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Cochlear Function Monitoring After Spinal Anesthesia

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Study Design A
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Statistical Analysis C
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Literature Search F
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ABCDEF 1 **Agata Janecka-Placek**
ABCDEF 2 **Grażyna Lisowska**
ABCDEF 3 **Andrzej Paradysz**
ABCDEF 1 **Hanna Misiótek**

1 Department of Anesthesiology and Intensive Therapy in Zabrze, Medical University of Silesia, Zabrze, Poland
2 Department of Otolaryngology in Zabrze, Medical University of Silesia, Zabrze, Poland
3 Department of Urology in Zabrze, Medical University of Silesia, Zabrze, Poland

Corresponding Author: Grażyna Lisowska, e-mail: grazyna.lisowska@onet.pl
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Background: The aim of the study was to examine the effect of spinal anesthesia on the function of cochlear outer hair cells (OHCs), determined by means of objective distortion product otoacoustic emissions (DPOAE) testing. To the best of our knowledge, our study was the second OAE-based analysis of cochlear function during spinal anesthesia, and the only experiment including such a large group of patients.





Material/Methods: The study included 20 patients (18 men and 2 women) subjected to a scheduled ureterorenoscopic lithotripsy with routine spinal anesthesia with 10 mg (2 ml) of 0.5% hyperbaric bupivacaine and 50 µg (1 ml) of fentanyl. The levels of DPOAEs and background noise at 1000–6000 Hz were recorded prior to and immediately after the anesthesia, and on the postoperative day 2.

Results: We did not find significant differences between DPOAEs values recorded prior to and immediately after the anesthesia. The only exception pertained to 5652 Hz, at which a significantly higher level of DPOAEs was observed immediately after the anesthesia. The levels of DPOAEs at 2002 Hz and 2380 Hz collected on the postoperative day 2 were significantly higher than the respective baseline values. Irrespective of the frequency and time of testing, we did not find any significant differences between the recorded levels of background noise.

Conclusions: Our findings point to the lack of a detrimental effect of spinal anesthesia on objectively evaluated cochlear function, and thus suggest that this method is safe, even for OHCs, which are extremely susceptible to exogenous and endogenous injuries.

MeSH Keywords: **Otoacoustic Emissions, Spontaneous • Urologic Surgical Procedures**

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Background

Spinal anesthesia is a frequently used anesthetic technique [1]. Whereas postoperative urinary retention and post-dural-puncture headache (PDPH) represent the most often reported complications of spinal anesthesia [1], the post-anesthesia loss or transient deficit of hearing are of markedly rarer evidence [2–13]. However, contrary to usually transient hearing loss, which was reported in individual cases only [3,4,14], the deficit of hearing, also transient, is observed more often. The incidence of hearing deficits in patients undergoing spinal anesthesia can be estimated at 0.2–0.8% based on patient-reported symptoms [3–5]. However, studies using pure tone audiometry in small groups of patients showed that the prevalence of hearing deficits after spinal anesthesia can range from 0% up to 27%. Deficits of hearing in patients after spinal anesthesia are defined as a transient sensorineural hearing loss, mostly at low frequencies, which resolves spontaneously within up to 3 weeks [2–9,15,16].

Functional impairment of cochlear outer hair cells (OHCs) is postulated as a primary mechanism of transient hearing loss related to spinal anesthesia. The impairment results from a decrease in the perilymph pressure in the cochlea, occurring secondarily to a drop-off in cerebrospinal fluid resulting from spinal puncture [6,10,11,13,15,16]. The cochlear duct forms an anatomical connection between these 2 fluid spaces, allowing even distribution of their hydrostatic pressures.

Otoacoustic emissions (OAEs) are sounds that arise in the ear canal when the tympanic membrane receives vibrations transmitted backward through the middle ear from the cochlea [17]. OAE tests constitute an excellent, non-invasive and objective, instrument for determining cochlear activity [18]. They can detect very early changes, which are still imperceptible for a patient, and thus allow detection of even slight alterations of cochlear micromechanics [18,19]. OHCs constitute the most sensitive cochlear structure, being most prone to injuries caused by exogenous factors, such as decreased oxygen saturation of the blood, ototoxic drugs, toxins, noise, and toxic metabolites [18,20,21]. OAE is an objective, accurate, frequency-specific, easy and rapid test, which can be used to detect both transient and permanent alterations [18].

