

## Temporary Sensorineural Hearing Loss (SNHL) as a Sequel of Ventriculoperitoneal Shunt for Hydrocephalus

Mohammad Taghyan M.D, \*Mohammad Salama Bakr M.D.

Neurosurgery & \*Audiology Depart., Assiut University Hospital, Assiut, Egypt

### ABSTRACT

Hearing loss in patients who have undergone shunt placement for a hydrocephalus is perhaps an underestimated complication rather than a rare event. There appears to be a correlation between the occurrence of hearing loss and patient characteristics consistent with excessive drainage of cerebrospinal fluid (CSF) and patent cochlear aqueduct (CA)<sup>[24]</sup>. This prospective study was performed to investigate the effect of ventriculoperitoneal shunt for hydrocephalus on hearing. Twenty patients had hydrocephalus for various etiologies were the participants in this study, both sexes were included and no restriction for age, but any patient had hearing loss or middle ear disorder clinically or audiologicaly in the preoperative evaluation was excluded. Medium-pressure valve VP shunt was used. Their hearing was assessed pre- and postoperatively by using pure tone audiometry, tympanometry and acoustic reflex thresholds (ARTs) and auditory brainstem response (ABR). Hearing loss was seen in ten of the twenty patients (50%) in the immediate post-operative period, with recovery to normal hearing over one to four weeks in nine infants and partial recovery in the tenth adult one. It is suggested that the mechanism of hearing loss results directly from a decrease in pressure and/or volume of the cerebrospinal fluid via a patent shunt, which is reflected within the perilymphatic fluid as a fluid pressure reduction being transmitted via a patent cochlear aqueduct into subarachnoid space, which could results in a relative endolymphatic hypertension which could produce changes in hearing comparable to that of endolymphatic hydrops. **Conclusion:** Hearing loss in patients who have undergone shunt placement for hydrocephalus is not a rare event, but an underestimated complication. A transient hearing decrease is a known sequel after various procedures that result in the loss of cerebrospinal fluid (CSF), including shunt placement for the treatment of hydrocephalus. However, persistent hearing loss or partial recovery was documented so, It is important to identify and to diagnose any sudden hearing problem following shunt placement, because early correction of a lowered CSF pressure may prevent persistent hearing loss. Hearing loss occurred as an early complication in the immediate post operative for VP shunt placement. The mechanism of hearing loss after intracranial and spinal procedures is not fully understood. However, there appears to be a correlation between the occurrence of hearing loss and patient characteristics consistent with excessive CSF drainage and a hyperpatent cochlear aqueduct where reduction the volume of CSF, and there is a decrease in ICP, a concomitant decrease in perilymphatic fluid pressure occurs. This decrease in perilymphatic fluid, although minimal, may be sufficient to disrupt cochlear hydrodynamics and has a deleterious effect on hearing. Also, the hypertensive state of the endolymphatic pressure within the membranous labyrinth as a result of the decreased perilymph pressure affects the displacement properties of Reissner's membrane leading to cochlear dysfunction and hearing loss.

**Key words:** cerebrospinal fluid pressure, sensorineural hearing loss, cochlear aqueduct

## INTRODUCTION

A transient hearing decrease is a known sequel after various procedures that result in the loss of cerebrospinal fluid (CSF), and has been described in patients that have undergone lumbar puncture, spinal anesthesia, myelography, and also after shunt placement for the treatment of hydrocephalus<sup>[3,7,16,17,21,22,28,31]</sup>. The etiology of the hearing loss experienced after shunt placement is unclear. However, the cochlear aqueduct (CA) plays a role in the transmission of pressure differentials between the inner ear and subarachnoid space, and may contribute to hearing loss after various neurosurgical procedures, including shunt placement<sup>[2,6]</sup>.

