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The Assessment of Recruitment in Rhinoscleroma Patients with Hearing Loss after Streptomycin Therapy

Ocena wyrównania głośności u chorych na twardziel z niedosłuchem po leczeniu streptomycyną

Оценка выравнивания громкости у больных склерозом с нарушением слуха после лечения стрептомицином

Streptomycin (STM) has been still used in the treatment of several chronic inflammatory diseases, especially tuberculosis and rhinoscleroma. It is administered for quite a long period, so the total dosis may reach 200 g or more. In our previous studies the incidence and the degree of STM induced hearing loss in rhinoscleroma patients was determined (9). The aim of these studies was to assess the site of lesion in the hearing organ of these patients.

MATERIAL AND METHODS

The studies were carried out in 19 rhinoscleroma patients that were given 47—240 g (average 150 g) of STM according to Durska-Zakrzewska method (1). The patients have been followed up since then. The age of the patients ranged from 32 to 66 years, with an average of 52 years. Except one ear with tympanosclerosis that was excluded from the studies, all the patients had normal tympanic membranes on appearance and normal pure tone threshold before treatment, no history of ear diseases, noise exposure and metabolic disorders.

Threshold audiometry, impedance audiometry, short increment sensitivity index (SISI), Carhart's tone decay test and fixed frequency Békésy tracing were performed in all the patients. Stapedial reflex, SISI test and tone decay test were administered at 0.5, 1, 2 and 4 kHz, whereas Békésy tracings were carried out at 2 and 4 kHz.

Routine audiometric testing and Carhart's test were performed using an AUG--69 audiometer. Impedance audiometry was performed using a Madsen ZO-70 electroacoustic bridge, with the acoustic stimuli provided by a Kamplex DA-11 audiometer. The same audiometer was used for SISI test. The Békésy recording by air conduction was performed using a Peters AP-6 audiometer with an accessory. The stimuli were pulsed at 2 pulses per second and an attenuation rate of 2.5 dB/sec. All the testing was performed in a sound-treated, isolated test room.

RESULTS

When the age dependent hearing loss determined by Gierek (3) was subtracted, normal hearing (up to 15 dB) was found in 22 ears, and a sensorineural hearing loss in 15 ears. Both groups of patients were roughly balanced as to the age. The hearing loss appeared during the treatment, just after that or in several months after the therapy. Of 15 ears with a hearing loss, 5 ears had a mild hearing loss (20—35 dB), 6 ears had a moderate one (40—60 dB) and 4 ears had a hearing loss greater than 60 dB.

Acoustic impedance measurements showed normal middle ear compliance and pressure with normal tympanogram in all the ears tested. In the patients without hearing loss, stapedial reflex threshold ranged from 55 to 95 dB. Three of 22 ears of that group had stapedial reflex threshold at 55 dB sensation level indicating the presence of recruitment. In the patients with STM induced hearing loss, stapedial reflex threshold ranged from 35 to 85 dB, and in 11 of 15 ears stapedial reflex was found at 55 dB or less. The difference between both groups in the number of ears with low stapedial reflex threshold indicating the presence of recruitment was significant (Table 1).

Table 1. Stapedial reflex threshold (SRT) in 37 ears of patients treated with STM

Groups of patients	Range of SRT dB	Number of ears with SRT $\leq$ 55 dB	Probability of difference
Normal hearing /22 ears/	55–95	3	$p < 0.01$
Hearing loss /15 ears/	35–85	11	

The SISI scores at 0.5, 1 and 2 kHz were low (0—30%) in all the ears tested. At 4 kHz in the group with normal hearing, 19 of 22 ears had low SISI score, two ears had a moderate one (35—65%) and one ear

had a high SISI score (70—100%). In the group with STM induced hearing loss, 3 of 15 ears had a low SISI score at 4 kHz, one ear had a moderate score, and eleven ears had a high SISI score indicating the presence of recruitment. The difference between both groups in the number of ears with high SISI score was significant (Table 2).

