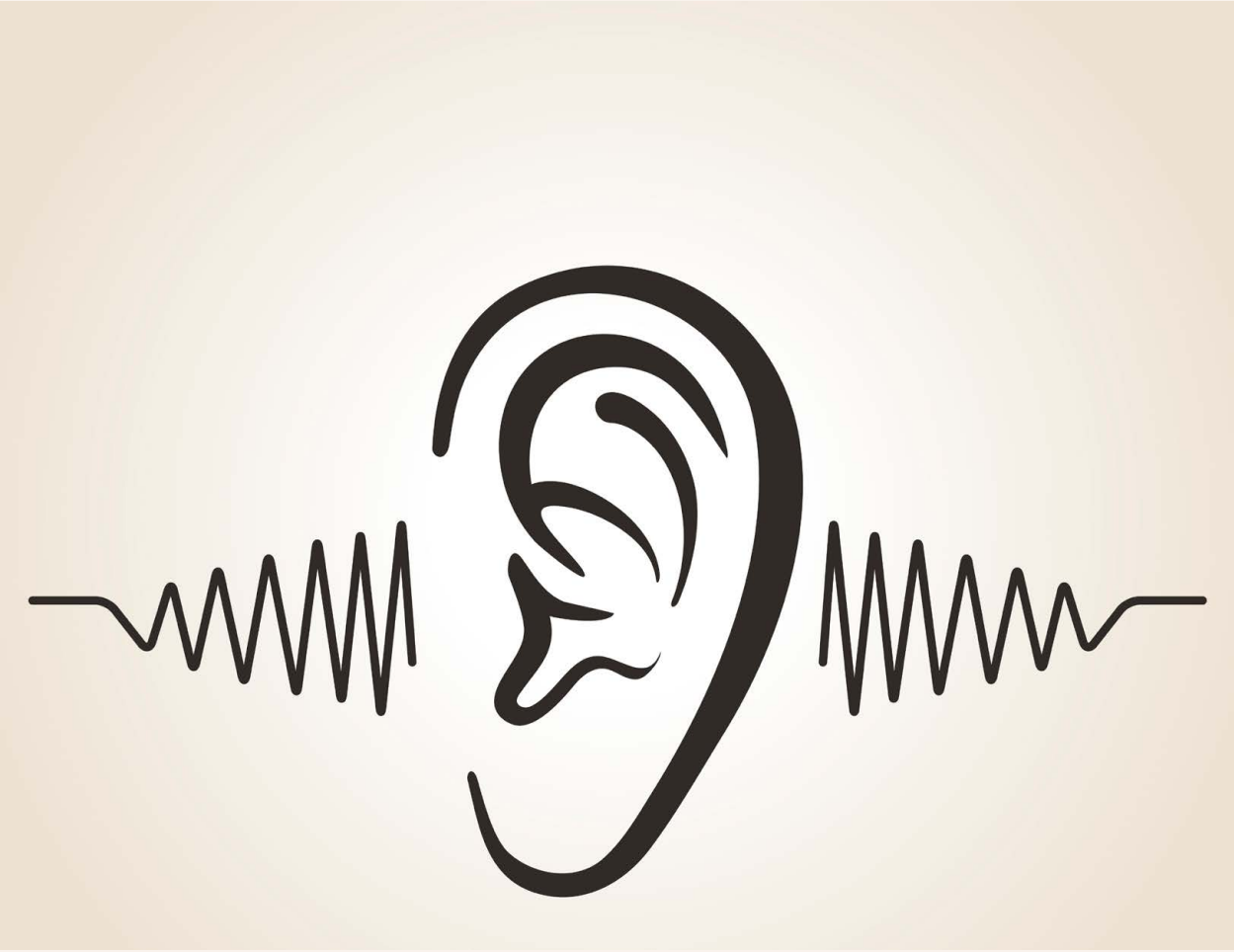
Canadian

Hearing Report

Revue canadienne d'audition

VOL. 15 NO. 2 2021



ISSN: 1718-1860

Published by
ANDREW JOHN PUBLISHING INC.
andrewjohnpublishing.com

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Journal Canadian Hearing Report

cordially invites researchers, audiologists, scholars and students to submit their work in the journal.

We welcome any type of article related to audiology and otology. Please send them at

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Stroke-Related Hearing Loss

By Elizabeth Mills*

INTRODUCTION

Stroke is the most prevalent neurologic disorder, with 130 000 strokes each year in the UK and over a million stroke survivors. It can cause both physical and cognitive disability, and after a year, around one-third of stroke survivors are functionally dependent. The majority of strokes (85–90%) are ischemic, meaning they are caused by a temporary or permanent blockage of a blood vessel, affecting vascular flow to the brain.