Located within the petrous portion of the temporal bone is the inner ear which has an intricate system of fluid filled spaces that are connected to the fluid filled spaces of the central nervous system. Mechanisms exist to maintain the homeostasis of pressure between the two cochlear fluids (perilymph and endolymph) and between the cochlear fluids and the CSF<sup>[8]</sup>. Perilymphatic pressure is maintained via the cochlear aqueduct (CA) which allows direct communication between perilymph and CSF. Endolymphatic pressure is maintained by the endolymphatic sac that lies within the subdural space of the posterior cranial fossa and is surrounded by CSF. Although there is any direct communication between endolymph and CSF, the surrounding CSF pressure is easily transmitted to endolymph<sup>[26]</sup>.

## PATIENTS & METHODS

### Patients population

This prospective study was conducted in neurosurgery and audiology departments, Assiut university hospital, Assiut, Egypt over 17 months duration between October 2005 and February 2007 and included twenty patients who had hydrocephalus due to different pathologies and subjected for insertion of V-P shunts of medium pressure to investigate the effect of ventriculoperitoneal shunt for hydrocephalus on hearing. The majority of them were males (15 out of 20 =75%), and their ages ranged from one month to 50 years. Patients were excluded from participation in this study if they had hearing loss or middle ear disorders clinically or audiologically in preoperative evaluation.

### Procedures and instrumentation

The shunt operations were done by the same neurosurgeon (the author) to avoid the personal variation in operative technique taking consideration in avoidance any CSF loss during insertion of the ventricular catheter; the right side were selected as usual in all patients apart from one where the left side was used due to significant ventricular asymmetry, and the medium pressure valve shunt was used in all patients (Medtronic, P.S medical).

Patients were examined audiologically preoperative in the same operation's day, one to three days post operative and one month later as follow:

In adults and cooperative children pure tone audiometry was done for air conduction thresholds at octave frequencies between (250-8000 Hz)

and for bone conduction thresholds at octave frequencies between (500-4000 Hz) using clinical audiometer (Madsen OB 822). Masking (soundproof bodies) was used for air conduction if the difference between air conduction of tested ear and bone conduction of none tested ear 40 dB or more. Any patient had pure tone average for frequencies 500, 1000, 2000 and 4000 Hz equal or more than 25 dBHL was considered having hearing loss.

Tympanometry and acoustic reflex thresholds (ARTs) were obtained using a calibrated (Interacoustic Az 7). A220 Hz probe tone was used in tympanometry and to monitor the ARTs that was measured ipsilaterally at 1000, 2000Hz, and contralaterally at 500, 1000, 2000 and 4000 Hz.

In infants and young children who could not give reliable thresholds in behavioral audiometry, Auditory Brain stem Response (ABR) was used for threshold determination of hearing using evoked potential audiometer (Nicolit spirit). Ipsilateral stimulation by condensation click with duration 0.1msec at repetition rate 11.1 pulses/sec was utilized. Number of

sweeps was 1500 and sweep time was 10ms. The stimulus was presented at 90 dBHL and decreased by 20 dB until wave V was completely abolished then stimulus was elevated by 10dBHL. The intensity below which wave V was not repeatable was considered the hearing threshold.

## RESULTS

### Patients' characteristics

Twenty patients were included in this study, their ages ranged from one month to 50 years with an average of 10.82 years and distributed into three age groups. Infants group (one month-4 years) and included 13 patients; 10 of them under one year, children group (>5-15 years) and included 3 patients and adults group (>15-50 years) which included 4 patients. They verified into 15 (75%) males and 5 (25%) females.

All patients had hydrocephalus due to various etiologies (**table 1**) and VP shunt was inserted in the right lateral ventricle in 19 patients and in the left lateral ventricle in one patient only.

