

CASE REPORT

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Collision injury-induced superior semicircular canal fracture and therapeutic effect of round and oval window reinforcements

Jing Zou, Guiliang Zheng, Rishunzi Peng, Yingna Gao, Shiyue Chen, Hongliang Zheng

ABSTRACT

Introduction: A patient demonstrated sound induced vertigo and hearing loss among others as a result of superior semicircular canal (SSC) fracture was successfully treated using a minimally invasive method of round and oval window reinforcements. **Case Report:** We report a case demonstrating SSC dehiscence syndrome among other symptoms that developed after a car accident. Vertigo was induced by pure tone at 1.5 kHz. An audiogram demonstrated a 10–15 dB air-bone gap at low frequencies and sensorineural hearing loss at frequencies higher than 4 kHz. Cervical vestibular evoked myogenic potential was absent on the left side and was successfully induced on the right side. An magnetic resonance imaging (MRI) using a 3D FIESTA sequence demonstrated an abnormal gap between brain and membranous SSC,

and a high-resolution computed tomography (CT) scan with reconstruction of the panel of SSC confirmed the fractures on the SSC wall. The symptoms were well controlled by round and oval window reinforcements. A fibrous connection between the caput stapedis and the upper structures was identified, and the ossicular chain was reconstructed using a partial ossicular replacement prosthesis. More than 20 dB improvement was obtained in bone conduction at 4 kHz. **Conclusion:** Collision injury-induced SSC wall fracture might display symptoms that are similar to SSC dehiscence syndrome. A combination of magnetic resonance imaging and high resolution CT with reconstruction of the panel of the potentially injured semicircular canal is recommended to identify the lesion site. Round and oval window reinforcements proved to be an efficient management technique for collision injury-induced SSC dehiscence syndrome.

Keywords: Imaging, Semicircular canal dehiscence, Surgery, Trauma, Vertigo

Jing Zou^{1,2}, Guiliang Zheng¹, Rishunzi Peng¹, Yingna Gao¹, Shiyue Chen³, Hongliang Zheng¹

Affiliations: ¹Department of Otolaryngology-Head and Neck Surgery, Center for Otolaryngology-Head & Neck Surgery of Chinese PLA, Changhai Hospital, Second Military Medical University, Shanghai, China; ²Hearing and Balance Research Unit, Field of Oto-laryngology, School of Medicine, University of Tampere, Tampere, Finland; ³Department of Radiology, National Key Discipline, Changhai Hospital, Second Military Medical University, Shanghai, China.

Corresponding Author: Jing Zou, MD, Ph.D, Professor, Department of Otolaryngology-Head and Neck Surgery, Changhai Hospital, Second Military Medical University, Changhai Road #168, Shanghai 200433, China; Tel: +86 21 81873220, Fax: +86 21 81873220; Email: zoujinghb@hotmail.com, jing.zou@uta.fi

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INTRODUCTION

Superior semicircular canal dehiscence (SSCD) syndrome was first reported by Minor et al. in 1998 [1]. The management of SSCD consists of either surgically plugging or rerooting the deficient SSC via a middle fossa craniotomy or transmastoid approach that results in the resolution or improvement of SSCD symptoms after a brief period of central compensation, but the technique is not a uniformly effective method [1–6]. Recently, round window reinforcement was shown to be an efficient, minimally invasive procedure for controlling symptoms of SSCD [7]. Hereby, we report a special case who presented with SSCD syndrome in addition to other symptoms that developed after a car accident and were well controlled by gentle round window (RW) and oval window (OW) reinforcements. All the protocols followed the Declaration of Helsinki developed by the World Medical Association.