As most previous studies of spinal anesthesia-related hearing loss were based on a subjective hearing test, namely pure tone audiometry (PTA), the pathomechanism of this complication is still not completely understood. The principal aim of this study was to examine the effect of spinal anesthesia of the function of OHC determined by means of distortion product otoacoustic emissions (DPOAE) testing. To the best of our knowledge, apart from the study by Karatas et al. [4], this was the first experiment employing both subjective (PTA) and

objective (tympanometry and DPOAE) tests to assess the organ of Corti prior to and after spinal anesthesia.

Material and Methods

Ethical approval for this prospective study was provided by the Local Bioethical Committee at the Medical University of Silesia in Katowice (Chairperson Prof. Stefan Kossmann) on September 21st, 2010 (decision no. KNW/0022/KB1/99/10).

Initially, we enrolled 36 consecutive patients (28 men and 8 women) qualified to a scheduled ureterorenoscopic lithotripsy. The age of the patients ranged between 18 and 45 years; all of them represented American Society of Anesthesiologists (ASA) physical status I, and gave their written informed consent to participate in the study. Exclusion criteria from the study were:

- present or past history of ear diseases,
- congenital deafness,
- exposure to noise,
- metabolic disorders,
- injuries of the head with resultant loss of consciousness,
- concomitant central nervous system disorders, meningitis,
- administration of ototoxic agents,
- contraindications to general anesthesia (severe spinal deformations, history of injuries or surgeries on the spine, purulent lesions or tattoos on the skin at a site of the puncture, use of anticoagulants, anti-aggregants, or fibrinolytic agents).

Normal tympanic membrane in otoscopy, normal results of tympanometry (tympanometric peak pressure between +25 daPa and –160 daPa, and maximum compliance of 0.3–1.4 ml), pure-tone auditory threshold of less than 30 dB HL, documented at 250–6000 Hz constituted inclusion criteria to the study.

The tympanometry was conducted with Minitymp Interacoustics AS DK-5610 impedance bridge, and tympanometric curves were determined for each ear separately.

The pure-tone audiometry was performed with Maico ST 20 audiometer, with bilateral determination of auditory threshold expressed as dB HL, at 250–6000 Hz for air-conduction and at 250–4000 Hz for bone-conduction audiometry. Both tests were conducted in the course of preoperative qualification of patients, in a soundproof room at the Department of Urology.

Eventually, a group of 20 patients (18 men and 2 women) was identified based on the above mentioned inclusion and exclusion criteria, and qualified for further analysis.

Subjects' preparation

The standardized protocol of anesthesia and audiological examination was applied to all the patients. The subjects were not premedicated.

Audiological testing

DPOAEs were collected in a quiet prep room of the Surgical Department, immediately before surgery. The otoacoustic emissions from the ear with the better result of pure-tone audiometry were recorded, with a patient placed in a supine position. The DPOAE tests were performed using an ILO 2.92 Otodynamics Analyser system, with non-linear distortion products evoked by 2 tonal signals of different frequencies, f_1 and f_2 . A 2-channel probe with 2 loudspeakers and one microphone was inserted within the external ear canal, and fit with a soft adapter to provide precise adaptation to the canal wall. Prior to the test, the software automatically checked the resonance of the external ear canal and the probe sealing. The data were collected using a DP-gram paradigm, i.e. during a step change in f_1 and f_2 frequencies, at a constant f_2/f_1 frequency ratio, and with constant levels of primary tones, L1 and L2. Pairs of primary tones ($L_1=L_2=70$ dB SPL) were delivered via the probe at a frequency ratio $f_2/f_1=1.22$. The non-linear distortions $f_3=2f_1 - f_2$ were recorded at various f_2 frequencies (1001, 1184, 1416, 1685, 2002, 2380, 2832, 3369, 4004, 4761, 5652, and 6299 Hz). Only the recordings with the signal to noise ratio of at least 3 dB were analyzed. Either the DPOAE response across all studied frequencies, or the levels of background noise, recorded under various experimental conditions, was subjected to analysis. Following the baseline DPOAE testing, each patient was transferred to the surgical theater, placed on an operating table and subjected to the endoscopic urological procedure under spinal anesthesia.

Immediately after the procedure, each patient was transferred to the prep room, and DPOAEs were collected from the same ear as before, with a subject placed in a supine position. The emissions were recorded no later than 7 minutes after the surgery (mean 5.2 ± 1.8 min). The last (third) DPAOE testing and the second PTA took place on postoperative day 2, in a quiet room at the Department of Urology; again the same ear was examined, with the patient in a supine position.