**Table 1): various etiologies of hydrocephalus**

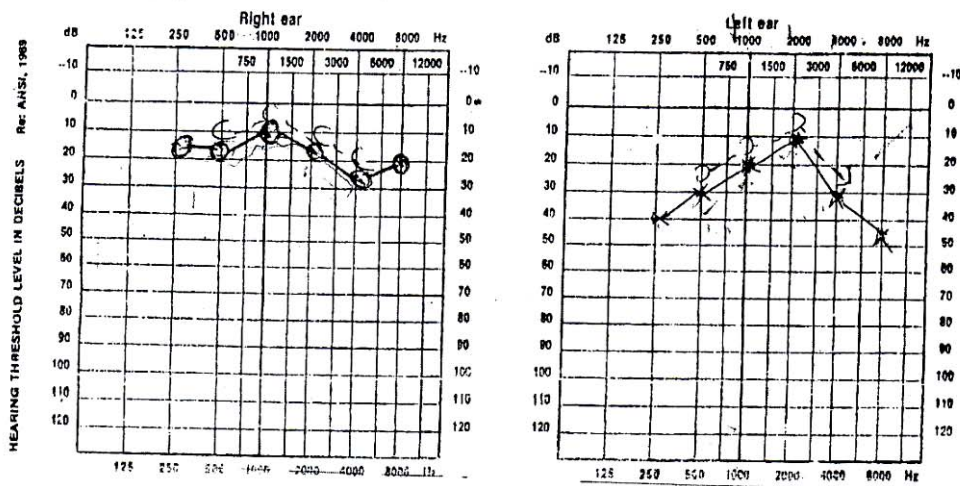
<i>Etiology of hydrocephalus</i>	<i>Number of cases</i>
Congenital	13
Late onset aqueductal stenosis	1
Posterior fossa tumour	1
Pineal body tumour	1
Craniopharyngioma	1
CPA tumour	1
3 <sup>rd</sup> ventricular tumour	1
Post traumatic	1
Total	20

**Hearing loss pattern**

Based on the pure tone average for frequencies 500, 1000, 2000 and 4000 Hz equal or more than 25 dB hearing loss and the intensity below which wave V was not repeatable in Auditory Brain stem Response (ABR); Hearing

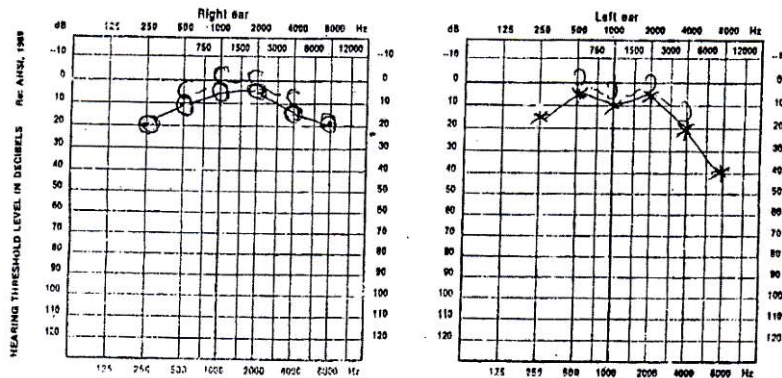
loss was seen in ten of the twenty patients (50%) in the immediate post-operative period (1-3days), with recovery to normal hearing over one to four weeks in nine infants and partial recovery in the tenth adult one. (figs.1, 2, 3)

**PURE TONE AUDIOGRAM**



**Fig. (1 a)**

♂ Patient, 46 years old . with Late-onset a queductal stenosis without audiological symptoms. (Lt) V – P Shunt was done → 2 days post operative developed Lt. tinnitus, ↓ of hearing and postural headache. → Pure tone audiogram showed → Rt. Ear within normal hearing, Lt. moderate hearing loss of 30 dB. At the average frequencies at 500, 1000, 2000, 4000 Hz.



**Fig. (1 b)**

Pat. Received medical treatment for a week → The patient got improvement as regards headach and tinnitus but little as regards hearing. 2 weeks Later marked hearing improvement and repetition of pure tone audiogram showed → Bil. within normal peripheral hearing with elevated threshold at 8 kHz in the Lt. side.