CASE REPORT

A 47-year-old woman presented with a four-year history of sound-induced vertigo, feelings of body uprush, blurred vision, autophony, and loss of proprioceptive sensation of the head (a feeling that her head did not belong to herself) after a car accident. Four years ago (July 2010), she was thrown into the air together with her bike by a driving car, fell to the floor, and experienced a loss of consciousness. She woke up with her entire body lying on the ground and immediately experienced high tone sound-induced intracranial pain. Five days later, she experienced a loss of proprioceptive sensation of the head, which was more severe on the left side. Twenty days later, she experienced low tone sound-induced vertigo accompanied by astasia, nausea, agitation, and sweating. Especially, she could not tolerate the low frequency vibration caused by a running bus. In addition, she experienced tinnitus, ear fullness, autophony, and hearing loss in the left ear. One year later, she experienced feelings of body uprush (which became more severe with fatigue), weakness, and floating as well as blurred vision. Vestibular hypofunction on the left side was detected in the Department of Neurology in a hospital in Xi'an, and flunarizine was administered. Her sound-induced vertigo was partially relieved, but recurred in relation to changes in head position and lasted for minutes or hours. Three months later, she visited the same hospital and was treated with flunarizine together with other medicines without further effects. She visited the Department of Otolaryngology, Head, and Neck Surgery in Changhai Hospital on April 30, 2014. Cervical vestibular-evoked myogenic potentials (cVEMPs) were absent on the left side and exhibited a low amplitude on the right side (Figure 1A). Vertigo lasting for several seconds was induced by 1.5 kHz transient pure tone (1 second) at 90 dB nHL,

and a vertigo lasting for 15 minutes post-stimulation was evoked by a pure tone exposure at 100 dB nHL. A high-resolution temporal bone computed tomography (CT) scan revealed a “bony defect” on the left SCC. She visited another hospital in Shanghai on May 8, 2014. An audiogram demonstrated 10–15 dB air-bone gaps at low frequencies and sensorineural hearing loss at frequencies higher than 4 kHz (Figure 2A). The SSCD diagnosis was denied, and the patient was treated with mecobalamin tablets and “neurotrophin” without any effects. The patient visited Changhai Hospital again and MRI scan was performed using three-dimensional fast imaging employing a steady-state acquisition (FIESTA) sequence in a 3.0 T machine and demonstrated a narrow distance between the brain and the membranous SSC (Figure 3). The CT reconstruction on the SSC plane demonstrated a thin bony roof of the left SSC with multiple fractures. The bony roof of the right SSC revealed a single fracture (Figure 4). cVEMPs remained absent on the left side and exhibited a higher amplitude on the right side compared with the previous examination (Figure 1B). Osteoporosis measured on the cervical spinal revealed normal results. The patient was hospitalized for treatment using round and oval window reinforcements. Her past medical history included a myringoplasty that was performed in 2002. An exploratory tympanotomy was performed using a traditional transcanal tympanomeatal flap approach under local anesthesia with lidocaine. Curettage was used to enlarge the posterior external auditory canal wall. A fibrous connection between the stapedis caput and the upper structures was identified, and the soft tissue was removed (Figure 5A). The promontory and floor of the RW niche were denuded of mucosa (Figure 5B). RW and OW reinforcements were inserted using abdominal fat to fill the round window, and the procedure continued using “Y-shaped” temporal fascia covering the fat on the round window and surrounding the edge of stapedial footplate (Figure 5C–E). Fibrin glue was used to secure the materials on the RW and OW (Figure 5F). The tympanic membrane was reinforced using cartilage of the conchal that was placed behind the malleus handle, and a titanium partial ossicular replacement prosthesis was used to reconstruct the connection between the tympanic membrane and stapes (Figure 5G). Finally, the tympanomeatal flap was restored and packed using hemostatic gauze as covering and pressed with chlortetracycline eye ointment containing gelatin sponge (Figure 5H). The patient was administered an antibiotic for four days postoperatively to avoid potential infection. Immediately after the operation, the symptoms of sound-induced vertigo, the feeling of body uprush, agitation, blurred vision, tinnitus, ear fullness, and autophony disappeared. The proprioceptive sensation became normal on the left side of the head and improved on the right side. The patient experienced significant improvement in hearing in the left ear. Fourteen days after operation, audiogram demonstrated that the bone conduction at 4 kHz recovered for more than 20 dB and

the air-bone gap increased throughout all the frequencies of in the left ear (Figure 2B), and cVEMP remained absence on the left side and was induced on the right side (Figure 1C).