Technique used for anesthesia

Following the baseline DPOAE testing, each patient was transferred to the surgical theater, and placed on an operating table. An 18-gauge (G) intravenous cannula (Venflon, Becton-Dickinson, Sweden) was inserted into the antebrachial vein, and an intravenous drip infusion of 500 ml 0.9% NaCl (Natrium Chloratum 0.9%, Fresenius Kabi, Poland) was started at 2 ml/kg

b.w./h, along with continuous monitoring of the electrographic curve, heart rate, and pulse oximetry. Moreover, a non-invasive measurement of the baseline arterial blood pressure (T_0) was taken. Subsequently, spinal anesthesia was delivered: a 27-G (0.4×90 mm) pencil-point Whitacre spinal needle (Becton-Dickinson, Spain) was inserted into L3/L4 intervertebral space through a 22-G (0.7×32 mm) introducer from the midline approach, with an opening oriented cranially. Upon obtaining a leakage of the cerebrospinal fluid, 10 mg (2 ml) of 0.5% hyperbaric bupivacaine (Marcaine Spinal 0.5% Heavy, AstraZeneca AB, Sweden) were administered, along with 50 µg (1 ml) of fentanyl (Fentanyl, Warszawskie Zakłady Farmaceutyczne Polfa S.A., Poland) at about 1 ml/4–5 s. Subsequently, a patient was placed in a supine position, and another measurement of arterial blood pressure (T_1) was taken.

During the surgery, oxygen was delivered via a nasal cannula, at a 6 l/min flow rate. The hemodynamic parameters of each patient, i.e. heart rate, systolic, diastolic and mean blood pressure, and pulse oximetry were recorded after 5, 10, 15, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, and 90 minutes of anesthesia, excepting the individuals who left the surgical theater earlier.

Any drop off in arterial blood pressure (at least 20% decrease from the baseline value) was compensated with intravenous ephedrine (Ephedrinum hydrochloricum, Polfa Warszawa, Poland) in 5-mg fractions, given every 3 minutes until normalization. Intravenous atropine (Atropinum sulfuricum, Polfa Warszawa, Poland) was administered in each case of decreased heart rate (at least 20% decrease from the baseline level), at 0.01 mg/kg b.w. until the cumulative dose of 3 mg was reached.

Statistical analysis

The results were subjected to statistical analysis with Statistica 10.0 package (StatSoft, USA). Normal distribution of the analyzed variables was verified with the Shapiro-Wilk test. Due to non-normal distribution of most study parameters, non-parametric tests were used for intra- and intergroup comparisons. The significance of differences between pairs of dependent variables was verified with the Wilcoxon's test, and the Mann-Whitney U test was used for intergroup comparisons of independent variables. The relationships between pairs of variables were tested with Spearman's coefficients of rank correlation. The threshold of statistical significance was set at $P<0.05$ for all the tests.

Results

The basic characteristics of the study 20 participants (age, sex, body height and weight, ASA physical status, prevalence of

Table 1. Basic characteristics of the study participants (n=20).

Parameter	Mean ±SD/n (%)
Age (years)	27.2±6.6
Men	18 (90%)
Women	2 (10%)
Body height (cm)	177±7.6
Body weight (kg)	79.6±15.9
ASA I	20 (100%)
Tobacco smoking	6 (30%)
Duration of the surgery (min)	37.5±15.0

Table 2. Hemodynamic parameters of the studied patients (arithmetic means of consecutive values recorded during the anesthesia).

Parameter	Mean ±SD
Systolic blood pressure (mm Hg)	131.3±14.9
Diastolic blood pressure (mm Hg)	71.8±11.5
Mean blood pressure (mm Hg)	95.5±11.6
Heart rate (beats/min)	70.4±10.2
Saturation (%)	99.1±0.8

Table 3. Results of pure tone audiometry: PTA-1 (mean threshold for 250 Hz, 500 Hz, 1000 Hz), PTA-2 (mean threshold for 1 kHz, 2 kHz, 4 kHz), and PTA-3 (mean threshold for 1 kHz, 2 kHz, 4 kHz, 6 kHz)*.

Group	PTA-1 (dB HL)	PTA-2 (dB HL)	PTA-3 (dB HL)
Subjects 18–45 years	7.3±3.9	6.3±4.6	7.05±5.1

* According to the methodologically accepted qualification criteria, all subjects had average hearing threshold level across the octave frequencies from 0.25 to 4.0 kHz (less than 30 dB HL).

cigarette smoking, and duration of the surgery) are presented in Table 1. All patients underwent a single spinal puncture. None of the participants showed any adverse events, such as pain, dizziness, nerve injuries, hypotension, bradycardia, or decreased arterial oxygen saturation during or after the anesthesia (Table 2).

