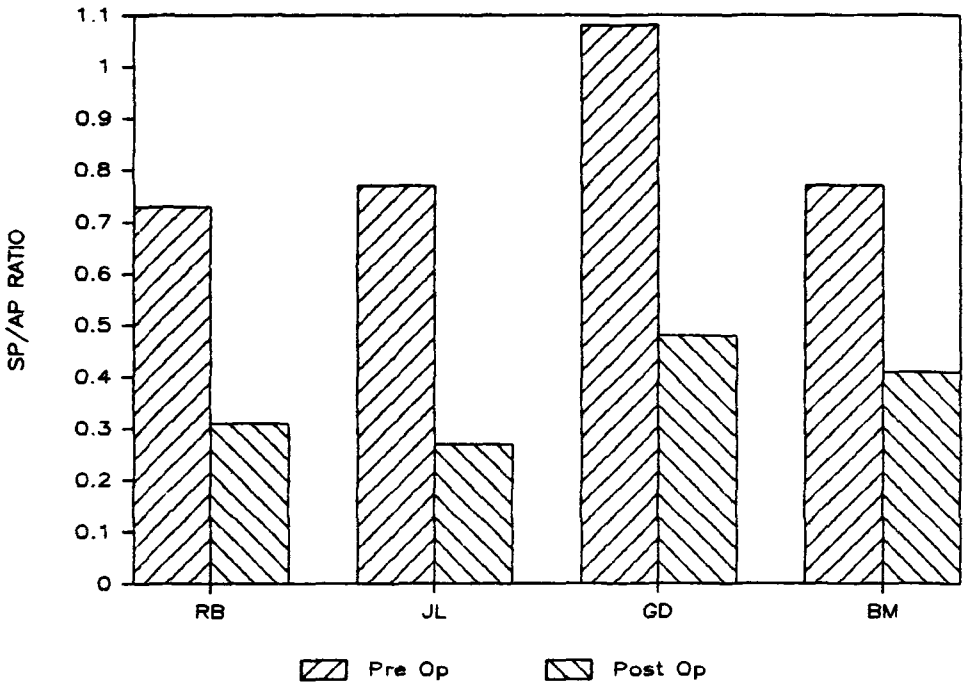

Fig. 1. Patients' SP/AP ratios: preoperative vs. postoperative ECoG.

Table 6. ENG fistula/caloric test results

N = 27	N = 23
+ENG PLF Test and +Caloric	n = 4 (17%)
+ENG PLF Test and −Caloric	n = 9 (39%)
−ENG PLF Test and +Caloric	n = 5 (22%)
−ENG PLF Test and −Caloric	n = 5 (22%)

Table 7.

ECoG Correlations
+ ECoG × + Obj. PLF test = 0.15
+ ECoG × + Subj. PLF test = 0.19
+ ECoG × + ENG = 0.22
+ ECoG × Pressure = 0.18*

*Complaint of pressure not necessarily reported at time of each ECoG test.

ENG fistula test was positive when the caloric result was abnormal in only 17% of the patients, but in 39% a normal caloric response occurred with a positive fistula test. Fistula test was negative when the caloric response was abnormal in five patients, and both tests were negative in five patients. Comparisons of objective (ENG) fistula test and caloric irrigation results are given in Table 6.

Correlation coefficients (Pearson's) were calculated for abnormal ECoG compared to (1) fistula test, (2) caloric response, and (3) report of aural pressure (Table 7). Correlation coefficients for all comparisons are low.

Pre- and postoperative ECoGs are shown for four subjects in Fig. 1. In all four cases, SP/AP ratios were enlarged before surgical repair for PLF, and after surgery the ratios were normal. Although this graph represents a small sample of the patients studied, it gives objective evidence that cochlear electrophysiologic changes can be changed by time and/or PLF surgical repair; i.e., that ECoG changes can fluctuate from abnormal to normal, just like the symptoms of hydrops.