The tone decay test showed normal auditory adaptation (20 dB and less) in all the ears without STM hearing loss. Of 15 ears with STM induced hearing loss, 13 ears had normal adaptation and two ears had slightly abnormal adaptation (25 dB) at 4 kHz. The separation of the fixed frequency Békésy tracings at 2 and 4 kHz did not exceed 10 dB in all the ears tested.

Table 2. SISI scores in 37 ears of patients treated with STM

Groups of patients	0.5, 1, 2 kHz	4 kHz			Probability of difference
	0–30 %	0–30%	35–65%	70–100%	
Normal hearing /22 ears/	22	19	2	1	$p < 0.001$
Hearing loss /15 ears/	15	3	1	11	

DISCUSSION

Tone decay test and fixed frequency Bekesy tracing showed auditory adaptation to be within normal limits. The high SISI score and the low stapedial reflex threshold we found in the patients with STM induced hearing loss indicate the presence of recruitment in these patients. Recruitment is known to be an indication of cochlear lesion in the auditory system. Hibner and Janczewski (6) found high SISI score (over 60%) in 92% of the ears with cochlear hearing loss in patients with Meniere's disease and in patients with acoustic trauma. In similar group of patients, Sanders et al. (14) found SISI score to be over 70% in 76% of ears and low stapedial reflex threshold (below 55 dB) in 37% of ears tested.

The data from the literature concerning the site of ototoxic effect of STM are ambiguous. Some authors found that STM deteriorated central pathways of the auditory and vestibular system within the brain stem and within the cerebellum. Histologic studies showed features of degeneration in the eighth nerve nuclei, in the ceribellum and in the spiral and vestibular ganglions (11, 16). The decrease in nucleic acids content in the vestibular ganglion was also found (2).

However, most recent studies support the view that the primary site of STM lesion is the peripheral hearing and equilibrium system. In animals treated with STM a degeneration of ciliary cells of the cochlear and vestibular organs was found (8, 15). In addition to the morphologic lesions, metabolic alterations in the cochlea (4, 12) and a decrease in cochlear microphonics was shown (5, 10). Hawkins (5) and Holtz et al. (7) found a deterioration of sensory cells of the cochlea but they also found a degeneration of the spiral and vestibular ganglions. According to Sokołowski (15), the primary site of the toxic effect of STM is the peripheral organ and central STM lesions are secondary to it and they appear after larger doses of STM. McGee and Olszewski (13) also found STM lesions to be limited to the inner ear and a secondary transsynaptic atrophy in the brain stem may be observed as the result of a decrease in afferent stimuli.

Conclusion

The results of this study showed the presence of recruitment in the ears of rhinoscleroma patients with STM induced hearing loss indicating the cochlear lesion.

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STRESZCZENIE

W 37 uszach u 19 chorych na twardziel, leczonych dużymi dawkami streptomycyny, wykonano audiometrię progową, impedancyjną i automatyczną Békésy'ego oraz próbę SISI i próbę zanikania tonu progowego Carharta. W 11 przypadkach spośród 15 uszu z odbiorczym ubytkiem słuchu stwierdzono wysokie wartości próby SISI i obniżony próg odruchu strzemiączkowego, wskazujące na obecność wyrównania głośności. Adaptacja słuchowa we wszystkich uszach znajdowała się w zakresie normy.

РЕЗЮМЕ

На 37 исследованных ушей больных склерозом леченных большими дозами стрептомицина, авторы произвели аудиометрию тональную, импедансионную и автоматическую по Бекеши, тест SISI и тест по Каргарту. Из 15 исследованных ушей с рецептивным нарушением слуха — 11 имело высокие результаты в тесте SISI и низкий порог стремянномышечного рефлекса, свидетельствующие о выравнивание громкости. Слуховая адаптация во всех ушах была нормальна.