The results of the first PTA are presented in Table 3. We did not observe a significant effect of spinal anesthesia on the results of PTA conducted on postoperative day 2.

The effects of spinal anesthesia on the level of DPOAEs

Comparative analysis of DPOAEs at 1000–6000 Hz did not reveal significant differences between values recorded prior to and immediately after the anesthesia. The only exception pertained to f2=5652 Hz, for which a significantly higher level of DPOAEs was observed immediately after anesthesia (P=0.007; Figure 1). Moreover, the post-anesthesia levels of DPOAEs at frequencies higher than 2000 Hz turned out to be higher than the respective baseline levels, but the differences did not prove statistically significant. To exclude the effect of noise on the results of DPOAE testing, we compared the levels of background noise recorded prior to and after the anesthesia (Figure 1). As no significant differences between the pre- and post-anesthesia levels of background noise were documented at any of the studied frequencies, one can exclude the confounding effect of noise on the DPOAE level.

The levels of otoacoustic emissions collected on postoperative day 2 were higher than at the baseline, for nearly all analyzed frequencies (except f2=1001 Hz and f2=1184 Hz), with statistically significant differences observed at f2=2002 Hz (P=0.016) and f2 = 2380 Hz (P=0.026; Figure 2). Irrespective of the frequency, we did not document any significant differences between the levels of background noise recorded prior to anesthesia and on the 2nd day post-surgery (P>0.05; Figure 2).

Discussion

The inner ear is composed of a number of interconnected spaces that form a membranous and bone labyrinth. The bone labyrinth consists of the vestibule, cochlea, and three semi-circular canals, and is filled with a fluid, referred to as perilymph. In turn, the membranous labyrinth contains endolymph. Perilymph, being a filtrate of blood and cerebrospinal fluid, is a substrate for the inner ear cells. Both passive diffusion and active ion transport occur between endolymph and perilymph. Cerebrospinal fluid dynamics is vital for the normal functioning of the inner ear. An imbalance between endolymph and perilymph probably underlies the hearing loss observed after spinal anesthesia, resulting from a drop-off in cerebrospinal fluid pressure. In most persons, the exchange of fluid between endolymphatic and perilymphatic space is low and well-balanced. According to Michel et al. [15,16], the mechanism of transient hearing loss after spinal anesthesia

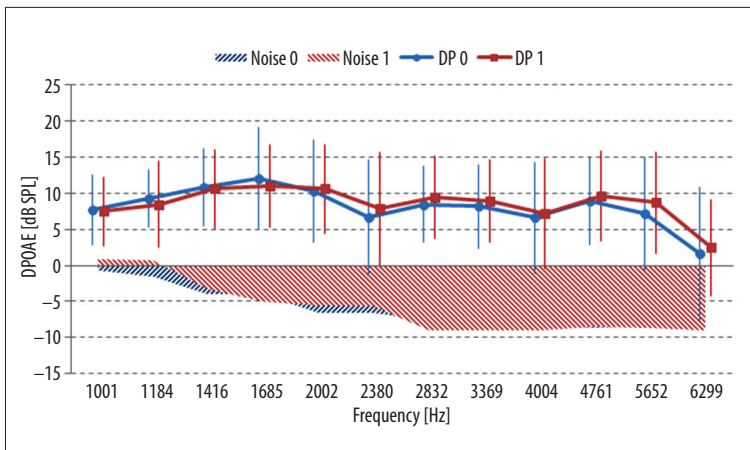


Figure 1. Levels of DPOAEs and background noise documented prior to (recording 0, circles) and immediately after the anesthesia (recording 1, squares). Significant differences at * $p \leq 0.05$, ** $p \leq 0.01$, and *** $p \leq 0.001$.

