

# Assessment of hearing via otoacoustic emission in patients that underwent spinal anesthesia

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**Abstract.** – **OBJECTIVE:** In this study, our aim is to show the differences between the preoperative and postoperative otoacoustic emissions (OAE) value in patients, who underwent spinal anesthesia.

**PATIENTS AND METHODS:** The presented study was carried out as a randomized, double-blinded, prospective study upon the approval of Ethics Committee of Medicine School, Erciyes University. The study was conducted in accordance with the Helsinki Declaration. The study involved 39 ASA I-II patients (aged 18-65 years), who underwent varicectomy operation in the Cardiovascular Surgery Department. For each of the patients, 3 OAE measurements were performed; the day before surgery, during surgery and the 1st day after surgery.

**RESULTS:** Significant differences were detected between the 2000 F2 measurements performed before, during and after the surgery ( $p<0.05$ ). The differences originated from measurements performed before and during surgery. Significant differences were detected among 3000 F1 measurements performed before, during and after surgery ( $p<0.05$ ). Hearing loss is one of the late complications of spinal anesthesia. In this study, we observed the differences between the preoperative and postoperative OAE values. The incidence of hearing loss detectable with auditory measurements has been reported to vary between 10 and 50%. Of these, 25% is clinically relevant or recognizable. However, it is considered to be a subjective test, because it is influenced from mental, motor and psychological status of the patient. But the management in hearing loss following spinal anesthesia is still controversial. Hearing loss is generally spontaneously resolved within a few days. However, there are case reports of hearing loss lasting for months.

**CONCLUSIONS:** In this study, we observed differences between some preoperative and postoperative OAE values.

## Key Words

Spinal anesthesia, Otoacoustic emission, Hearing loss.

## Introduction

Spinal anesthesia has still unknown aspects and complications in terms of anatomy, physiology and pharmacology<sup>1</sup>. Loss of hearing is one of the rare but important complications of spinal anesthesia. As a result of a puncture in the dural membrane after spinal anesthesia and leakage of cerebrospinal liquid (CSL), bilateral loss of hearing may be rarely seen<sup>2</sup> at low frequencies. The real reason in loss of hearing is the decreased CSL pressure, perilymphatic hypotonia and endolymphatic hydrops<sup>3</sup>.

Otoacoustic emissions (OAE) are the sounds originating from cochlea's hairy cells, and can be recorded via a sensitive device to be placed in external auditory canal<sup>4</sup>. This test is a fast, objective and easy method for revealing the function of the cochlea. Emission measurements are very sensitive and also useful for determining the loss of hearing at even dysfunction phase<sup>5</sup>.

In this study, our aim is to reveal the differences between the preoperative and postoperative OAE value in patients, who underwent spinal anesthesia.

## Patients and Methods

This study was planned as a prospective clinical trial upon the approval of EPK of Kayseri Training and Research Hospital. The presented study was conducted as a randomized, double-blinded, and prospective study upon approval of Ethics Committee of Erciyes University's Medicine School. The study was conducted following the Helsinki Declaration. The study involved 39 ASA I-II patients (aged 18-65 years), who underwent varicectomy in cardiovascular surgery department (Table I). Patients having previous ear surgery and/or hearing loss history were excluded.

**Table I.** Demographic characteristics of patients.

| Gender      | Mean age (year) | Mean body weight (kg) | Mean height (cm) | Mean duration of operation (min) |
|-------------|-----------------|-----------------------|------------------|----------------------------------|
| Female (25) | 38              | 65                    | 158              | 45                               |
| Male (14)   | 44              | 72                    | 174              | 45                               |

**Table II.** 2000 F2 measurements.

|                 | N  | Mean  | Std. deviation | Minimum | Maximum | p value |
|-----------------|----|-------|----------------|---------|---------|---------|
| 2000F2 Preop.   | 39 | 54.50 | 1.01           | 50.50   | 55.90   | 0.047*  |
| 2000F2 Intraop. | 39 | 54.10 | 1.09           | 52.20   | 56.10   |         |
| 2000F2 Postop.  | 39 | 54.32 | 0.97           | 52.20   | 56.10   |         |
| Male (14)       | 44 | 72    | 174            | 45      |         |         |