DISCUSSION

In addition to the symptoms of SSCD, her feelings of body uprush are potentially related to the disturbance of the saccule and SSC. Blurred vision resulted from impaired vestibular ocular reflex and loss of proprioceptive sensation of the head resulted from the vestibular disorder, which was supported by the recovery immediately after RW and OW reinforcements. High tone sound-induced intracranial pain during the early stage potentially resulted from the stimulation of the dura caused by the vibration of sharp bone chips of

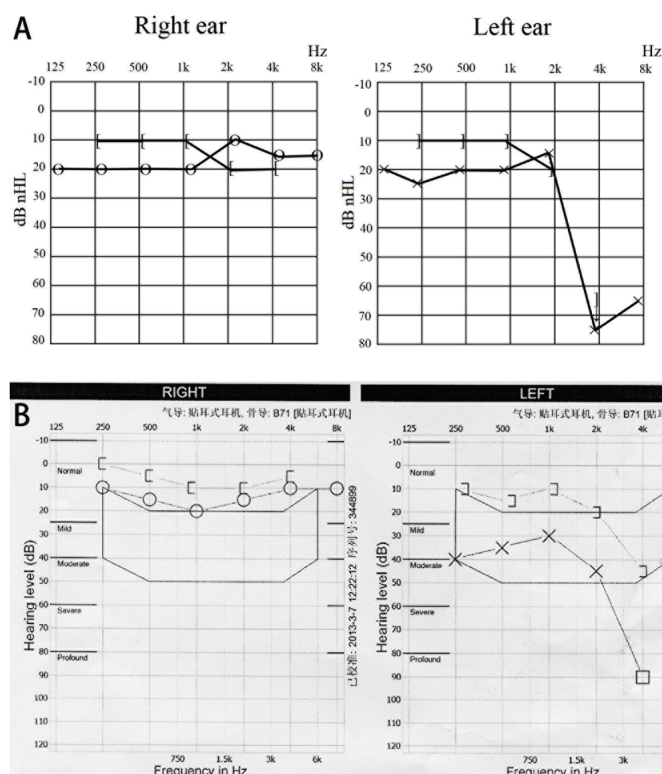


Figure 2: Audiogram performed before and after operation. (A) Before operation, 10–15 dB air-bone gaps at frequencies below 4 kHz appeared in both ears, and sensorineural hearing loss at frequencies higher than 4 kHz was detected in the left ear. (B) After operation, the bone conduction at 4 kHz recovered for more than 20 dB in the left ear.