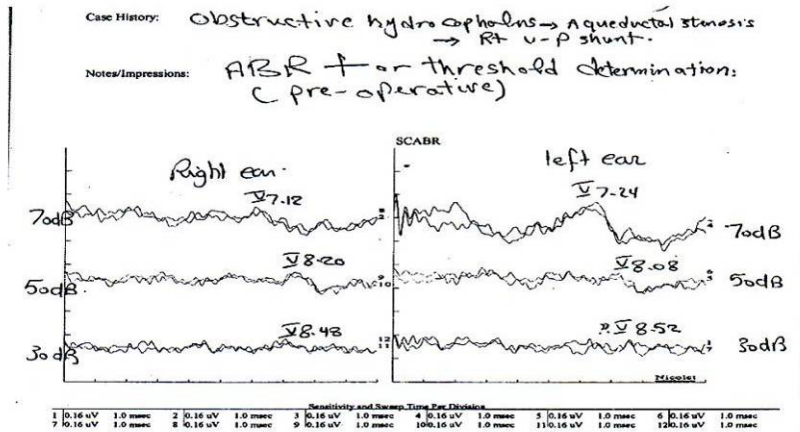


Fig. (2 a): Bilateral identifiable and repeatable ABR waves were detected down to (30 dBHL) on ipsilateral click stimulation. Conclusion Bilateral within normal hearing at least at frequency region (2 – 4 KHz).

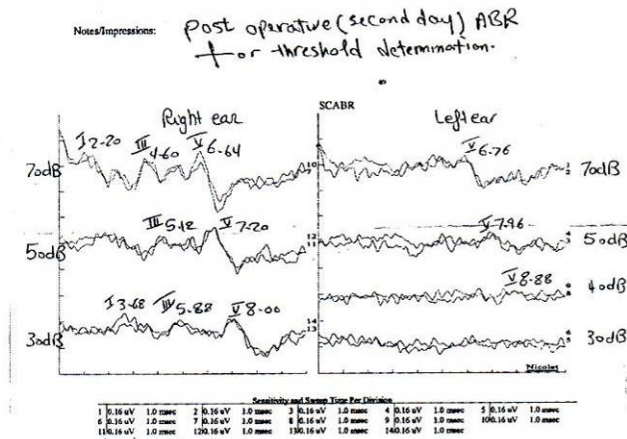


Fig. (2 b): Bilateral identifiable and repeatable ABR waves were detected down to (30 dBHL) in right ear and to (40dBHL) in left ear on ipsilateral click stimulation. Conclusion: Right within normal, left mild hearing loss at least at frequency region (2 – 4 KHz).

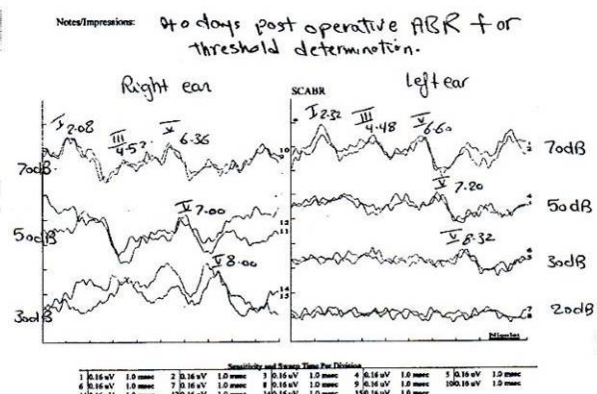


Fig. (2 c): Bilateral identifiable and repeatable ABR waves were detected down to (30 dBHL) on ipsilateral click stimulation. Conclusion: Bilateral within normal hearing at least at frequency region (2 – 4 KHz).

