Effects of Different Local Anaesthetics on Oto-acoustic Emission Tests after Spinal Anaesthesia

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ABSTRACT

Purpose: *To evaluate whether there is any effect of different local anaesthetic agents on hearing loss after spinal anaesthesia.*

Methods: *Thirty American Society of Anesthesiologists (ASA) I patients without a hearing problem were included in the study. Transient evoked oto-acoustic emissions (TEOAE) were examined. Patients were randomly separated into two groups: Group L, 15 mg isobaric levobupivacaine application and Group B, 15 mg isobaric bupivacaine application, for spinal anaesthesia. Oto-acoustic emission measurements were repeated on patients at the 24th hour after spinal anaesthesia application.*

Results: *No significant difference was found in the output values of the right and left ears according to the input values in group L at no kHz (*p *> 0.05). No significant difference was found in the output values of the right and left ears according to the input values in any kHz of Group B (*p *> 0.05).*

Conclusion: *Fifteen milligrammes of isobaric bupivacaine and levobupivacaine had no significant difference on the effect of hearing loss after spinal anaesthesia. Hearing loss after spinal anaesthesia has many unknown issues, and deserves to be explored by performing many studies.*

Keywords: Bupivacaine, levobupivacaine, spinal anaesthesia, transient evoked oto-acoustic emission

Efectos de diferentes anestésicos locales en las pruebas de emisiones oto-acústicas después de la anestesia raquídea

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RESUMEN

Propósito: *Evaluar si hay efectos de diferentes agentes anestésicos locales sobre la pérdida de la audición después de la anestesia raquídea o espinal.*

Métodos: *Treinta pacientes de la Sociedad Americana de Anestesiólogos (ASA) I sin problemas de audición fueron incluidos en el estudio. Se examinaron las emisiones otoacústicas evocadas transitorias (EOAET). Los pacientes fueron separados aleatoriamente en dos grupos: Grupo L con una aplicación de 15 mg de levobupivacaína isobárica, y grupo B con una aplicación de 15 mg de bupivacaína isobárica, para la anestesia raquídea. Las mediciones de las emisiones otoacústicas se repitieron a los pacientes en las 24 horas después de la aplicación de la anestesia raquídea.*

Resultados: *No se encontró ninguna diferencia significativa en los valores de salida de los oídos derechos e izquierdos de acuerdo con los valores de entrada en el grupo L en ningún kHz (p > 0.05). No se encontró ninguna diferencia significativa en los valores de salida de los oídos derechos e izquierdos de acuerdo con los valores de entrada en otros kHz del grupo B (*p *> 0.05).*

Conclusión: *Los 15 mg de bupivacaína isobárica y de levobupivacaína no tuvieron ninguna diferencia significativa en el efecto de la pérdida de la audición después de la anestesia raquídea spinal. La pérdida de la audición después de anestesia raquídea tiene muchas cuestiones desconocidas y merece ser explorada mediante la realización de numerosos estudios.*

Palabras claves: bupivacaína, levobupivacaína, anestesia raquídea, emisiones oto-acústicas evocadas transitorias

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INTRODUCTION

One of the complications that may arise after spinal anaesthesia application is hearing loss, and studies related to the subject are quite limited. One of the most important reasons for this situation is the non-existence of objective tests used in the past to evaluate hearing, and many studies requiring patient cooperation. This may raise some questions in terms of the reliability of existing studies (1).

Oto-acoustic emission tests enabling us to evaluate hearing ability objectively have been developed and their usage has gradually become prevalent. Oto-acoustic emission is based on the principle that the receiver perceives and records the response given by cochlear cells with cilia for sounds at various wavelengths sent by a sound transmitter-receiver placed into the external ear canal. Unlike pure sound audiometry, it is an objective, sensitive method which can notice hearing loss even in the dysfunctional phase (2). This complication is only mentioned with few notes in basic books on the subject, and it may be deemed negligible due to the fact that it occurs at very low wavelengths which cannot be noticed by patients and it is generally reversible. The aim of this study is to assess whether levobupivacaine or bupivacaine has any effect on loss of hearing after spinal anaesthesia.

SUBJECTS AND METHODS

After getting approval from the hospital and the local ethics board, we recruited 30 American Society of Anesthesiologists (ASA) I patients for urological surgery under spinal anaes-thesia, aged between 20–45 years and who had no hearing problem.

Measurements of transient evoked oto-acoustic emissions (TEOAE) were done by Otodynamics ILO 288 on 'Quickscreen' mode. Transient behaviours were averaged 260 times. The stimulus was given at 80 (± 3) dB pkSPL. The amplitudes and repeatability of emissions obtained from 1, 1.4, 2.0, 2.8 and 4.0 frequency bands in TEOAE measurements were examined. Existence criteria of TEOAEs were determined as being over 70% of repeatability, with emission amplitude over 3 dB in at least three frequencies. Patients who had amplitude below 3 dB in at least three frequency bands were deemed unable to pass the pre-operative oto-acoustic emission test and were excluded from the study. The analysis of TEOAE results is based on the signal-to-noise ratio values that indicate the difference between the oto-acoustic emission response and the noise level at a particular frequency.

Patients were informed about the study and separated randomly into two groups, namely levobupivacaine (Group L) and bupivacaine (Group B). Before operation, patients were taken to an audiology laboratory for oto-acoustic emission measurements. Intravenous access was obtained by 22 g branula in the operating room; standard monitoring and fluid replacement was done by giving 0.9% saline solution at 4 mL/kg/hour.

Spinal anaesthesia was performed by the same anaesthetist, in a sitting position, through the L3–L4 juncture using the midline approach, either with 15 mg levobupivacaine or isobaric bupivacaine with a 25 g Quincke spinal needle. Surgery was allowed to begin after it was understood that sensory block was provided at T12–L1 level. Twenty-four hours after spinal anaesthesia application, patients were taken to the audiology laboratory again and their TOAE measurements were performed.