EXPERIMENTAL PERILYMPH FISTULA

Methods (Guinea Pigs). Twelve (12) pigmented guinea pigs (250 to 350 g each) were anesthetized intramuscularly with ketamine hydrochloride and xylazine hydrochloride (Rompun) (ratio of 5:1; 50 mg/kg body weight), and anesthesia maintained with hourly injection of ketamine hydrochloride (20 mg/kg BW). The animals were held in lateral decubitus position with a guinea pig headholder, and ventral entry through the experimental (right) auditory bulla was performed. A silver-tipped needle electrode was inserted into a saline-saturated cotton pledget at the round window niche. Acoustic transients (clicks) stimulated the ear via a 2-cm long silicone rubber tubing extension connected

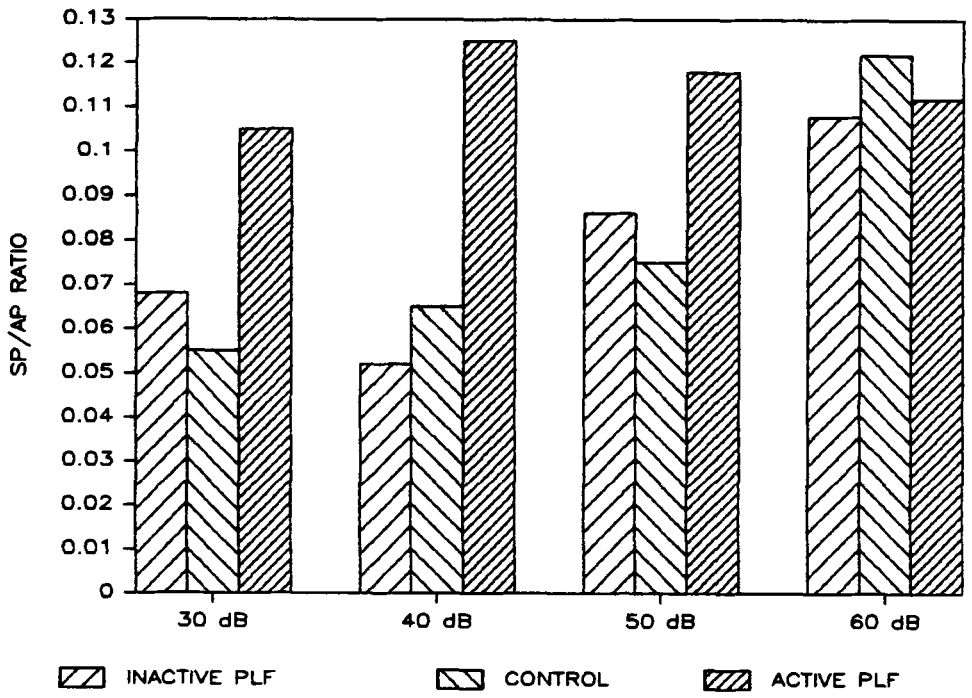

Fig. 2. SP/AP ratio; experimental PLF in guinea pigs.

to an infant ear speculum housed in an MX-41/AR cushion surrounding a TDH-49 earphone.

This animal model of "inactive" and "active" PLF measured electrophysiologic parameters as well as morphologic changes. Potentials N1 and N2 were recorded on either a Nicolet CA 1000 or Cadwell 5200 Signal-Averager, after transducing 100 condensation and 100 rarefaction 0.1 msec rectangular pulses. A series of four baseline waveforms were obtained after stimulation at 30, 40, 50, and 60 dB (re: N1 threshold). Threshold was identified as the lowest amplitude waveform evoked by the minimum intensity acoustic stimulus (*Condition 1*—Control). The cochlear aqueduct was then obliterated and blocked with bone wax after drilling was performed medially from within the tympanic bulla, beginning at a point 2-mm caudal to the basal cochlear turn (*Condition 2*—Control with blocked cochlear aqueduct). A 0.30 mm hole was drilled through the bony cochlea into the scala tympani of the basal turn (*Condition 3*—Experimental "inactive" PLF). In *Condition 4*, the cochlear aqueduct was blocked and the "inactive" PLF converted to an "active" PLF when five microliters of perilymph were removed. Waves N1 and N2 and the summating potentials were recorded in all conditions. After all electrophysiologic recordings were performed on both ears, the cochlea was perfused with glutaraldehyde, removed, and prepared for microscopic examination and photomicrography in a Wild-Heerbrugg dissecting microscope.