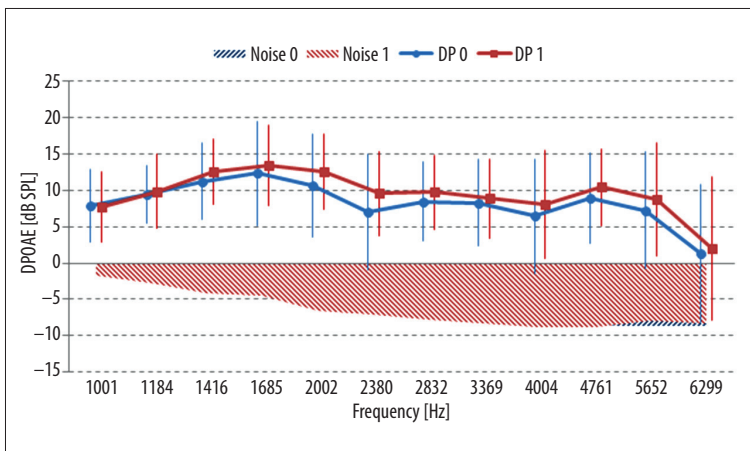


Figure 2. Levels of DPOAEs and background noise documented prior to (recording 0, circles) and on the postoperative day 2 (recording 2, squares). Significant differences at * $p \leq 0.05$, ** $p \leq 0.01$, and *** $p \leq 0.001$.

can be associated with an anatomical anomaly of the cochlear aqueduct, leading to enhanced flow of cerebrospinal fluid through the latter. This anomaly is observed in approximately 2 per 1000 patients [15,16]. Due to a decrease in cerebrospinal fluid pressure and the presence of post-puncture opening in the dura mater, larger volumes of perilymph get into cerebrospinal space, which is reflected by a decreased perilymphatic pressure in the labyrinth. This leads to an increase in the endolymphatic pressure and development of endolymphatic hydrops, similar to that observed in the Ménière's disease [22]. The presence of hydrops causes a translocation of OHCs settled on the basilar membrane, which results in low-frequency hearing loss [13,15,16]. Nevertheless, some authors point to potential ischemic etiology of the loss of hearing or suggest that it is associated with microembolism or infection [3].

Hearing loss, observed after spinal anesthesia may be induced by a number of factors. According to literature, the volume of cerebrospinal leakage, and resultant incidence of hearing loss and other concomitant morbidities, especially post-dural-puncture headache, are determined by the type and diameter of the spinal needle. Hearing loss was reported more frequently following a puncture with a 22- or 23-G needle, than

after using a 25- or 26-G needle [9,10], and was not observed in patients who received anesthesia with a 27-G needle [3]. Lamberg et al. [8] compared the risk of hearing loss due to single-shot spinal puncture and continuous spinal anesthesia, with spinal catheter left in the cerebrospinal space for 24 hours. The incidence of hearing impairment after continuous anesthesia turned out to be higher (43%) than after the single shot (37%) [8]. Erol et al. [6] observed that the incidence of hearing loss among patients in whom a 25-G Quincke spinal needle was used for spinal anesthesia was higher, than in those who received spinal anesthesia through a 25-G pencil-point or ball-pen needles. The same authors did not document significant differences in the incidence of hearing loss among patients subjected to anesthesia with 25-G pencil-point and ball-pen spinal needles [6]. We used a 27-G pencil-point spinal needle in order to minimize the leakage of the cerebrospinal fluid through the post-puncture opening in the dura mater. All procedures were performed by the same anesthesiologist. None of our patients required repeated puncture of the cerebrospinal space, cerebrospinal fluid withdrawal, or barbotage.

The degree of circulating blood loss and administration of infusion solutions also seem to be associated with the incidence

of low-frequency hearing loss [2,5,12]. Acute changes in the blood volume, intracranial pressure, and osmolality of the plasma were postulated to be the risk factors of hypoacusia. Proper and adequate fluid therapy can prevent loss of hearing as it improves perfusion of the inner ear [2,5,12]. Yildiz et al. [5] analyzed the effect osmolality of administered infusion solutions on the organ of hearing. They observed that the incidence of hearing loss in patients who received lactated Ringer's was higher than in those administered gelatin polysuccinate (Gelofusine), but the difference turned out to be insignificant [5]. Schaffartzik et al. [12] documented hearing loss after both spinal and general anesthesia. Furthermore, low-frequency hearing loss seemed to correlate with intraoperative volume replacement. In this study, 500 ml of crystalloids were used for the initial intravenous volume replacement, followed by a colloid infusion at an individually adjusted dose [12]. Our patients underwent endoscopic surgery for urolithiasis. The endoscopic procedure is not associated with the loss of blood and does not require irrigation of the bladder with distilled water. All our patients received infusions of physiological saline during the 2 initial postoperative days, and the surgeries lasted no longer than 50 minutes. None of the subjects required administration of any additional medications, and no cases of hypotonia or decreased arterial blood oxygen saturation were documented.