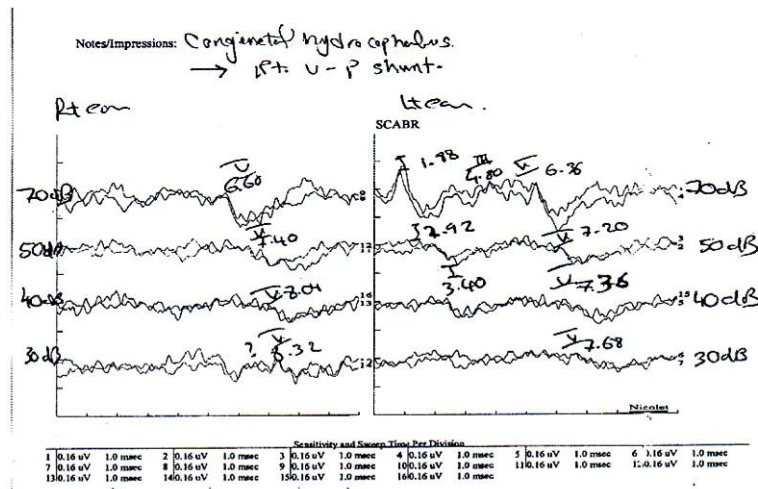


Fig. (3 a): ABR for threshold determination (Pre - operative) Bilateral identifiable and repeatable ABR waves were detected down to (30 dBHL) on ipsilateral click stimulation. Conclusion: Bilateral within normal hearing sensitivity at least at frequency region (2 - 4 KHz).

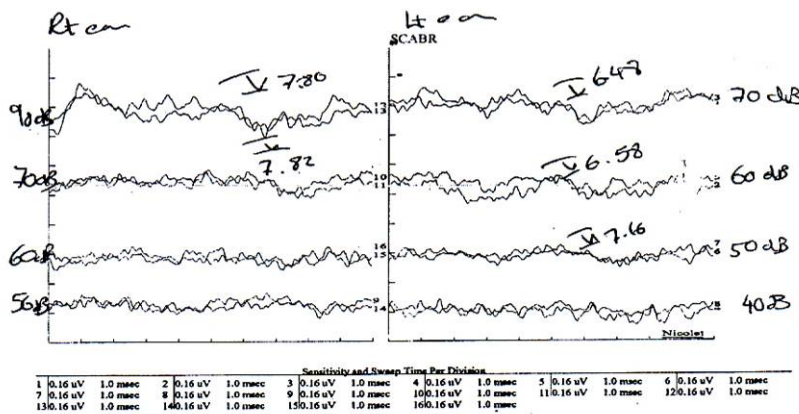


Fig. (3 b): ABR for threshold determination (A week Post - operative):- Right identifiable and repeatable ABR waves could be detected down to 70 dB. Left identifiable and repeatable ABR waves could be detected down to 50 dB on ipsilateral click stimulation. Conclusion: Right moderately severe hearing loss. Left moderate hearing loss at least at frequency region (2 - 4 KHz).

**Table2: Clinical data and hearing loss characteristics among the affected patients.**

<i>Case</i>	<i>Age</i>	<i>Sex</i>	<i>Diagnosis</i>	<i>Intervention</i>	<i>Side affected</i>	<i>Hearing loss</i>	<i>Recovery</i>
1	46 yrs.	M	Late onset aqu.stenosis	Lt. VP shunt	Left	25dB	Partial
2	2 yrs.	M	Pineal body +hydro. turs.	Rt. VP shunt	Left	30dB	Complete
3	8 mon.	F	Cong.hydro.	Rt.VP shunt	Bilateral	70dB	Complete
4	2 mon.	F	Cong.hydro.	Rt.VP shunt	Bilateral	Rt.40 dB&Lt. 30dB	Complete
5	8 mon.	M	Cong.hydro.	Rt.VP shunt	Bilateral	RT.60 dB&Lt. 40dB	Complete
6	4 mon.	M	Cong.hydro.	Rt.VP shunt	Left	30dB	Complete
7	3 mon.	M	Cong.hydro.	Rt.VP shunt	Bilateral	40dB	Complete
8	5 mon.	M	Cong.hydro.	Rt.VP shunt	Bilateral	Rt.30 dB&Lt. 50dB	Complete
9	6 mon.	M	Cong.hydro.	Rt.VP shunt	Bilateral	Rt.30 dB&Lt. 40dB	Complete
10	4 yrs.	F	Cong.hydro.	Rt.VP shunt	Bilateral	Rt.60dB& Lt.40 dB	Complete