Results (Guinea Pigs). The purpose of this investigation was to obtain electrophysiologic and cochlear histologic information from an experimentally induced "inactive" and "active" PLF. The ECoGs were analyzed in three ways for all four conditions:

1. summating potential/action potential amplitude ratio;
2. action potential duration; and
3. summating potential polarity/amplitude.

In addition, light and electron photomicrographs of guinea pig inner ears provided morphologic evidence of cochlear membrane changes between the normal control groups, the "inactive" PLF experimental group, and the "active" PLF group.

Experimental ECoG results differed from control and pre-experimental ECoGs for the "active" PLF group in two respects: (1) summating potential/action potential amplitude ratio, and (2) summating potential polarity/amplitude. However, no change was observed among any of the four groups in the action potential duration.

Means of combined SP/AP ratios of all twelve animals are illustrated in Fig. 2. This graph shows an enhanced SP/AP ratio at 30, 40, and 50 dB, but not at 60 dB. The enhanced SP/AP ratio at 40 dB exceeded

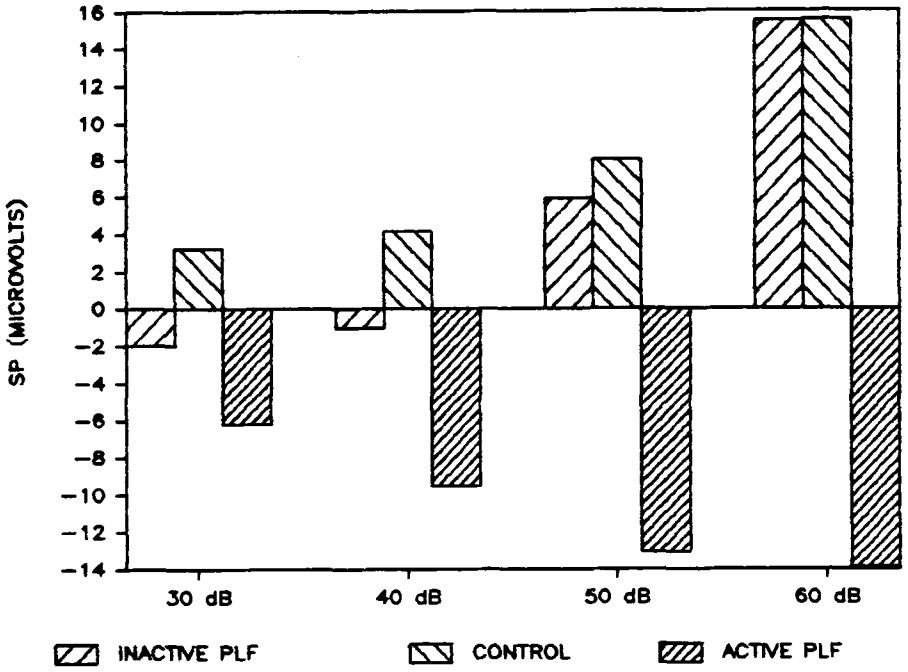

Fig. 3. SP polarity/amplitude; experimental PLF in guinea pigs.