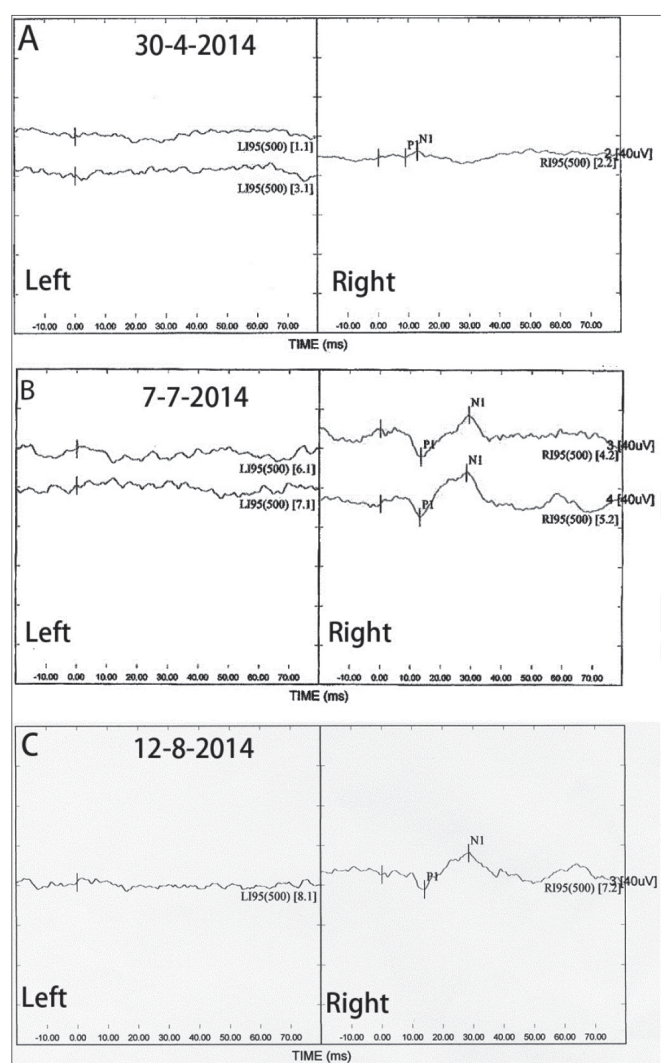


Figure 1: Cervical vestibular-evoked myogenic potential (cVEMP) measurements of the patient at various time points. (A, B) cVEMP was absent on the left side at two measurements before the operation (A, B), and (C) remained no change at 14 days post-round and oval window reinforcement (C). cVEMP was always detected on the right side.

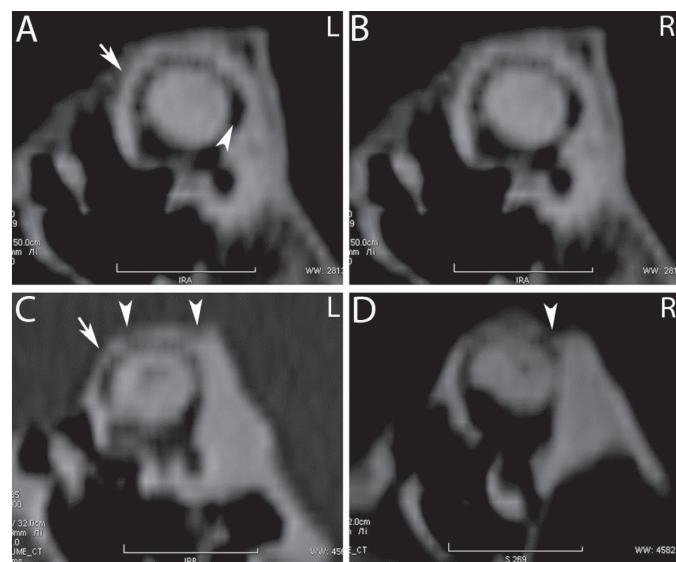


Figure 3: Computed tomography comparison of superior semicircular canals (SSC) between the patient showing superior semicircular canals dehiscence (SSCD) syndrome and a patient without vertigo. Temporal bone volume CT was performed using a Toshiba Aquilion One 640 CT machine. (A, B) Normal SSC of a patient without vertigo exhibited a continuous high intensity signal of the bony wall (arrow) and dark region of the membranous SSC (arrowhead). (C, D) The patient with SSCD syndrome exhibited a thin SSC wall (arrow) and fractures (arrowheads). Abbreviation: L left side, R right side Scale bar = 1.0 cm