From **table 2:** we could see hearing loss pronounced in the infantile group (9/10=90%) and was bilateral in 7 cases and unilateral in 2 cases in the contralateral ear (left ear hearing loss with Rt. VP shunt), while the tenth case was in adult patient and was ipsilateral to the shunt (left ear hearing loss with Lt.VP shunt). The severity of hearing loss was pronounced in bilateral affection and ranged between 30to 70 dB regardless the affected side, while in the unilateral affection the hearing loss was mild (30dB approximately) and predilected the left side regardless the age group.

## DISCUSSION

Although hearing loss after lumbar puncture, spinal anesthesia, and other procedures related with CSF removal

or drainage is well recognized, hearing loss after CSF shunting is rarely mentioned in the neurosurgical literature<sup>[3,11]</sup>. The etiology of hearing loss after intracranial and spinal procedures is not fully understood. However, there appears to be a correlation between the occurrence of hearing loss and patient characteristics consistent with excessive CSF drainage and a hyperpatent cochlear aqueduct (CA)<sup>[31]</sup>.

Knowledge of the anatomy and function of the CA is relevant to understanding theories about the pathogenesis of hearing decrease after CSF loss. The CA is a bony canal filled with a meshwork of loose connective tissue that connects the perilymphatic compartment of the inner ear with the subarachnoid space of the posterior cranial fossa. The canal

traverses the petrous bone from the scala tympani of the basal turn of the cochlea, close to the round window membrane, to an external, funnel-shaped aperture on the inferior surface of the petrous pyramid at the anterior division of the jugular foramen<sup>[23]</sup>. The function of the CA is not very well understood, but experimental data show that there is only small amounts of fluid are thought to move between the cochlea and the subdural space since only the membranous windows are compliant in the bony cochlear capsule<sup>[22,29]</sup>. When CSF pressure changes occur, the transmitted variations give rise to very small displacements within the cochlea as there is only a minimal pressure difference between endolymph and perilymph. However, these pressure differentials can effect displacement of Reissner's membrane and affect hearing. This link between hearing loss and low perilymphatic pressure has been confirmed using animal models. Funai et al (1988) reported threshold elevations after perilymphatic aspiration and bulging, and collapse and rupture of Reissner's membrane<sup>[9]</sup>. From anatomical studies it is known that the CA is functionally open in children<sup>[19]</sup>, but whether it stays patent with age is controversial<sup>[20,30,32]</sup>.

Walsted, et al. (1991) hypothesized that decreases in the CSF pressure are transmitted to the perilymph through a hyperpatent or enlarged CA, which results in a decrease in the perilymphatic pressure. This condition is believed to induce a relative endolymphatic over pressure and endolymphatic hydrops in a similar manner to Meniere's disease<sup>[31]</sup>. The reported risk factors for post-shunt hearing loss are as follows: inter-individual differences of patency and dimensions of the cochlear aqueduct;

the amount of CSF drainage and the pre-morbid conditions, age being an important factor. Interms of CA patency, Jackler and Hwang (1993) concluded that deficiency in the membranous mesh that fills the CA might result in increased flow even in the presence of a normal-sized aqueduct<sup>[12]</sup>. In Sun-Ho Lee et al. (2007) case report a retrospective review of the patient's temporal CT scans also revealed patent cochlear duct unilaterally only on concurrent side with hearing loss<sup>[24]</sup>.