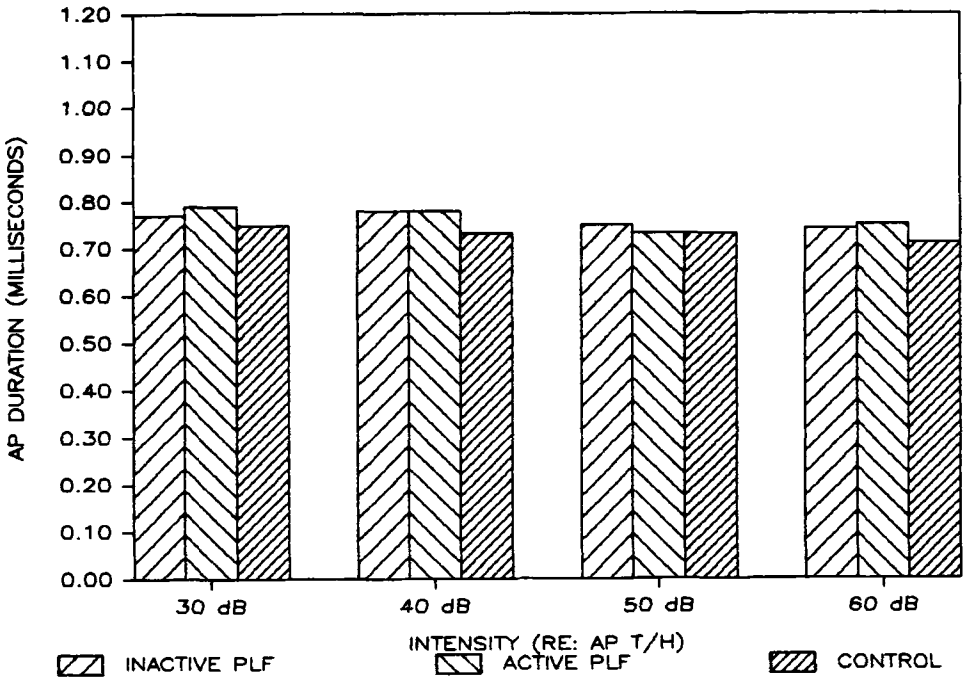

Fig. 4. AP duration; experimental PLF in guinea pigs.

that of the pre-experimental and control conditions by two standard deviations. Therefore, the experimentally induced enhancement of the summating potential by an "active" PLF is most evident at 40 dB SL (re: N1 threshold), and not evident at all at 60 dB.

Means of the combined SP amplitude/polarity values were calculated in microvolts for the twelve animals and are graphed in Fig. 3. Pre-experimental PLF lesion and control SP is slightly negative (< -0.20 microvolts) at 30 and 40 dB, and becomes increasingly pos-