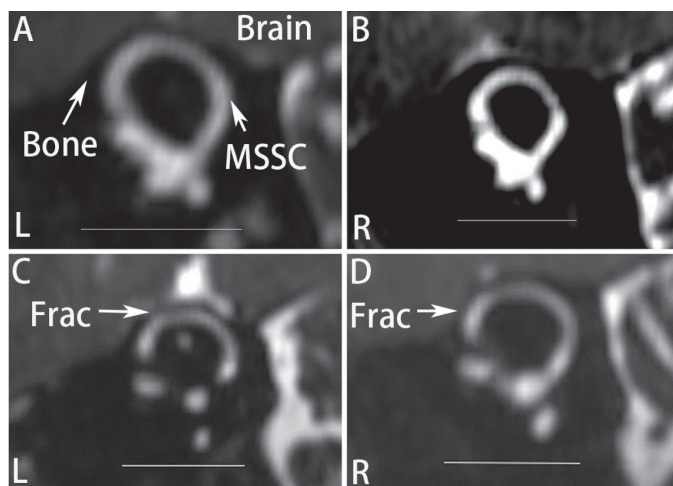


Figure 4: Magnetic resonance imaging scan comparison of superior semicircular canals (SSC) between the patient with SSC dehiscence (SSCD) syndrome and a patient without vertigo. Three-dimensional fast imaging employing a steady-state acquisition (FIESTA) sequence was used to acquire the images with a 3.0 T machine (GE Signa HDxt 3.0T MRI system, 8-channel HD head coil). (A, B) A continuous dark border was clearly observed between the grey signal of the brain and the bright membranous SSC (MSSC) in a patient without vertigo, and (C, D) The distance between the grey brain and bright MSSC in the patient with SSCD syndrome was narrow even disappeared.

Abbreviation: L: left side, R: right side, Frac, fracture
Scale bar = 1.0 cm

the fractured SSC. The absence of the intracranial pain 20 days after trauma can be interpreted as a result of the smoothing of the sharp bone chips caused by bone remodeling. The atypical audiogram is potentially explained by the fact that the mobility of the fractured bony roof of the SSC is reduced compared with typical dehiscent SSC. The labyrinth concussion also potentially complicated the hearing loss pattern. It was reported that 54.8% patients with labyrinth concussion exhibited a descending type hearing loss [8]. The evident recovery on bone conduction at 4 kHz did not support that the contribution of labyrinth concussion to the hearing loss and the mechanism is unknown. An incomplete ossicular discontinuity caused an air-bone gap that is more severe at higher frequencies and peaked at 4 kHz [9]. These unusual contributions produced a unique hearing loss in the patient and made a diagnosis difficult.

The therapeutic mechanism of RW and OW reinforcements is to reduce the third window effect by inhibiting energy transfer within the vestibule and finally eliminate the symptom of vestibular over irrigation which was observed in the patient immediately after the surgery. Complete occlusion of the round window using varied materials including cartilage, bone wax, perichondrium, muscle, and fascia was reported by Silverstein and Van Ess in 2009 and followed by Nikkar-Esfahani et al. However, the outcome was reportedly