Statistical Package for Social Sciences (SPSS) for Windows 10.0 programme was used for statistical analyses. Student's *t*-test was used for intergroup comparisons between parameters showing normal distribution in comparing quantitative data, as well as definitive statistical methods (average, standard deviation). Mann-Whitney U test was used for intergroup comparisons of parameters not displaying normal distribution, and for the determination of groups causing the difference. Results were evaluated with a 95% confidence interval and significance was about $p < 0.05$.

RESULTS

In the evaluation of hearing measurement by oto-acoustic emission, no statistically significant difference was found in the postoperative values of the right and left ears in Group L at any kHz according to the pre-operative values ($p > 0.05$) (Table 1). Statistically, no significant difference was found in the postoperative values of the right and left ears at any kHz in Group B according to the pre-operative values ($p > 0.05$, Table 2).

DISCUSSION

Though hearing loss after spinal anaesthesia is seen especially at low frequencies, this has not been reported as a prevalent complication (3–5). The reason may be that the situation is not noticed by patients, or patients may fail to report their complaints about it (6). Hearing loss after spinal anaesthesia generally occurs at low frequencies and is recovered between three days and seven months (7).

According to the theory, instead of cerebrospinal fluid leak-

Table 1: Transient evoked oto-acoustic emissions values for Group L

Group L	Pre-operative		Postoperative		
	Mean	SD	Mean	SD	*p*
Left					
1.0 kHZ	4.52	8.44	-1.39	8.42	0.074
1.4 kHZ	6.46	8.24	5.52	9.08	0.910
2.0 kHZ	7.10	6.06	8.21	9.38	0.427
2.8 kHZ	7.57	4.56	9.49	5.39	0.244
4.0 kHZ	7.43	7.32	8.71	5.90	0.443
Right					
1.0 kHZ	1.08	9.07	-1.36	10.38	0.307
1.4 kHZ	7.48	9.25	8.47	5.17	0.532
2.0 kHZ	5.72	6.69	7.84	5.60	0.307
2.8 kHZ	6.77	6.10	7.33	6.70	0.650
4.0 kHZ	5.99	7.00	7.33	7.50	0.470

Table 2: Transient evoked oto-acoustic emissions for Group B

Group L	Preo-perative		Postoperative		
	Mean	SD	Mean	SD	*p*
Left					
1.0 kHZ	3.33	7.37	-0.11	7.40	0.124
1.4 kHZ	9.03	6.37	9.09	7.19	0.865
2.0 kHZ	9.05	7.44	8.55	8.61	0.460
2.8 kHZ	9.73	9.25	8.84	6.33	0.268
4.0 kHZ	9.47	10.30	7.71	10.67	0.426
Right					
1.0 kHZ	1.39	8.80	-0.68	7.68	0.334
1.4 kHZ	7.26	8.64	9.16	5.58	0.426
2.0 kHZ	6.23	5.56	8.01	8.77	0.478
2.8 kHZ	6.09	10.16	10.15	7.38	0.245
4.0 kHZ	5.54	11.80	10.51	5.92	0.041*

ing into the epidural cavity from the subdural area due to the hole made by the spinal needle in the dura, perilymph, which is the fluid of the inner ear, passes through the subarachnoid area over the cochlear canal. As a result, the pressure of perilymph within the inner ear decreases. Thus, the pressure loss arising there from the situation (perilymphatic hypotension) and relative endolymphatic pressure developing secondarily (hydrops) may be responsible for hearing loss (8, 9).

In a study by Öncel *et al* comparing the effects of spinal and epidural anaesthesia on postoperative hearing loss, a direct connection was established between hearing loss and dural puncture, in that the hearing loss occurred after neuroaxial block, thus supporting this theory. While hearing loss was not seen in the epidural group, it was seen in the spinal group, according to the pre-operative levels (10).

There is a correlation between cerebrospinal fluid loss after spinal anaesthesia and hearing loss observed at low frequency (11). Walsted *et al* observed a parallelism between increased cerebrospinal fluid loss developed after spinal anaesthesia, neurosurgery and acoustic neurinoma resection and patients with hearing loss after these procedures (12). This situation may also be explained by the perilymphatic hypotension theory, as developing secondarily to cerebrospinal fluid loss.

Also, Sundberg *et al* thought that the shape of the spinal needle tip affected the amount of cerebrospinal fluid leakage (13). We kept these factors constant in our study by using 25 g and a Quincke-tipped spinal anaesthesia needle in both groups.

Venous ponding due to adrenergic tonus loss as a side effect of anaesthesia, decreasing perioperative central venous pressure, bleeding and late intra-operative volume replacement may cause a temporary decrease in intracranial pressure and, accordingly, the disruption of hearing. Some studies have reported a significant correlation between intra-operative volume replacement and hearing loss at low frequency (14, 15).

Hearing loss after spinal anaesthesia generally disappears by itself. Nevertheless, permanent hearing loss has also been reported. According to the literature, treatment options can be listed as epidural blood patch, vasodilator drugs and steroids (16, 17).

There are cases of hearing loss after spinal anaesthesia in the literature which are sudden, symptomatic, and may even be permanent. Thus, clinicians should become aware of this complication of spinal anaesthesia and should inform their patients about the possibility of this complication, as part of their medico-legal responsibilities.

According to our study, isobaric bupivacaine and levobupivacaine had no significant difference on the effect of hearing loss after spinal anaesthesia. To determine the reliability of these two agents in terms of this complication, they should be compared with other local anaesthetic agents. Hearing loss after spinal anaesthesia, because it has many unknown issues, deserves to be explained more widely by performing many studies.

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