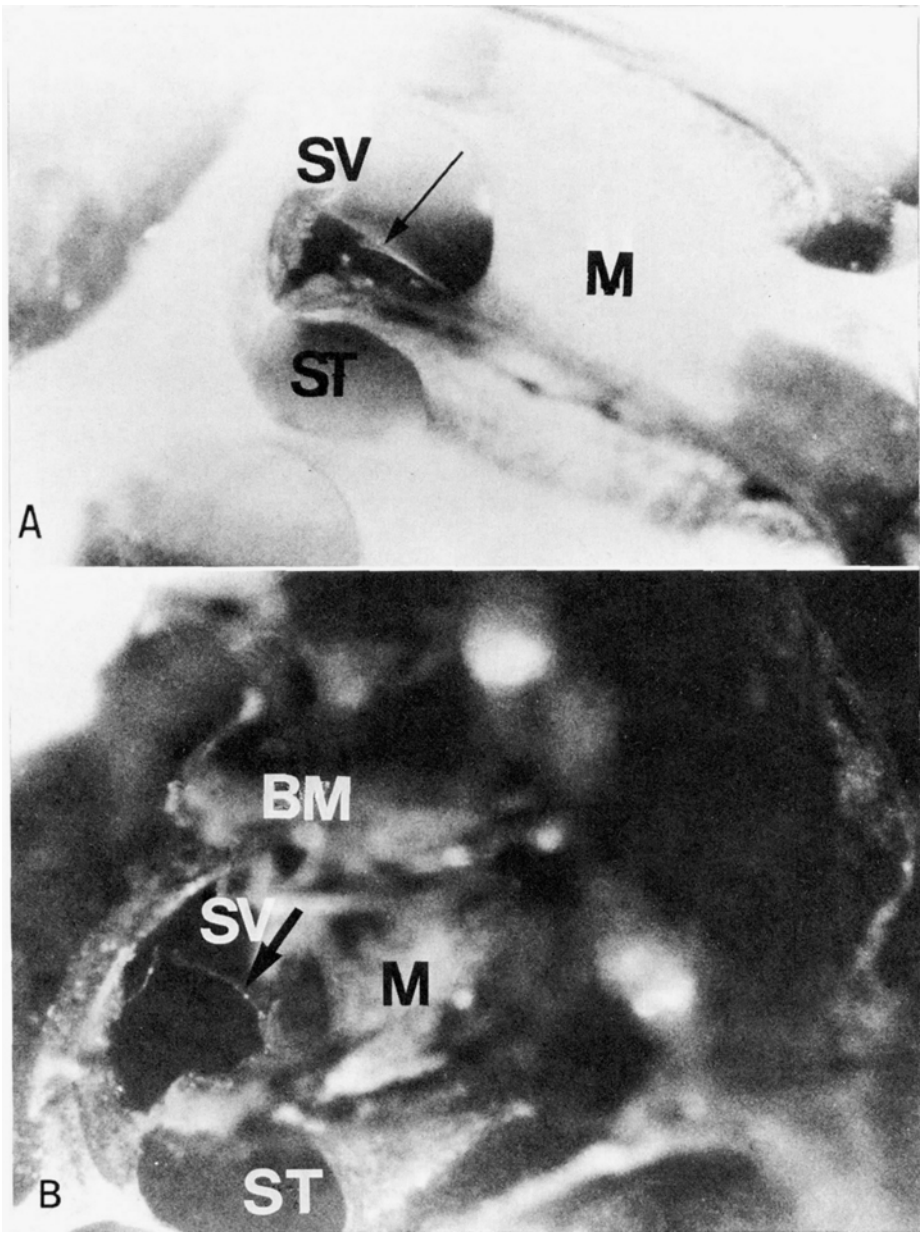

Fig. 5. Photomicrographs of both cochlea from guinea pig 4. **A**, The control cochlea shows no evidence of Reissner's membrane distention in the second cochlear turn, as is evident in **B**, the experimental PLF cochlea. *SV*, scala vestibuli; *ST*, scala tympani; *M*, modiolus; and *BM*, basilar membrane. The *arrow* in both photomicrographs identifies Reissner's membrane.

itive at 50 and 60 dB, reaching a maximum positive amplitude of nearly 16 microvolts at 60 dB. However, experimental SP is negative at all stimulus intensities and progressively increases in negative amplitude from −6 microvolts at 30 dB to −14 microvolts at 60 dB.

Means of the combined AP duration values were calculated in milliseconds for the twelve animals and are graphed in Fig. 4. Significant differences were not found at any stimulus intensity among the four conditions.

Preliminary experimental data suggest that ECoG may be helpful in differentiating (acute) PLF and (secondary) ELH. ECoG data obtained in intensity series showed an intensity effect greatest at 40 dB and no effect at 60 dB. Further studies using similar intensity series information will better determine the significance of these observations.

