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Effects of drill noise on the Auditory Threshold of the guinea pigs under occluding the round window and Its Correlation with the Pathological Mechanisms of Noise-Induced Hearing Loss

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Abstract

Objective: To investigate the synergistic damaging effects of electric drill noise on auditory thresholds under round window occlusion conditions, and to systematically analyze the pathological mechanisms of noise-induced hearing loss (NIHL) with advances in integrated Traditional Chinese and Western medicine prevention and treatment strategies. *Methods:* Thirty-six healthy male guinea pigs were randomly divided into control, round window occlusion-only, noise exposure-only, and occlusion + noise combined groups. A round window occlusion model was established, and animals were exposed to 92.5 dB SPL electric drill noise with a dominant frequency of 10 kHz for 30 minutes. Auditory brainstem response (ABR) testing was used to evaluate changes in auditory thresholds. *Results:* The occlusion + noise combined group exhibited a significant and persistent increase in ABR thresholds at 16 kHz. *Conclusion:* Round window occlusion enhances inner ear susceptibility to electric drill noise, leading to frequency-specific auditory threshold elevation. Combined with NIHL mechanisms such as oxidative stress and neuroinflammation, integrated Traditional Chinese and Western medicine interventions may offer novel clinical strategies for prevention and treatment.

Keywords

Round window occlusion; Electric drill noise; Noise-induced hearing loss; Auditory threshold; Oxidative stress; Integrated Chinese and Western medicine prevention and treatment

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1. Introduction

The round window membrane, a critical structure between the middle and inner ear, plays an indispensable role in maintaining normal auditory physiology, particularly by ensuring cochlear fluid pressure balance and lymphatic vibration stability. Clinically, round window occlusion may occur due to congenital anomalies, otosclerosis, chronic otitis media, or post-middle ear surgery [1–3]. Early

studies, such as those by Tonndorf et al., suggested that isolated round window occlusion has limited impact on auditory thresholds [4]. However, further research indicates that alterations in round window status may profoundly influence the inner ear's response to various insults.

Perez et al. demonstrated that while round window occlusion alone does not alter thresholds, it significantly increases susceptibility to subsequent noise exposure, exacerbating hearing loss [5]. This finding highlights the round window's functional integrity as a key defense against external stressors. Concurrently, electric drills are essential tools in otological microsurgery, but their high-intensity noise poses a risk of iatrogenic hearing damage. Domenech et al. reported high-frequency sensorineural hearing loss following tympanoplasty, attributing it to drill-generated acoustic trauma [6]. Bao Shiping's studies further confirmed the direct impact of drill noise on guinea pig auditory function [7].

A critical clinical question arises: Does pre-existing round window occlusion synergize with inevitable intraoperative drill noise exposure to produce a "1 + 1 > 2" effect? Although prior research has explored round window physiology and noise damage separately, systematic evidence on synergistic injury, particularly regarding frequency specificity, remains scarce. This study establishes a guinea pig model of round window occlusion combined with electric drill noise exposure to systematically investigate this synergistic effect and provide theoretical insights for clinical practice.

2. Literature review: Pathological mechanisms and prevention strategies for NIHL

2.1. Definition and epidemiology of NIHL

NIHL typically presents as threshold shifts characterized by high-frequency hearing loss. Based on reversibility, it is classified as temporary threshold shift (TTS), permanent threshold shifts (PTS), or hidden hearing loss. Globally, noise exposure is a leading preventable cause of hearing disability, with prevalence rates of 30–50% among high-risk occupational groups. Notably, recreational noise exposure from personal audio devices has led to increasing NIHL incidence among adolescents, posing a significant public health burden [8,9].

2.2. Multilevel pathogenesis

2.2.1. Mechanical and metabolic damage

High-intensity sound waves first cause direct mechanical trauma to cochlear structures, disrupting CDH23 protein links between stereocilia of inner and outer hair cells (OHCs), leading to disarray, bending, or rupturing. Severe mechanical damage may perforate Vestibular membrane, mix perilymph and endolymph, and induce potassium influx that exacerbates hair cell dysfunction [8,10]. Metabolic damage, secondary to mechanical injury, is central to hair cell apoptosis, with oxidative stress as a core mechanism.

2.2.2. Neuroinflammation and excitotoxicity

Following noise exposure, inflammatory cells such as macrophages in the bloodstream infiltrate the cochlea and become locally activated, releasing potent pro-inflammatory factors like tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β). While this neuroinflammatory response helps clear cellular debris, it can also inadvertently damage nearby surviving hair cells and supporting cells, thereby expanding the initial area of injury [11]. On the other hand, excessive acoustic stimulation can lead to the over-release of the neurotransmitter glutamate from inner hair cells to the auditory nerve terminals, resulting in excitotoxicity.