Hypothetically, in a relative endolymphatic hydrops, the CA plays an important role because it connects the CSF space of the posterior cranial fossa with the perilymphatic compartment of the inner ear,<sup>[18]</sup> and several studies<sup>[2,5,6]</sup> have shown that the perilymphatic pressure varies with the CSF pressure. However, measurements of perilymphatic pressure after obstruction of the CA and the endolymphatic duct in cats revealed that a sudden increase of the perilymphatic pressure after elevation of the CSF pressure was lost by obstruction of the CA. For long-term fluctuations the two pressures remained highly correlated, indicating other, still unknown routes of influence of CSF pressure on perilymphatic pressure<sup>[13]</sup>. In their study in guinea pigs, Böhmer and Andrews (1989) showed that a decrease in perilymphatic pressure by rupture of the round window membrane led to an immediate concomitant decrease in endolymphatic pressure. Thus, there is very little evidence for the hypothesis that a transmission of lowered CSF pressure to the perilymphatic space via a hyperpatent CA results in a relative endolymphatic hydrops; instead there is sound experimental and anatomical evidence against it. The problem is

obviously related to CSF pressure decrease, but the exact pathophysiological mechanism remains unknown<sup>[4]</sup>.

Walstead et al (1994) in their series reported bilateral high frequency hearing losses specifically in adult patients after lumbar puncture or drainage of CSF during surgery. However, the hearing loss resolved within one week post surgery once the CSF level returned to normal. The authors hypothesized that the decrease in volume and pressure in the CSF transmitted to the perilymph by the CA resulted in bilateral endolymphatic hypertension, which produced the observed bilateral changes in hearing<sup>[31]</sup>. Also, the participants in the Lopponen et al study (1989) showed an equal number of bilateral and unilateral high-frequency hearing losses. There is not a reference as to whether the unilateral hearing losses occurred in the ear ipsilateral to shunt placement; however, the authors did not report a significant difference between the occurrence of right-ear versus the left-ear unilateral losses. They attributed the observed hearing loss to a retrocochlear dysfunction that is long-term shunting may cause excessive drainage of CSF and produce cranial base hypoplasia resulting in brain stem involvement<sup>[14]</sup>. Likewise in the present study 7/10 (70%) cases experienced bilateral hearing losses and the other 3 cases showed unilateral hearing loss. We suggest that bilaterality of hearing loss could be attributed to multiple factors; these are the ages of the patients where all of them were infants which enhance the vulnerability of their cochlear system to any decrease in volume and pressure in the CSF, bilateral functionally open of cochlear aqueducts (CA) which is well known anatomically in

children<sup>[8,19]</sup> and over CSF drainage which could be supported by improvement of chronic hearing loss (10 years after the shunt was placed) after ventriculoperitoneal shunt revision by changing the valve to a higher-pressure device in the case of Russel et al.<sup>[21]</sup>. All of the previous mentioned factors reduce the volume of CSF, and there is a decrease in ICP, a concomitant decrease in perilymphatic fluid pressure occurs. This decrease in perilymphatic fluid, although minimal, may be sufficient to disrupt cochlear hydrodynamics and has a deleterious effect on hearing. Also, we further agree Susan E. and Raymond M. 2003, who suggested that the hypertensive state of the endolymphatic pressure within the membranous labyrinth as a result of the decreased perilymph pressure affects the displacement properties of Reissner's membrane leading to cochlear dysfunction and hearing loss<sup>[25]</sup>.

Regarding the unilateral hearing loss: the three patients in the present study are similar to half cases of Lopponen et al study (1989) and all cases of Susan E. and Raymond M. study (2003), in the unilateral hearing loss, but in the present study one case had hearing loss ipsilateral to the shunt and the two others contralateral to the shunt (all the three cases had left-ear unilateral loss) in contrast to Susan E. and Raymond M cases where all of them had unilateral hearing loss ipsilateral to the shunt. In Lopponen et al study, no verification whether the hearing loss was ipsilateral or contralateral to the shunt and the authors did not report a significant difference between the occurrence of right-ear versus the left-ear unilateral losses. They attributed the observed hearing loss to a retrocochlear

dysfunction the same as bilateral effect previously mentioned<sup>[14]</sup>, but Susan E. and Raymond M attributed the unilateral low-frequency hearing loss to a patent CA combined with a patent VPshunt that provides an open communication pathway for fluid pressure variations while the unilateral high-frequency hearing loss to a retrocochlear dysfunction<sup>[25]</sup> the same as Lopponen et al. The unilateral hearing loss pattern in the present study which occurred in the immediate post operative and selected the left side could be attributed to the presence of a patent CA only on concurrent side with hearing loss as Sun-Ho Lee et al, mentioned in their case report via a postoperative high-resolution computed tomography (CT) scan of the petrous bone and was confirmed by an MRI of the same area, which ruled out further or additional pathology<sup>[24]</sup>.