Microscopic examination of the cochlear duct revealed normal-appearing Reissner's membrane in all control cochlea and distention of Reissner's membrane

and distention of scala media into scala vestibuli in seven of the twelve experimental cochlear ducts of those animals with "active" PLF. Fig. 5 shows photomicrographs of both cochlea from guinea pig 4. The control cochlea *(A)* shows no evidence of Reissner's membrane distention in the second cochlear turn, as is evident in the experimental PLF cochlea *(B)*. This finding characterized seven of the twelve experimental ears. All control cochlea were normal.

DISCUSSION

Inner ear hydrodynamic fluid distortion of the membranous cochlear duct and basilar membrane is a plausible explanation for enlarged SP found either in cases of Meniere's disease and ELH or secondary hydrops caused by active PLF. The mechanism—relative excess of endolymph as a result of decreased perilymph (an "active" PLF)—would result in a real cochlear fluid/cochlear duct imbalance that would distend Reissner's membrane and distort the basilar membrane. Such a fluid expansion of the cochlear duct would necessarily shift the basilar membrane toward the scala tympani and thereby increase negative SP, as demonstrated by Durrant and Dallos, who experimentally biased the basilar membrane toward the scala tympani and thus increased negative SP.[25,27]

Inner ear hydrodynamic fluid imbalance may occur without significant sensorineural hearing loss, as is found clinically in cases of vestibular hydrops and PLF. This situation of normal hearing was reported in laboratory animals with round window rupture.[12] In such cases, ECoG would reflect basilar membrane biasing as an enlarged negative SP, even though hearing threshold (audiometric) might be unaffected—a result found for 40% of the subjects in this study.

This study shows that audiometric hearing (SRT) cannot routinely identify distortion within the cochlear duct caused by secondary hydrops following an active PLF. Therefore, normal hearing should not preclude ECoG testing for ELH and/or a PLF. Further, caloric responses do not diagnose PLF nor do they correlate with ENG fistula test results. Thus a normal ENG should not preclude ENG fistula testing.

An enhanced negative SP is an indicator of ELH that can be obtained in a noninvasive way. In this study, more than half of the surgically confirmed patients had this ECoG abnormality. ENG fistula test was also positive in more than half of the patients in this series. However, a reliable test or battery of tests, or symptom complex (singly or in combination) that consistently diagnose PLF was not found in this study.

Experimental electrophysiologic results from guinea pigs with active PLF corroborated ECoG data from patients with surgically confirmed PLF. That is, enlarged negative SP occurred only after active flow of perilymph from the cochlea with a bony fistula. Preexperimental surgical procedures, which included creating a cochlear bony fistula but without actively removing perilymph (an "inactive" PLF), did not change the normal ECoG. The control cochleas were also unaffected electrophysiologically. The differences between the experimental and nonexperimental SP/AP ratios at 30, 40, and 50 dB (Fig. 2) indicate a change in the ECoG following active PLF only. Because the experimental effect was most evident at 40 dB SL (67 dB peSPL), ECoG testing at this intensity might prove to be most diagnostically sensitive to active PLF in experimental animals. Further study is needed to confirm this conclusion.

Histopathologic data suggest that loss of perilymph (active fistula) from the closed inner ear fluid system (blocked cochlear aqueduct) within the cochlea does indeed cause secondary hydrops in more than half of the guinea pigs. However, the fact that hydrops could not be morphologically demonstrated in all animals, although the ECoG consistently changed in all animals with active PLF and none with "inactive" PLF, suggests the possibility of an alternative mechanism to alter endocochlear hydrodynamics and pressures.

The goal of this study was to identify an ECoG that is unique to PLF cases and different from ELH cases. This was not found. Rather, our data indicate that a majority of patients with active PLF are symptomatically and electrophysiologically indistinguishable from patients with Meniere's disease. The underlying common denominator for the two disorders of inner ear fluid dynamics is ELH. It is the ELH, either primary or secondary, that is detected by the enhancement of the summating potential. Since the abnormal ECoG can occur in patients with vertigo but no clinical hearing loss, it is presumed that ELH can occur in the vestibular labyrinth as well as in the cochlear duct.

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