The patients were randomly divided into groups by using the sealed envelope method. Patients declining regional anesthesia, having bleeding diathesis, severe hypotension and/or increased intracranial pressure were also excluded. The patients were then transferred to the operating room by an anesthesiologist blinded to the group of patient. Standard monitoring via systolic and diastolic blood pressure, heart rate and peripheral oxygen saturation measurements were performed for all of the patients. In patients with sufficient cardiac reserve, the pre hydration was achieved via 10 ml/kg normal saline injection and spinal anesthesia was performed by using 25-gauge Quincke spinal needle (Braunmedical, Melsungen, Germany) at L3-4 or L4-5 level in the patient in sitting position. Heavy bupivacaine (0.5%) was used in spinal anesthesia. Sensorial blockade was assessed by using the pin prick test along mid-clavicular line bilaterally, where as the motor blockade was assessed by using modified Bromage scale. Duration between intrathecal anesthetic administration and achieving Bromage score 2 or 3 was considered as the time to onset of motor blockade (Bromage score 2: the patient can move feet but not knee; Bromage score 3: the patient can move neither feet nor knee). Surgery was started when the blockade reached at T10 level. Bradycardia was defined as the heart rate <50 bpm, and treated by using 0.5 mg atropine. If systolic blood pressure was <90 mmHg or decreased by 20% in proportion to baseline, it was treated with the ephedrine. Need for intraoperative analgesic and intraoperative or postoperative nausea, vom-

iting and other adverse effects were recorded in all patients. The patient was transferred to post-operative recovery unit (PACU) at the end of surgery and then discharged to ward when Bromage score was 0. All of the patients were examined by an ETN specialist before auditory evaluations. In each of the patients, 3 otoacoustic emissions (OAE) measurements were performed: the day before surgery, during surgery and the 1<sup>st</sup> day after surgery. OAE measurements were performed by using MADSEN Capella Cochlear Emission Analyzer for Windows. Correlation value and signal/noise ratio were also examined. The presence of emission was defined as the signal/noise ratio >3 dB for 3 times or more.

### Statistical Analysis

Dependent group *t*-test was used in order to assess the OAE measurements. *p*-value <0.05 was considered as statistically significant.

### Results

In this study, the preoperative, intraoperative and postoperative OAE measurements were performed in every patient. Significant differences were found between the 2000 F2 measurements performed before, during and after the surgery ( $p < 0.05$ ). The differences originated from measurements performed before and during surgery (Table II). Significant differences were detected between the 3000 F1 measurements performed

before, during and after surgery ( $p<0.05$ ). These differences were between the measurements performed before, during and after surgery (Table III). No significant difference was detected in other frequencies evaluated.

## Discussion

Hearing loss is one of the late complications of spinal anesthesia. In this study, we observed the differences between the preoperative and postoperative OAE values. To date, hearing loss after spinal anesthesia has been investigated via PTO (Pure Tone Audiometry). This test involves pressing a button when patient heard sound stimulus in a silent cabinet. However, it is considered to be a subjective test, because it is affected by mental, motor and psychological status of the patient<sup>5</sup>. OAE measurement is an objective test indicating the functions of hairy cells.

Transient OAE is the primary measurement. Loss of transient OAE at postoperative period, which was present at preoperative period, indicates the hearing loss of  $\geq 30$  dB<sup>6</sup>. In literature, there is a limited number of studies on hearing loss following spinal anesthesia, and majority of these publications are case reports<sup>7-13</sup>.

The occurrence of hearing loss detectable with auditory measurements has been reported to vary between 10 and 50%. Of these, 25% is clinically relevant or recognizable. Finegold et al<sup>14</sup> haven't observed any hearing loss following the spinal and epidural anesthesia; rather, they have observed significant increase in hearing. This is the only publication, in which the authors proposed that there was no hearing loss following the spinal anesthesia. Improvement in hearing has been explained with better concentration in PTO test after surgery.

Following the spinal anesthesia, endolymph decreases through cochlear aqueduct<sup>15</sup>. Relative endolymphatic hydrops affects the entire basilar membrane, particularly the cochlear apex. This

theory is based on the anatomical structures. In the present study, the role of the cochlea in hearing loss following the spinal anesthesia was objectively demonstrated for the first time. In healthy individuals, this could be tolerated without causing any problem, and decreased CSF can be replaced within one week. However, in case of aqueduct obstruction or Ménière disease, the restoration is delayed and hearing loss may develop. Since there is no feasible, direct, non-invasive and ethical method of visualizing the aqueduct anatomy in human, this factor couldn't be eliminated at preoperative period<sup>16</sup>.