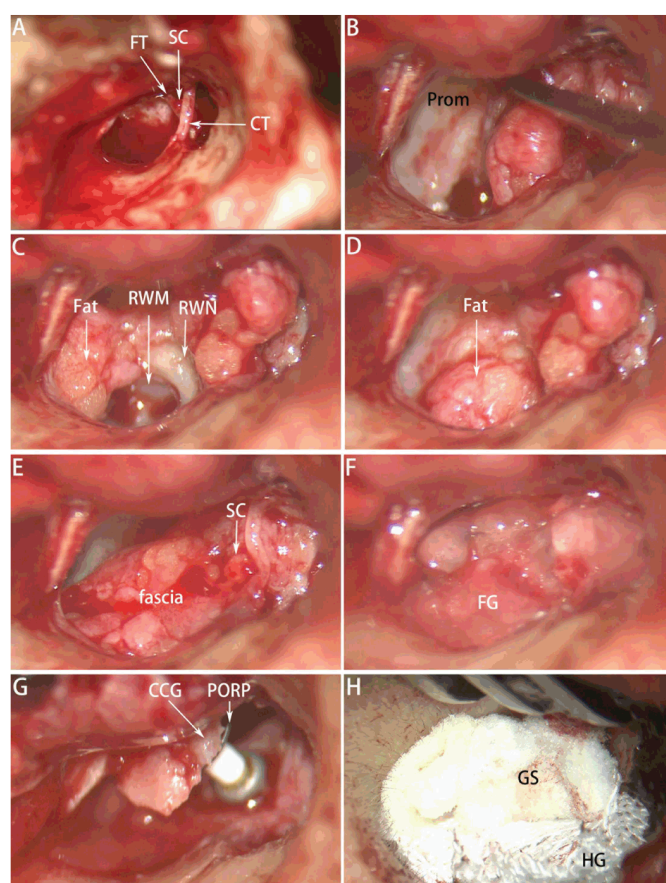


Figure 5: Procedures for round and oval window reinforcements and partial ossicular replacement prosthesis implantation. (A) A fibrous connection exists between the stapedis caput and the upper structures, (B) The promontory (Prom) and floor of the round window niche were denuded of mucosa, (C, D) A small piece of fat was used to fill the round window, (E) A shaped temporal fascia was used to cover the fat on the round window and surround the edge of the stapedial footplate, (F) Fibrin glue was used to secure the materials on the round and oval windows, (G) A conchal cartilage graft was placed behind the malleus handle, and a titanium PORP was used to rebuild connection between the tympanic membrane and stapes, and (H) Hemostatic gauze was placed on the surface of the restored tympanomeatal flap and pressed with a chlortetracycline eye ointment containing gelatin sponge.

Abbreviation: CCG Conchal cartilage graft, CT chorda tympani, FG Fibrin glue, FT fibrous tissue, GS gelatin sponge, HG Hemostatic gauze, PORP Partial ossicular replacement prosthesis, RWM round window membrane, RWN round window niche, SC stapedis caput.

unrepeatable and unstable [7, 10]. It was postulated that gently reinforcing both the OW and RW would be safer and more effective than severely dampening the RW alone. This method may obviate the need for a second procedure if RW reinforcement alone proves unhelpful for a particular patient [7]. The dramatic change is that the patient had a sensation of her head and the body stopped uprushing when the RW and OW reinforcements were done during operation that indicates an elimination of the vestibular over irrigation and recovery of the proprioceptive sensation. Hearing recovery for 20 dB at 4

kHz postoperative supported that the previous reduction in bone conduction at 4 kHz was associated with an incomplete ossicular discontinuity. disappearance of autophony and enlarged air-bone gaps in the left ear after operation may be due to immobilization of the stapedial foot plate caused by the fibrin glue and reinforcements of the two windows. However, the patient experiences an improvement in the hearing. This indicates that the hearing sensation in the human is a complex course other than the pure tone detection. Although the fractured bony roof of the right SSC was not treated, the vestibular symptoms almost disappeared on both sides. We suspected that the bone chip in the right ear was still linked to the main part of the SCC through either a thin bony structure which is out of the resolution of the CT scan for demonstration or a soft tissue with low mobility.

The absence of cVEMPs was potentially caused by lesion either on the ipsilateral descending projections via the lateral vestibulospinal tract, or on the pathway of vestibular nuclei —medial vestibulospinal tract—accessory nucleus. The efficient control on the vestibular symptom after round window and oval window reinforcement that inhibits energy transfer within the vestibule immediately after the surgery does not support lesions in the brainstem or cortex.

CONCLUSION

In conclusion, collision injury-induced superior semicircular canals (SSC) wall fracture may display symptoms that are similar to superior semicircular canal dehiscence (SSCD) syndrome but more complicated depending on the extent of the impairment. Round window and oval window reinforcements is a minimally invasive procedure that serves as an efficient management method for collision injury-induced SSCD syndrome.

