Assessment of hearing ability with pure-tone audiometry and otoacoustic emission methods in patients undergoing spinal anesthesia

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Aim: To investigate the frequency of hearing loss after spinal anesthesia in patients undergoing spinal anesthesia using pure-tone audiometry and otoacoustic emission methods.

Materials and methods: A total of 80 patients (40 females, 40 males, aged 18–65 years) were included in the study. For spinal anesthesia, a 27-gauge Quincke spinal needle was entered at the first attempt into the subarachnoidal space through the L4–L5 interval with the patient in the lateral decubitus position. The first day before spinal anesthesia, pure-tone audiometry, transient evoked otoacoustic emission, distortion product otoacoustic emission, and tympanometry were performed on all patients, and the same procedures were repeated on the postoperative second day.

Results: No problem was encountered related to surgery and anesthesia in any of the patients. Comparative pre- and postoperative results of pure-tone audiometry performed on the right and left ears were not significantly different in any of the patients ($P > 0.05$). Additionally, the outcomes of pre- and postoperatively performed transient evoked otoacoustic emission and of distortion product otoacoustic emission of both ears were not significantly different ($P > 0.05$).

Conclusion: According to our study, spinal anesthesia performed with a 27-gauge Quincke needle does not cause transient or permanent hearing loss.

Key words: Hearing, spinal anesthesia, spinal needle, otoacoustic emission

1. Introduction

Single-dose spinal anesthesia (SA) is one of the most frequently used, easily applicable, and effective central blockage methods with perfect tolerability. SA is performed in lower extremity, lower abdominal quadrant, and perineal regional surgeries (1–5). However, SA may have some complications, such as headache, hypotension, tinnitus, vertigo, paresthesia, nausea, vomiting, and cranial nerve lesions (3–6). Following SA, unilateral or bilateral, transient or persistent hearing loss can occur. Most hearing loss is detected at very low frequencies and recovers spontaneously (5,7,8). The side effects of the drugs used, leakage of cerebrospinal fluid (CSF) from the dura mater after SA, or an ischemic episode may cause hearing loss. It has been indicated that leakage from the dura mater could decrease intracochlear pressure, leading to hearing loss (2,12). Furthermore, various studies have demonstrated that because of effects on the amount of CSF coming from ruptured dura mater, the size and the shape

of the needle play important roles in hearing loss (5,9,10). Patient age has also been cited as an influential factor in hearing loss following SA (9–11).

After the description by Kemp in 1978, the otoacoustic emission (OAE) method has become a widely used clinical and diagnostic hearing test. OAE is a low-pitch sound created by outer cochlear hairy cells as a response to a spontaneous or acoustic stimulus. It is an objective, noninvasive, frequency-specific, and easily applicable method evaluating intracochlear activity (12,13).

In this study, we aimed to assess the presence of hearing loss (if any) in patients undergoing SA using pure tone audiometry, transient evoked otoacoustic emission (TEOAE), and distortion product otoacoustic emission (DPOAE) methods.

2. Materials and methods

Following approval (2009-12/14) of the Ethics Committee of Clinical Research of the Cumhuriyet University Faculty

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of Medicine, written and undersigned informed consent forms were obtained from all study participants. Eighty patients (40 female, 40 male) aged 18–65 years and scheduled for American Society of Anesthesiologists I–II orthopedic surgery (lower extremity surgery), urologic surgery, and general surgery (lower abdominal quadrant surgery) were included in the study. Patients with normal otoscopic examination findings, hearing thresholds lower than 25 dB in pure-tone audiometry, and type A tympanograms were enrolled. Previous ear surgery, presence of structural and mechanical occlusive problems leading to hearing loss, tympanic membrane perforation, frequent episodes of otitis media, tinnitus, inability to cooperate, hypertension, diabetes mellitus, and known allergic reaction to amide-type local anesthetics were the exclusion criteria of the study.

All patients received 2 mg of intravenous (IV) midazolam 30 min prior to surgery. Concomitant with the premedication, 10 mL/kg hydroxyethyl starch was infused for 20 min, and then infusion was maintained with isotonic solution (0.09%) at an hourly rate of 10 mL/kg. Bradycardia (≤50 beats/min) was treated with 0.5 mg atropine, and hypotension (≤30% decrease from the baseline value) was treated with 5–10 mg IV ephedrine. After access into the subarachnoidal space with a 27-gauge Quincke needle through the L4–L5 interval at the first attempt while the patient was in the lateral decubitus position, 15 mg 0.5% heavy bupivacaine was injected into the subarachnoidal space. Sensorial block level was evaluated with a pin prick test, and when sensorial block reached the T10 level, surgery was started.