Lamberg et al<sup>17</sup> have found the occurrence of hearing loss to be 37% after the continuous spinal anesthesia and 43% after the single-dose spinal anesthesia. Authors have found that the recovery times for hearing were 3 and 1.4 days, respectively. Lamberg et al<sup>17</sup> have suggested that the edema at dura around the catheter prevents the CSF leakage, which is due to smaller catheter diameter when compared to Tuohy needle. Both of systemic hypotension and cochlear ischemia can also play role in hearing loss. Cochlear ischemia is of significant importance since the damage is irreversible due to the insufficient collateral flow<sup>18</sup>.

Lee et al<sup>19</sup> have reported that the hearing loss developed in 1 out of 6 patients, in whom arterial blood pressure decreased by  $\geq 44$  in proportion to baseline, in the way corroborating the hypothesis that cochlear ischemia leads hearing loss.

Kılıçkan et al<sup>20</sup> have evaluated effects of combined spinal epidural anesthesia on hearing loss and found no significant difference between combined spinal epidural (by using 25 G Whitacre needle) and spinal anesthesia group. The fact that fluids given via epidural route failed in preventing the hearing loss suggests that CSF loss isn't the only factor involved in hearing loss. Hearing loss is also associated with certain number of unintentional dural puncture. In a study<sup>21</sup>, it has

**Table III.** 3000 F1 measurements.

|                    | N  | Mean  | Std. deviation | Minimum | Maximum | p value |
|--------------------|----|-------|----------------|---------|---------|---------|
| 3000F1<br>Preop.   | 39 | 65.26 | 0.70           | 63.20   | 66.60   | 0.005*  |
| 3000F1<br>Intraop. | 39 | 64.88 | 0.76           | 62.90   | 66.10   |         |
| 3000F1<br>Postop.  | 39 | 64.68 | 0.57           | 63.50   | 66.00   |         |

been shown that 71 attempts are needed for 90% success during anesthesia training. In our study, all spinal blockades were performed by same experienced anesthesiologist, and the patient was excluded in puncture when failed in the first attempt.

The management in hearing loss following spinal anesthesia is controversial. Hearing loss is spontaneously resolved within a few days in general<sup>18</sup>. However, there are case reports suggesting hearing loss over months<sup>19,22,23</sup>.

Some authors advocated that there is no need for treatment<sup>24</sup>, while others recommended not waiting for spontaneous recovery<sup>25</sup>. Management options include epidural blood patch<sup>19,22</sup>, vasodilator agents<sup>19</sup> and steroids<sup>23</sup>.

In case of marked improvement in hearing loss with epidural blood patch, high level of suspicion should be considered about the etiology of hearing loss, especially if auditory measurement is unavailable.

Another important factor is age in hearing loss. Gültekin et al<sup>8</sup> found occurrence of hearing loss to be 52% in younger adults and 16% in elder individuals. Authors attributed this finding to the greater extent of CSF loss in younger individuals. In our study population, patients were at 4<sup>th</sup> decade mostly and subclinical hearing loss was found in 20% of patients.

In this study, the preoperative, intraoperative and postoperative otoacoustic emission measurements were performed. Significant differences were detected among 2000 F2 measurements performed before, during and after the surgery ( $p<0.05$ ). Significant differences were also detected among 3000 F1 measurements performed before, during and after the surgery ( $p<0.05$ ). The differences in only 2 frequencies can be explained by heterogeneous study population and limited sample size. Further studies with larger sample size are needed in this context.

## Conclusions

Hearing loss is one of the late complications of spinal anesthesia. There are a lot of studies about spinal anesthesia and hearing loss. In this study, we observed the differences between some preoperative and postoperative OAE values.

## Conflict of Interests

The Authors declare that they have no conflict of interests.