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Author Contributions

Jing Zou – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guiliang Zheng – Acquisition of data, Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Rishunzi Peng – Acquisition of data, Analysis and interpretation of data, Revising it critically for important

intellectual content, Final approval of the version to be published

Yingna Gao – Acquisition of data, Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Shiyue Chen – Acquisition of data, Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Hongliang Zheng – Acquisition of data, Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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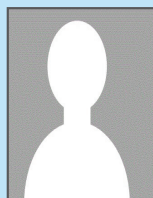
ABOUT THE AUTHORS

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Jing Zou is Professor in Department of Otolaryngology-Head & Neck Surgery at Changhai Hospital, Second Military Medical University, Changhai, China, and Head of Hearing and Balance Research Unit at School of Medicine, University of Tampere, Tampere, Finland. He earned the Bachelor's Degree from Third Military Medical University, Chongqing, China and Master's Degree from Department of Otolaryngology-Head & Neck Surgery, Daping Hospital, Third Military Medical University, Chongqing, China and Doctor's Degree from, Department of Otolaryngology-Head & Neck Surgery, National Key Discipline, Chinese PLA General Hospital, Beijing, China. His research topic for both the Master's

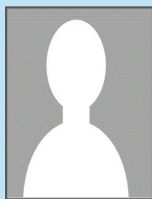
Degree and Doctor's Degree was autoimmune inner ear disease. He did the Post-doc research in Department of Otolaryngology, Karolinska Hospital, Karolinska Institutet, Stockholm, Sweden, on the topic of inner ear MRI and vibration induced inner ear responses, and found that gadolinium-enhanced MRI is capable of demonstrating endolymphatic hydrops in animal model for the first time in the world. He has published 82 research papers in national and international academic journals and authored four books. His research interests include inner ear disease, inner ear imaging, and nanomedicine. We first visualization of endolymphatic hydrops in vivo in animal model and perilymphatic and endolymphatic spaces separately in human using MRI have significantly changed the clinical practice of Otology globally. Afterwards, we have been developing advanced diagnosis and smart drug delivery for the inner ear therapy using nanotechnology using superparamagnetic iron oxide nanoparticles, liposome nanocarriers, gadolinium-fullerenes (C60) derivatives, lipid nanocapsules, polymersomes, hyperbranched polylysine nanoparticles, chitosan nanoparticles, and silica nanogel. Developing neuroprosthesis with a gapless interface to auditory nerve fibers is one of my research aims. Cone beam CT was used to identify scala location of the electrodes of cochlear implant.



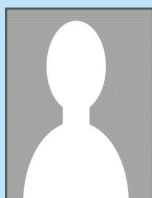
Guiliang Zheng is Attending Physician in Department of Otolaryngology-Head and Neck Surgery, Center for Otolaryngology-Head & Neck Surgery of Chinese PLA, Changhai Hospital, Second Military Medical University, Shanghai, China. He earned Doctor's Degree from, Department of Otolaryngology-Head & Neck Surgery, National Key Discipline, Chinese PLA General Hospital, Beijing, China. His research focuses on noise-induced hearing loss.



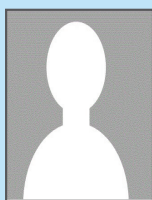
Rishunzi Peng is a candidate of Master's Degree in is Department of Otolaryngology-Head and Neck Surgery, Center for Otolaryngology-Head & Neck Surgery of Chinese PLA, Changhai Hospital, Second Military Medical University, Shanghai, China.



Yingna Gao is Resident in Department of Otolaryngology-Head and Neck Surgery, Center for Otolaryngology-Head & Neck Surgery of Chinese PLA, Changhai Hospital, Second Military Medical University, Shanghai, China.



Shiyue Chen is Resident in Department of Radiology, National Key Discipline, Changhai Hospital, Second Military Medical University, Shanghai, China.



Hongliang Zheng is Professor in Department of Otolaryngology-Head and Neck Surgery, Center for Otolaryngology-Head & Neck Surgery of Chinese PLA, Changhai Hospital, Second Military Medical University, Shanghai, China. He earned Bachelor's Degree, Master's Degree, and Doctor's Degree from, Department of Otolaryngology-Head & Neck Surgery, Changhai Hospital, Second Military Medical University Shanghai, China. His research focuses on vocal cord disorders and head & neck cancers.

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