The influence of age on the incidence of post-spinal anesthesia hearing loss still raises controversies. We did not observe any negative effects of spinal anesthesia on hearing in younger patients (i.e., between 18 and 42 years of age). Nevertheless, our participants showed higher levels of DPOAEs, both immediately after the anesthesia and on postoperative day 2. However, this finding seems clinically unrelated, as none of the subjects reported subjective improvement of hearing. Due to limitations of otoacoustic emission testing (physiological loss of response with age), this method cannot be used in older patients. The results, similar to our findings, were documented in 1 previous study of women subjected to cesarean section, who received spinal anesthesia through a 24-G pencil-point needle or a 25-G Quincke needle. Furthermore, this group did not present with negative effects of spinal anesthesia on hearing, and a slight, albeit significant, transient improvement of auditory threshold was documented on PTA. However, none of the patients reported subjective improvement of hearing [23]. Similar transient improvement of PTA hearing threshold was also reported by Ok et al. [24], who used pure tone audiometry and tympanometry to examine 20- to 40-year-old patients receiving anesthesia through a 22-G or 25-G spinal needles. In contrast, Gultekin and Ozcan [7] observed higher incidence of hearing loss in younger patients (i.e., below 30 years of age). They explained this phenomenon as a consequence of a more severe leak of the cerebrospinal fluid after dural puncture in the young patients, which was also associated with higher

incidence of post-dural-puncture headache in this group. The same authors highlighted the potential confounding effect of preexisting hypoacusia on the incidence of post-spinal anesthesia hearing loss in older subjects (>60 years of age) [7]. In order to neutralize this potential confounder, we analyzed only the patients with PTA evidence of normal auditory threshold at 250–6000 kHz. Consequently, our results seem to be more objective than those documented in previous studies.

The abovementioned phenomenon of transient improvement of auditory parameters immediately after spinal anesthesia, documented on both PTA [23,24] and during otoacoustic emission testing (present study), is an intriguing and still unexplained finding. The presence of enhanced amplitude of otoacoustic emissions after spinal anesthesia is difficult to explain. It may be associated with the influence of medications used for spinal anesthesia (bupivacaine and fentanyl) on the function of the efferent system (ie, the medial olivocochlear (MOC) system). The principal role of the MOC system is reduction (inhibition) of the cochlear enhancement, which is reflected by a decrease in the basilar membrane motility and the resultant slight decrease in the otoacoustic emission level [17,25,26]. Efferent inhibition of OHC is mediated by nicotinic acetylcholine receptors (nAChR) that form channels for calcium ions, thus allowing the activation of calcium-dependent potassium channels [27]. Blockade or decreased responsiveness of these receptors lead to withdrawal of the inhibitory effect of the MOC system and resultant transient increase in the otoacoustic emission level. The influence of bupivacaine and fentanyl on the cholinergic receptors of the MOC system is still not understood; therefore, it cannot be excluded that they impair neurotransmission in the MOC system, which causes blockade or decrease in the efferent inhibition of the cochlea and resultant transient increase in OAE amplitude.

Available data on the objectively evaluated spinal anesthesia-induced changes taking place in the organ of hearing are sparse. Most of the published research was based on subjective hearing tests ie, pure tone audiometry). We evaluated the organ of hearing both by means of otoacoustic emission testing and PTA. OAE testing is an objective method and its results are tightly correlated with the degree of cochlear injury. Only Karatas et al. [4] used a similar method in order to determine the objective effect of spinal anesthesia on the organ of hearing. These authors performed spinal puncture with a 25-G Quincke spinal needle. They used passive oxygen therapy with a nasal cannula at 3 l/min flow rate and intravenous sedation with Midazolam. The study revealed a transient negative effect of spinal anesthesia on cochlear function (i.e., a decrease in the otoemission level), with progressive return to its pre-anesthesia values within 48 hours post-surgery [4]. In our patients, oxygen was delivered at a higher flow rate (approximately 6 l/min); furthermore, they received the anesthesia via a smaller needle and were not premedicated.

Conclusions

Our findings point to the lack of a detrimental effect of spinal anesthesia on objectively evaluated cochlear function. Consequently, this method of anesthesia seems to be safe in the case of short bloodless endoscopic urological procedures,

even in the context of OHCs, which are extremely susceptible to exogenous and endogenous injuries.

Conflicts of interest

No conflicts of interest.

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