2.3. Advances in Western medicine interventions

2.3.1. Pharmacological strategies

Targeting the core mechanism of oxidative stress, antioxidants are currently the most extensively studied pharmacological agents. For instance, N-acetylcysteine, a precursor of glutathione, effectively elevates cochlear glutathione levels by approximately 30%, thereby significantly enhancing cellular antioxidant capacity and shortening the recovery time of temporary threshold shifts by 40%. Neurotrophic drugs such as methionine combined with vitamin B12 can cross the blood-labyrinth barrier; animal studies have demonstrated their ability to reduce permanent threshold shifts by up to 50% [10]. To improve drug delivery efficiency, novel delivery systems, such as liposome-encapsulated superoxide dismutase, have been developed. These systems can increase local drug concentrations in the cochlea by tenfold,

substantially enhancing therapeutic efficacy.

2.3.2. Emerging therapies

With the advancement of molecular biology, gene therapy and cell therapy have brought new hope for the repair of noise-induced hearing loss (NIHL). Studies utilizing adenoviral vectors to deliver the Math1 gene into the cochlea, leading to its overexpression in supporting cells, have successfully induced the transdifferentiation of supporting cells into functional new hair cells. Animal models have demonstrated approximately 30 dB of hearing improvement. Additionally, mesenchymal stem cell transplantation has been shown to promote the repair of damaged synapses and enhance cell survival through its paracrine effects, which include the release of various neurotrophic factors such as brain-derived neurotrophic factor (BDNF) [9].

2.4. Traditional Chinese medicine (TCM) approach

2.4.1. Etiology and treatment principles

NIHL falls under "chronic deafness" or "sudden deafness" in TCM. The core pathogenesis is primarily attributed to deficiency of the liver-kidney, resulting in insufficient essence and blood failing to nourish the ears, leading to malnourishment of the auditory orifices; or due to invasion of external wind pathogens that ascend along the meridians, obstructing the clear orifices; or phlegm-fire stagnation that blocks the ear collaterals, causing disharmony of Qi and blood and occlusion of the auditory orifices [12]. The fundamental treatment principle is based on "dredging the meridians' Qi and harmonizing Qi and blood", emphasizing a holistic therapeutic approach that combines local acupoint selection with distal points based on pattern differentiation.

2.4.2. TCM therapies and mechanisms

Acupuncture therapy is a characteristic modality in Traditional Chinese Medicine for the treatment of hearing loss. Local acupoints around the ear, such as Yifeng (TE17), Tinggong (SI19), Ermen (TE21), Tinghui (GB2), and Jiaosun (TE20), are frequently selected to dredge the Qi of the Shaoyang Meridian and directly stimulate Qi and blood circulation in the auditory region. Distal acupoints like Taixi (KI3) and Sanyinjiao (SP6) are combined to nourish the kidney and replenish essence, thereby reinforcing the congenital foundation, while Fenglong (ST40) and Hegu (LI4) are used to resolve phlegm, clear fire, and diffuse lung Qi. Clinical studies have shown that acupuncture treatment can improve the pure-tone hearing thresholds of patients with sensorineural hearing loss by an average of 15–25 dB, with a total efficacy rate reaching 82.3% [12]. Integrated therapies demonstrate greater advantages; for example, the combined application of Tongqiao Huoxue Tang (Orifice-Unblocking and Blood-Invigorating Decoction) and acupuncture has been shown to significantly increase cochlear blood flow by up to 30%, thereby improving the microcirculation of the inner ear.

3. Materials and methods

3.1. Animals and grouping

Thirty-six specific-pathogen-free (SPF) healthy male guinea pigs (250–300 g) were acclimatized for one week and randomly divided into four groups (n = 12/group): control, occlusion-only, noise-only, and occlusion + noise combined.

3.2. Model establishment

3.2.1. Round window occlusion

The animals were anesthetized via intraperitoneal injection of sodium pentobarbital. After the anesthesia took effect, the surgical site was disinfected with povidone-iodine. An incision was made layer by layer through the skin and subcutaneous tissues along the medial side of the mandible. Under microscopic guidance, blunt dissection was performed to separate the submandibular gland, digastric muscle, and surrounding connective tissues to fully expose the tympanic bulla. The bulla was opened approximately 0.5 cm anterior to the styloid process, adequately exposing the cochlea, stapes, round window niche, and membranous structures. Autologous periosteal tissue harvested from the animal's own tympanic bulla was trimmed to an appropriate size and tightly packed into the round window niche. Medical fibrin glue was applied to secure the graft, ensuring complete occlusion of the round window. Postoperatively, the incision was closed layer by layer, and intramuscular injections of cefradine were administered for three

consecutive days to prevent infection.