Likewise the two children who failed to exhibit any hearing loss in Susan E. and Raymond M study and the unaffected ten patients in the present study may reflect a better homeostatic relationship between the cochlear fluids and CSF and/or their CA may not be patent<sup>[25]</sup>, or they are not experiencing over drainage of CSF<sup>[10,14,26]</sup>.

A transient hearing decrease is a known sequel after various procedures that result in the loss of cerebrospinal fluid (CSF), and has been described in patients that have undergone lumbar puncture, spinal anesthesia, myelography, and also after shunt placement for the treatment of hydrocephalus<sup>[3,7,16,17,21,22,28,31]</sup>. The same was happened in nine infants out of the affected our ten patients where hearing loss recovered completely within one to four weeks and confirmed audiotically. However, persistent hearing loss or partial

recovery was documented by several authors<sup>[3,15,16,22,24,27]</sup>. Likewise our tenth adult patient who recovered partially.

Hearing loss could occurred either as a delayed complication for VP shunt placement and reported by some authors like Russel et al. 2001 who reported bilateral hearing loss 4 years after ventriculoperitoneal shunting for communicating hydrocephalus<sup>[21]</sup> and Albanese et al.2007 who reported a hearing loss 6 months after shunt placement<sup>[1]</sup> or as an early complication as occurred in the present study.

In the present study, the nine infants (9/10) recovered completely within one to four weeks although they don't received specific treatment, but the tenth adult patient received oral steroids (prednisolone 30 mg/day), intravenous fluids and bed rest for 7 days and recovered partially in the same period which may suggest restoring the homeostasis of CSF and hydrodynamics of the cochlear fluids in infants better or earlier than adults. However, the correction of delayed onset hearing loss necessitated specific treatment that was replacement of the valve by one with a higher pressure and recovery was partially or not fully<sup>[1,21]</sup>.

## CONCLUSION

Hearing loss in patients who have undergone shunt placement for hydrocephalus is not a rare event, but an underestimated complication. A transient hearing decrease is a known sequel after various procedures that result in the loss of cerebrospinal fluid (CSF), including shunt placement for the treatment of hydrocephalus. However, persistent hearing loss or

partial recovery was documented so, It is important to identify and to diagnose any sudden hearing problem following shunt placement, because early correction of a lowered CSF pressure may prevent persistent hearing loss. Hearing loss occurred as an early complication in the immediate post operative for VP shunt placement. The mechanism of hearing loss after intracranial and spinal procedures is not fully understood. However, there appears to be a correlation between the occurrence of hearing loss and patient characteristics consistent with excessive CSF drainage and a hyperpatent cochlear aqueduct where reduction the volume of CSF, and there is a decrease in ICP, a concomitant decrease in perilymphatic fluid pressure occurs. This decrease in perilymphatic fluid, although minimal, may be sufficient to disrupt cochlear hydrodynamics and has a deleterious effect on hearing. Also, the hypertensive state of the endolymphatic pressure within the membranous labyrinth as a result of the decreased perilymph pressure affects the displacement properties of Reissner's membrane leading to cochlear dysfunction and hearing loss. Complete recovery without specific treatment is expected in the early onset hearing loss notably in infants. However, specific and early treatment via oral steroids, I V fluids, bed rest and/ or replacement of the valve by one with a higher pressure to avoid persistent hearing loss notably in adults and delayed onset hearing loss.

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