The first day before spinal anesthesia, all patients underwent pure-tone audiometry (Interacoustics AC 40, Clinical Audiometer, Denmark), tympanometry (Interacoustics AZ 26, Impedance Audiometer, Denmark), and TEOAE and DPOAE (Capella Cochlear Emission Analyzer, Madsen, Denmark) tests. The same procedures were repeated on the postoperative second day. All hearing tests were performed in a noiseless room. TEOAE wave forms were retrieved as a response to a series of acoustic click stimuli for 80 µs, peaking at 80 ± 2 dB. A total of 260 sweeps at a level of >47 dB were created in a setting where effects of noise were minimized. Four successive clicks were applied, and the fourth click was delivered at a 3-fold higher amplitude than the sum of the first 3 clicks, at inverse polarity and in nonlinear mode. The presence of TEOAE was accepted when the responses were elicited at ≥3 dB above the baseline noise level. All TEOAE responses were analyzed between frequencies of 1 and 5 kHz. DPOAE was defined as the response evoked with pure-tone stimuli of 2 different frequencies ($2f_1$–f_2) applied at an f_2/f_1 ratio of 1.2. The intensities of f_1 and f_2 were determined as 65 and 55 dB, respectively. The presence of DPOAE was accepted when a $2f_1$–f_2 response was elicited at ≥3 dB above the baseline noise level. DPOAE responses were elicited and analyzed within a frequency interval of 750–8000 Hz (750, 1000, 1500, 2000, 3000, 4000, 6000, and 8000 Hz). In pure-tone audiometry, a frequency interval of 250–6000 Hz (250, 500, 1000, 2000, 4000 and 6000 Hz) was measured, and an increase of ≥10 dB above the hearing threshold detected for air and/or bone conduction pathways was evaluated as hearing loss.

3. Results

No intraoperative surgical or anesthetic problems were encountered. In none of the patients were urinary retention, headache, vertigo, or nerve damage observed. Preoperative and postoperative blood pressures were similar in all groups (P > 0.05). Intraoperative bleeding was negligible in all patients.

No significant difference was found between the pre- and postoperative pure-tone audiometry results of the right and left ears at frequencies of 250, 500, 1000, 2000, 4000, and 6000 Hz (P > 0.05) (Table).

There were no differences between the pre- and postoperative DPOAE results of both ears at frequencies at 750, 1000, 1500, 2000, 3000, 4000, 6000, and 8000 Hz (P > 0.05) (Figures 1a and 1b).

Table. The evaluation of hearing level in spinal anesthesia.

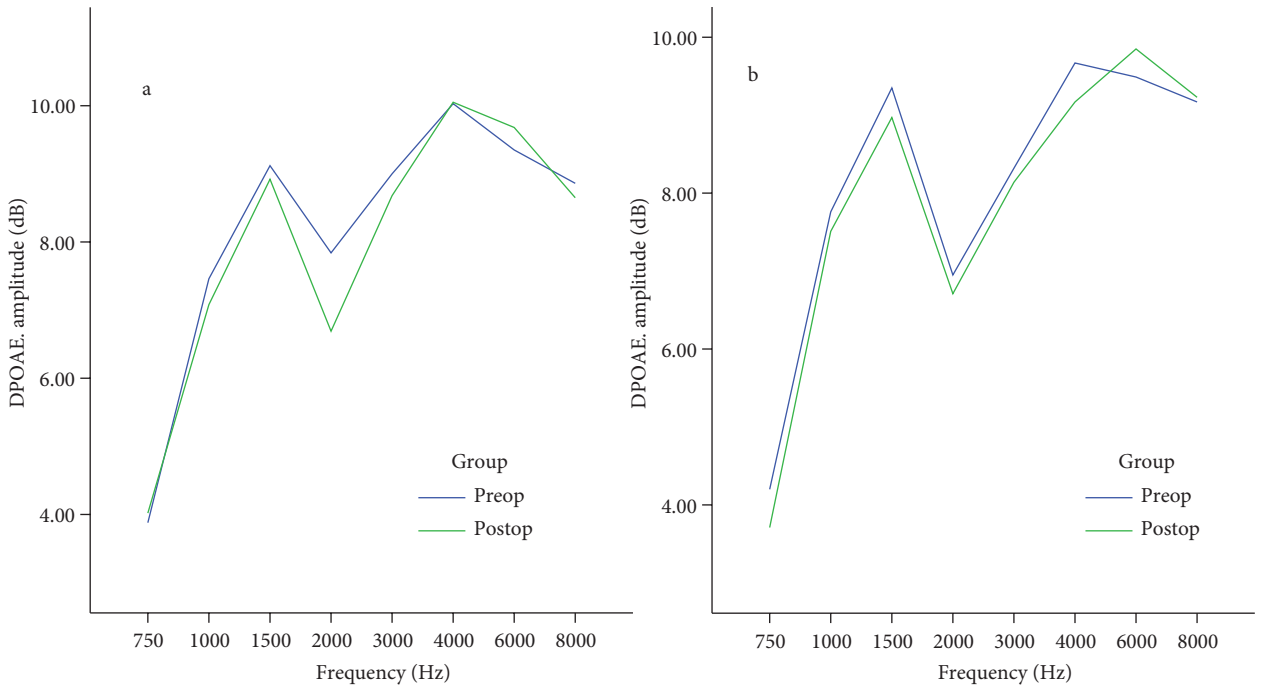| Frequency (Hz)	Preoperative		Postoperative		P-value
	R	L	R	L	
250	14.37 ± 4.16	14.81 ± 4.99	14.75 ± 4.69	15.06 ± 5.36	NS
500	11.56 ± 4.74	11.31 ± 4.19	12.81 ± 11.90	12.00 ± 4.67	NS
1000	10.93 ± 4.04	9.75 ± 4.35	11.08 ± 4.44	9.93 ± 4.53	NS
2000	8.37 ± 4.11	9.50 ± 5.43	8.68 ± 4.68	9.75 ± 5.33	NS
4000	11.68 ± 5.89	12.50 ± 5.95	11.18 ± 5.52	11.68 ± 5.89	NS
6000	12.75 ± 6.00	12.87 ± 6.45	12.43 ± 5.45	13.75 ± 5.70	NS