3.2.2. Noise exposure

The noise source utilized a NOUVAG high-speed electric drill, commonly employed in clinical otological surgery, operating at a speed of 26,000 bpm and equipped with a 2 mm diameter diamond polishing burr. Within a soundproof chamber, calibrated using a precision sound level meter, the noise intensity generated by the drill was measured at the animal's head position as 92.5 dB SPL, with the primary energy peak of the noise located at 10,000 Hz. A steel ring was positioned behind the left ear of the guinea pig, snug against the temporal bone. The polishing burr was then used to deliver drill noise stimulation for a total duration of 30 minutes, administered in cycles of 5 minutes of noise exposure followed by 1-minute intervals, amounting to a total procedure time of 35 minutes.

3.3. Auditory assessment

ABR thresholds were measured at 8, 16, and 24 kHz preoperatively, immediately post-exposure, and at 3 months. Wave III was used as the response threshold.

Auditory brainstem response (ABR) testing was employed to evaluate auditory thresholds in guinea pigs. Measurements were conducted at three time points: preoperatively, immediately post-exposure, and 3 months postoperatively. After anesthesia, the external auditory canal was cleared of cerumen, and otoscopic examination confirmed intact tympanic membranes without perforation or discharge. Animals were placed in a prone position on a testing platform within a soundproofed electrically shielded chamber.

A needle recording electrode was positioned at the midline of the cranial vertex, with a reference electrode placed on the ipsilateral earlobe and a ground electrode attached to the nasal tip. Tone burst stimuli at frequencies of 8 kHz, 16 kHz, and 24 kHz were delivered. The auditory threshold was defined as the lowest intensity level that elicited a clear and reproducible Wave III response.

3.4. Statistical analysis

All data are expressed as mean ± standard deviation. Statistical analyses were performed using SPSS 20.0 software. To verify baseline equivalence across experimental groups prior to interventions, a one-way analysis of variance (ANOVA) was conducted to compare preoperative ABR thresholds among the four groups. For longitudinal comparisons, independent samples *t*-tests were employed to analyze: Intergroup differences in ABR thresholds immediately post-operation, Intergroup differences in ABR thresholds at the 3-month postoperative timepoint changes from preoperative baseline values at both postoperative intervals. The threshold for statistical significance was set at $p < 0.05$ for all analyses.

4. Results

4.1. ABR threshold changes

Preoperatively, no significant differences were observed in the baseline ABR thresholds at the three test frequencies (8 kHz, 16 kHz, and 24 kHz) among the four experimental groups, indicating comparable baseline hearing levels across all groups (**Figure 1**).

Postoperative testing immediately following the intervention revealed that neither the round window occlusion-only group nor the noise exposure-only group showed statistically significant differences in ABR thresholds compared to the control group. In stark contrast, the occlusion + noise combined group demonstrated significant and frequency-specific alterations in auditory thresholds. Specifically, at the 16 kHz frequency, this group exhibited an abrupt elevation in ABR threshold from a preoperative value of 4.58 dB to 43.3 dB immediately after the procedure, representing a substantial threshold shift of 38.72 dB. This change was determined to be highly statistically significant ($p < 0.01$). Long-term follow-up assessments conducted at 3 months postoperatively demonstrated that the occlusion + noise combined group maintained persistently elevated thresholds at 16 kHz, with an average value of 42.9 dB. This sustained threshold elevation provides compelling evidence that the synergistic damage (**Figure 2** to **6**).

5. Discussion

This study, by establishing a guinea pig model combining round window occlusion with electric drill noise exposure,

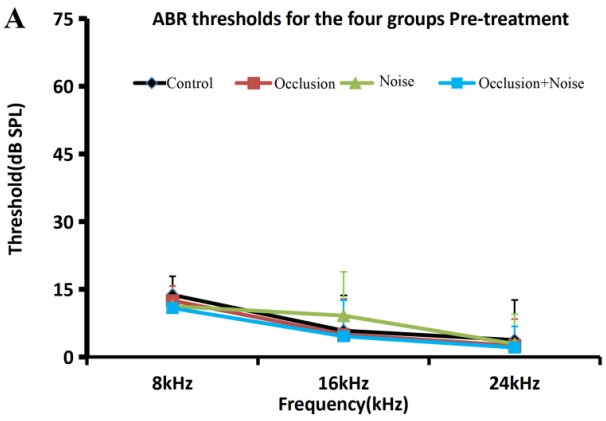

Figure 1. ABR thresholds for the four groups Pretreatment.