## References

- 1) LIU SS, McDONALD SB. Current issues in spinal anesthesia. *Anesthesiology* 2001; 94: 888-906.
- 2) SPRUNG J, BOURKE DL, CONTRERAS MG, WARNER ME, FINDLAY J. Perioperative hearing impairment. *Anesthesiology* 2003; 98: 241-257.
- 3) WALSTED A, SALOMON G, OLSEN KS. Low frequency hearing loss after spinal anesthesia. Perilymphatic hypotonia? *Scand Audiol* 1991; 20: 211-215.
- 4) KEMP DT. Otoacoustic emissions, their origin in cochlear function and use. *British Medical Bulletin* 2002; 63: 223-241.
- 5) AKYILDIZ N. İ. FIZYOLOJİSİ. Kulak hastalıkları ve mikrocerrahisi-I (1. baskı). Bilimsel Tıp Yayınevi, Ankara, 1998; pp. 77-92.
- 6) KEMP DT, RYAN S, BRAY P. A guide to the effective use of otoacoustic emissions. *Ear Hear* 1990; 11: 93-105.
- 7) WANG LP. Sudden bilateral hearing loss after spinal anesthesia (A case report). *Acta Anaesthesiol Scand* 1986; 30: 412-413.
- 8) GÜLTEKİN S, ÖZCAN Ş. Does hearing loss after spinal anesthesia differ between young and elderly patients? *Anesth Analg* 2002; 94: 1318-1320.
- 9) LEE CM, PEACHMAN FA. Unilateral hearing loss after spinal anesthesia treated with epidural blood patch. *Anesth Analg* 1986; 65: 312.
- 10) MICHEL O. Hearing loss as a sequel of lumbar puncture. *Ann Otol Rhinol Laryngol* 1992; 101: 390-394.
- 11) SUNDBERG A, WANG LP, FOG J. Influence on hearing of 22 G Whitacre and 22 G Quincke. *Anesthesia* 1992; 47: 981-983.
- 12) GÜLCÜ N, MADENOĞLU H, DOĞRU K, KETENCİ İ, UĞUR F, BOYACI A. Spinal Anestezi Sonrası İşitme Kaybının İki Ayrı İşitme Testi İle Değerlendirilmesi. *Van Tıp Dergisi*: 2006; 13: 121-125.
- 13) KARATAS E, GÖKSU S, DURUCU C, İSİK Y, KANLIKAMA M. Evaluation of hearing loss after spinal anesthesia with otoacoustic emissions. *Eur Arch Otorhinolaryngol* 2006; 263: 705-710.
- 14) FINEGOLD H, MANDELL G, VALLEJO M, RAMANATHAN S. Does spinal anesthesia cause hearing loss in the obstetric population? *Anesth Analg* 2002; 95: 198-203.
- 15) WEMAMA JP, DELECROIX M, NYARWARA JB, HORBER RK. Permanent unilateral vestibulocochlear dysfunction after spinal anesthesia. *Anesth Analg* 1996; 82: 406-408.
- 16) SCHAFFARTZIK W, HIRSCH J, FRICKMANN F, KULY P, ERNST A. Hearing loss after spinal and general anesthesia: A comparative study. *Anesth Analg* 2000; 91: 1466-1472.
- 17) LAMBERG T, PITKANEN MT, MARTTILA T, ROSENBERG PH. Hearing loss after continuous or single shot spinal anesthesia. *Reg Anesth* 1997; 22: 539-542.
- 18) SPRUNG J, BOURKE DL, CONTRERAS MG, WARNER ME, FINDLAY J. Perioperative hearing impairment. *Anesthesiology* 2003; 98: 241-257.

- 19) LEE MC. Hearing loss after spinal anesthesia. *Anesth Analg* 1990; 71: 561.
- 20) KILICKAN L, GÜRKAN Y, AYDIN O, ETILER N. The effect of combined spinal-epidural (CSE) anaesthesia and size of spinal needle on postoperative hearing loss after elective caesarean section. *Clin Otolaryngol* 2003; 28: 267-272.
- 21) KONRAD C, SCHÜPHER G, WIETLISBACH M, GERBER H. Learning manual skills in anesthesiology: is there a recommended number of cases for anesthetic procedures? *Anesth Analg* 1998; 86: 635-639.
- 22) WALSTED A, SALOMON G, OLSEN KS. Low frequency hearing loss after spinal anesthesia. Perilymphatic hypotonia? *Scand Audiol* 1991; 20: 211-215.
- 23) KILIÇKAN L, GÜRKAN Y, OZKARAKAS H. Permanent sensorineural hearing loss following spinal anesthesia. *Acta Anesthesiol Scand* 2002; 46: 1155-1157.
- 24) PANNING B, PIEPENBROCK S. Hearing loss after spinal anesthesia. *Anesth Analg* 1987; 66: 800.
- 25) MICHEL O. Hearing loss as a sequel of lumbar puncture *Ann Otol Rhinol Laryngol* 1992; 101: 390-394.