R: right ear. L: left ear. NS: not significant.

Figure 1. Diagram demonstrating pre- and postoperative mean DPOAE amplitudes for a) the right ears and b) the left ears.
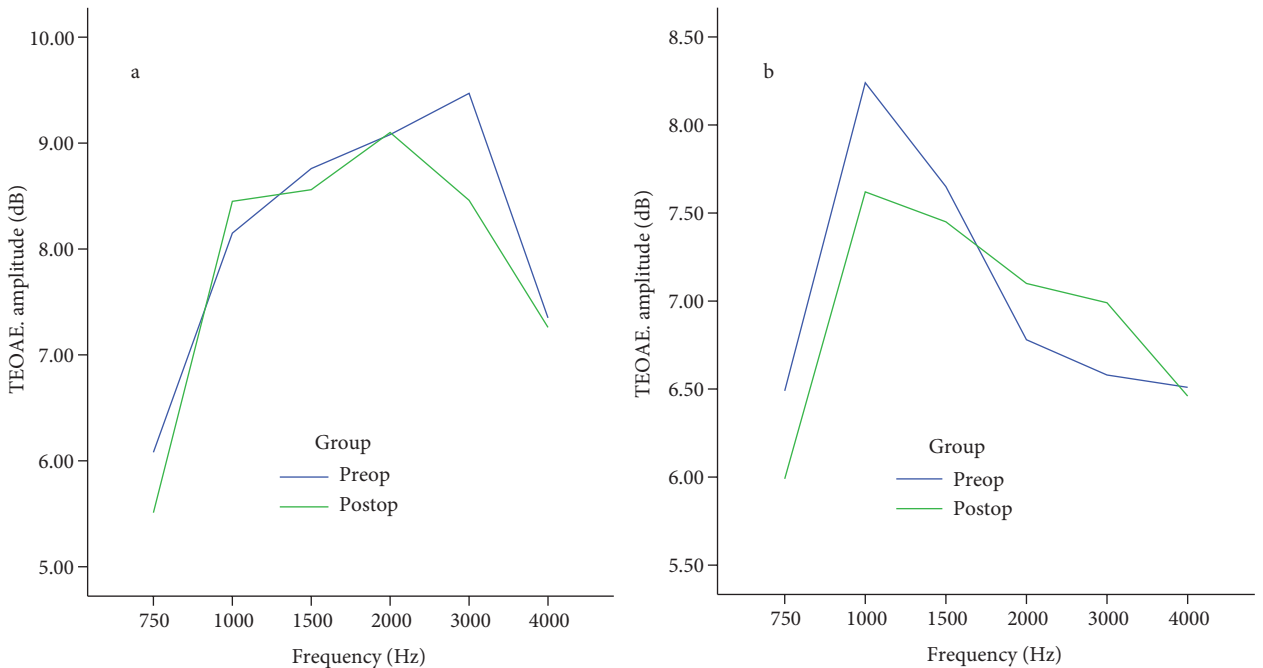

Figure 2. Diagram demonstrating pre- and postoperative mean TEOAE amplitudes for a) the right ears and b) the left ears.

The mean preoperative and postoperative TEOAE amplitudes of both ears at frequencies of 0.75, 1.25, 1.75, 2.5, 3.5, and 4.5 kHz were not different ($P > 0.05$) (Figures 2a and 2b).