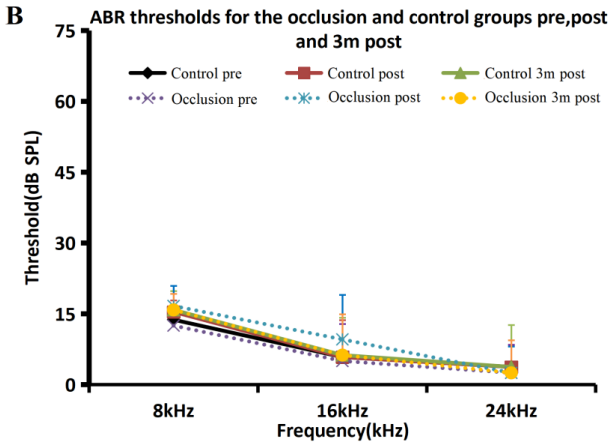

Figure 2. ABR thresholds for the occlusion and control groups pre, post and 3m post.

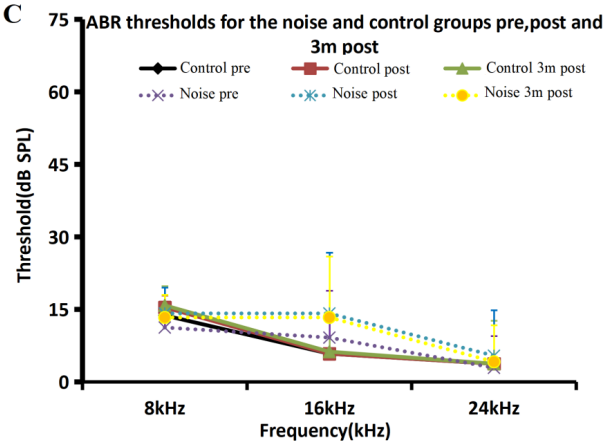

Figure 3. ABR thresholds for the noise and control groups pre, post and 3m post.

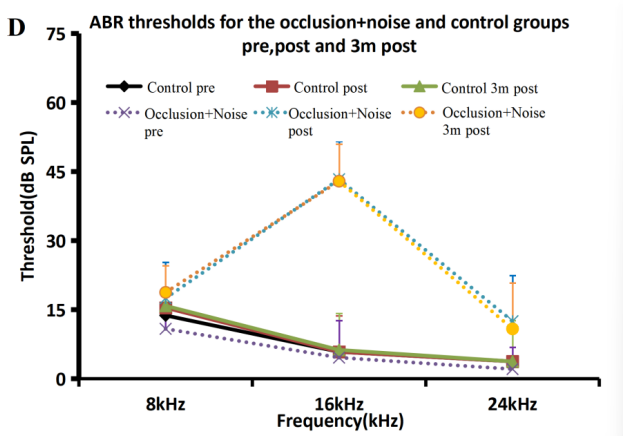

Figure 4. ABR thresholds for the occlusion + noise and control groups pre, post and 3 m post.

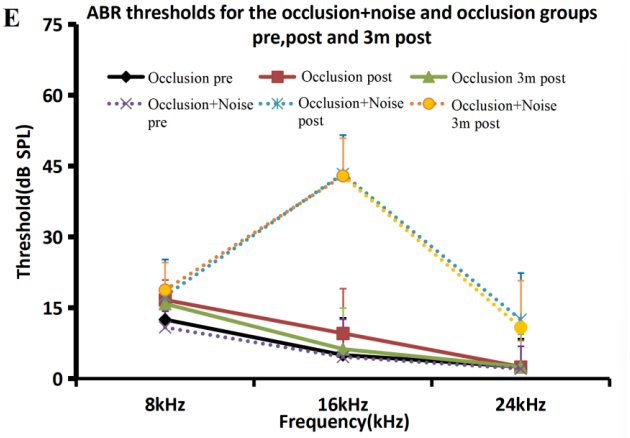

Figure 5. ABR thresholds for the occlusion + noise and occlusion groups pre, post and 3m post.