4. Discussion

Although hearing loss after SA is not a prevalent complication, many case reports have nonetheless been cited in the literature (5–11). Generally, these hearing

losses are of sudden onset, developing at most 2 days after SA and resolving spontaneously without any treatment (5–11). However, some cases persist up to 2 years after SA, and permanent hearing loss has been observed (5,7,8).

Although the mechanism of hearing loss is not known in detail, the development of hearing loss as an outcome of endolymphatic hydrops has been suggested. The cochlea has a hard bony structure that allows the free movement of the intracochlear fluid only through oval and round windows. The inner ear fluid is made up of the endolymph and the perilymph, which are found in different incompressible compartments separated by membranes. Since total bone volume does not change, a decrease in fluid volume in one compartment might lead to a relative increase in the other fluid component or a positional change of the windows. The perilymph communicates with the subarachnoidal space through the cochlear aqueduct. Therefore, a decrease in CSF volume conceivably might result in a decrease in the volume of perilymph fluid and thus in transient endolymphatic hydrops, and this fluid imbalance might be normalized by replacing perilymph fluid loss (5,7,14–18). This hypothesis prompted studies to be carried out on the evaluation of hearing loss after SA. Walsted et al. (16) investigated the loss in CSF volume in hearing loss. They divided patients into 4 study groups as follows: the SA group with minimal CSF loss, the neurosurgery group with moderate CSF loss, the acoustic neurinoma surgery group with maximal CSF loss, and the control group. They found that hearing loss, deterioration in hearing frequencies and loss in hearing threshold, and CSF volume were at maximal levels in the acoustic neurinoma surgery group. They also showed that these deficiencies in hearing ability had spontaneously and completely returned to their normal levels.

Many investigations have also demonstrated the critical importance of the size and shape of the spinal needles used in spinal anesthesia in the development of hearing loss (2,5–7). Erol et al. (2) conducted investigations using 25-gauge pencil point, ball pen, and Quincke needles and reported that hearing loss was most frequently observed in the Quincke needle group, emphasizing the preference for blunt-tipped needles. In another study where 22- and 27-gauge pencil point needles were compared, the authors detected a lower incidence of hearing loss with small-sized needles, and the use of small-sized needles in SA was emphasized (5). Similarly, Malhotra et al. (7) compared 23- and 26-gauge Quincke needles and recommended the use of small-sized needles because a lower incidence of hearing loss was encountered.

Some studies have also demonstrated the potential impact of patient age on hearing loss occurring after SA (9–11,19). Ok et al. (10) and Güçlü et al. (9) detected higher incidences of hearing loss at lower frequencies after SA more often in the elderly, and they attributed this phenomenon to an increased sensitivity of the middle ear to minor alterations in cerebrospinal fluid in the elderly. Similar outcomes were reported in a study performed on females receiving cesarean sections under SA (19). Contrary to these findings, Gültekin et al. (11) asserted that hearing loss is more frequently seen in individuals of ≤30 years of age than in older patients and associated this finding with more abundant CSF leakage due to the perforation of dura mater. They stated that the incidence of postdural headache increased in direct proportion to CSF leakage, and they asserted that a decreased incidence of this type of headache in the elderly supported the outcomes of their investigation.

The impact of SA on hearing ability has also been evaluated by tests other than pure-tone audiometry (20,21). Schaffartzik et al. (21) applied a test evaluating tympanic membrane displacement in order to measure intracochlear pressure, but they found no difference between pre- and postoperative measurements. Karatas et al. (20) investigated the OAE responses of outer hair cells so as to analyze intracochlear activity in 11 patients. Comparisons of preoperative and postoperative first day values in TEOAE tests revealed decreases in mean amplitudes at frequencies of 1000, 2000, and 3000 Hz. Meanwhile, DPOAE analyses detected decrements in mean amplitudes at frequencies of 1500, 2000, and 3000 Hz.

Various studies have reported the presence or, conversely, the absence of hearing loss in patients after SA (5,7,10,19). In our study, hearing loss did not develop in any of our patients after SA. Furthermore, no significant difference was observed between mean air conduction thresholds or mean TEOAE and DPOAE amplitudes before and after SA. On the other hand, Malhotra et al. (7) and Cosar et al. (5) respectively used 26- and 27-gauge Quincke needles during SA, and none of their study patients suffered from hearing loss. In our study, we used 27-gauge needles and thus minimized CSF leakage with a resultant absence of hearing loss. Consequently, we did not find any difference between mean TEOAE and DPOAE values estimated before and after SA. Therefore, decreasing the needle size might decrease the risk of hearing loss or even reduce it to zero.

In conclusion, in the present study there were no differences between pre- and postoperative measurement in pure-tone audiometry, TEOAE, and DPOAE. In light of our study, the use of smaller-sized Quincke needles during SA is thought to be effective in preventing the occurrence of hearing loss. However, further studies are needed to substantiate our results.

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