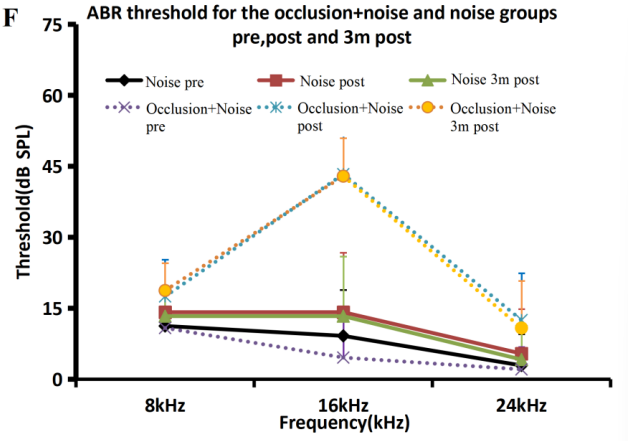

Figure 6. ABR threshold for the occlusion + noise and noise groups pre, post and 3m post.

confirmed the existence of a synergistic damaging effect between the two factors. The results demonstrated that neither round window occlusion alone nor exposure to 92.5 dB electric drill noise alone was sufficient to induce significant permanent threshold shifts. However, when combined, they resulted in severe hearing loss centered at 16 kHz. This finding not only corroborates the academic viewpoint proposed by Perez et al. that round window occlusion enhances inner ear susceptibility to noise, but also deepens the understanding of its harm from the perspectives of frequency specificity and persistence [13].

From a mechanistic standpoint, the round window serves as a critical "pressure release valve" for the cochlea, and its normal function is essential for maintaining the hydrodynamic balance of inner ear fluids. Given that the mobility of the round window is five times greater than that of the oval window, and the volume ratio of the scala vestibuli to the scala tympani is 5:3, during the condensation phase of the sound wave, the compressed bony wall causes perilymph in the semicircular canals to be forced into the larger-volume scala vestibuli, which then flows into the smaller-volume scala tympani. Furthermore, the mobility of the round window membrane exceeds that of the stapes footplate, leading to basilar membrane displacement towards the scala tympani (downward) and outward bulging of the round window membrane. During the rarefaction phase, the bony labyrinth wall rebounds, the lymph fluids return to their original positions, the basilar membrane displaces upward to its original state, and the oval window bulges outward [14–16]. Therefore, both air and bone conduction stimulation require movement between the cochlear wall and the oval window, the presence of two mobile windows, and a pressure difference across the basilar membrane [14]. This highlights the critical importance of the oval and round windows. Conventionally, it is believed that during this process, when the stapes footplate moves, the round window membrane acts to cushion pressure changes within the cochlea and is a necessary condition for vibration of the intracochlear structures. Because inner ear fluids are incompressible, complete occlusion of the round window should reduce the mobility of the stapes footplate, consequently leading to conductive hearing loss [17]. When the round window is occluded by packing material, the compliance of cochlear fluid vibration decreases significantly, potentially altering the normal traveling wave pattern of the basilar membrane and the pathway of pressure transmission, thereby weakening the cochlea's natural protective mechanisms against intense sound stimuli.

Combined with the pathological mechanisms of noise-induced hearing loss, this initial mechanical damage rapidly triggers a cascade of secondary pathological events. Hair cells, hypermetabolic in response to overstimulation, undergo a sharp increase in oxidative stress levels. Excessive ROS production attacks stereocilia structures and cell membrane systems, which likely directly contributed to the observed widespread stereocilia disarray, breakage, and even fusion in the experiment. Concurrently, metabolic disturbances lead to calcium influx and activation of apoptotic pathways, ultimately resulting in programmed cell death of hair cells, manifesting as extensive cell loss. Additionally, damaged hair cells and activated supporting cells release chemokines, attracting macrophage infiltration and triggering local neuroinflammation. The release of inflammatory cytokines further expands the scope of damage.

The findings of this study carry clear clinical implications. For patients with pre-existing conditions such as otosclerosis or chronic otitis media, who may inherently have round window occlusion or similar structural abnormalities, the risk of synergistic noise damage during otologic surgeries requiring drill use is significantly elevated. Based on this, we recommend preoperative assessment of round window status for such high-risk patients using methods like high-resolution temporal bone CT. If definite round window occlusion is identified intraoperatively, and ensuring the primary surgical goal is achievable, careful removal of the obstructive material to restore the mobility of the round window membrane could be considered prior to subsequent drilling procedures.

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Disclosure statement

The author declares no conflict of interest.

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