Ischemic stroke can be thrombotic, embolic, or caused by venous thrombosis or systemic hypoperfusion. The main portion of the brain afflicted will die immediately because to the presence of collateral circulation, however the surrounding areas (penumbra) have the ability to heal, especially when targeted with suitable therapeutic intervention. Hemorrhagic stroke is caused by the rupture of a blood artery in the brain and occurs in 10–15 percent of all stroke cases.

Hypoxia, irritating effects of the haemorrhage on brain tissues and vasculature, and elevated intracranial pressure owing to bleeding are all possible causes of injury. Both forms of stroke can damage all levels of the auditory pathway, resulting in hearing and/or perception abnormalities that can appear as a wide range of symptoms and clinical presentations that begin promptly before, during, or shortly after the stroke.

Furthermore, risk factors for stroke, such as cigarette smoking, atherosclerosis, and others, have been linked to a more gradual start of hearing loss as people get older, known as age-related hearing loss.

Blood Supply of the Auditory System

Acute stroke damage is more likely to affect the hearing end-organ and auditory nerve in particular. This is because the internal auditory artery (IAA), a small end artery with few types of collateral, provides circulatory supply to the hearing end-organ and nerve. The anterior inferior cerebellar artery (AICA) is the most common source; however there are various anatomic variations. The IAA, as well as collaterals from arteries that supply the dura mater and petrous bone within the internal auditory meatus, as well as an anastamosing network from the AICA, PICA, and vertebral arteries at the cerebellopontine angle and the root entry zone to the brainstem level, provides blood supply to the auditory nerve.

The auditory peripheral, which includes the hearing end-organ and nerve, receives circulatory supply from the same source as the low sections of the brainstem and parts of the cerebellum. As a result, in certain acute-onset post stroke hearing diseases, peripheral-type hearing loss may coexist with auditory brainstem type impairments and/or cerebellar neurologic symptoms and indications.

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Sound Implant in the Active Middle Ear Vibrant Soundbridge

By Elizabeth Mills*

INTRODUCTION

For people who have a solely sensorineural hearing loss, middle ear implants (MEIs) can help. MEIs with unresolvable middle ear conductive/mixed losses were the first clinically accessible MEIs (developed by Suzuki and Yanagihara in Japan). Modern MEIs, on the other hand, require a well-functioning ossicular chain. Since 1935, when Dr. Wilska dusted some iron filings into a person's eardrum, MEIs have existed in some form or another. The iron filings were subjected to a magnetic field created by a coil of wire inside an earphone. Despite the fact that there was no acoustic sound energy flowing from the earpiece, the participants experienced 'hearing.' The iron filings vibrated in time with the magnetic field due to the earphone's magnetic field. This vibration prompted the eardrum to vibrate as well, allowing sound to be transmitted normally to the inner ear. Several research groups across the world have attempted to develop a wearable MEI since the 1930s. With less than 3 mA, current MEIs can create 85 dB.

The receiver or the complete hearing aid is surgically implanted into the middle ear with a MEI hearing aid. There are two benefits to having such an implant. First, if the ossicles can be driven directly, sound quality may be enhanced without any feedback. Second, a MEI may be implanted entirely without any external components. Two companies have now developed fully implanted middle ear implants. Furthermore, depending on the MEI, there is no insertion loss with a net gain in high-frequency sound transmission if there is no device in the ear canal.

Direct-drive, middle-ear implanted hearing devices are a new type of hearing aid. Direct drive MEI systems employ mechanical vibrations sent directly to the ossicular chain, leaving the ear canal entirely open, rather of transmitting acoustic energy into the external auditory canal (as with standard hearing aid systems). The capacity to deliver enhanced sound quality to hearing challenged people is one of the primary advantages of direct drive devices.

In the chosen ear, word recognition should be at least 50% accurate when measured with headphones. Clinical history, tympanometry, and observation should reveal normal middle ear function. Realistic expectations should be discussed with the patient. Patients who are looking for direct drive middle ear hearing aids are frequently dissatisfied with the sound quality of their own voices. Despite numerous visits to their hearing healthcare expert, these people are unable to create speech sounds that sound "natural" or "pleasant," and they are unable to overcome this barrier.

On August 31, 2000, the Food and Drug Administration (FDA) gave the first certification for a direct drive MEI system. Clinical tests have demonstrated that the Vibrant Soundbridge is both safe and effective. A study of 81 individuals as part of the FDA clearance procedure found that the participants could hear just as well with the device as they did with standard hearing aids (FDA, 2000).

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Hair Cells Protection Against Hearing Loss Caused by Ototoxic Drugs

By Elizabeth Mills*

INTRODUCTION

Hearing loss is the most common kind of sensory impairment in the globe. Hearing loss of higher than 20 decibels is the second most prevalent disability, after anaemia, according to worldwide estimates, impacting 1.33 billion people in 2015. According to the World Health Organization (WHO), 466 million people (6.1 percent of the world population) suffer from debilitating hearing loss, which is defined as a hearing loss of more than 40 decibels (dB) in the better-hearing ear in adults and children, respectively. Furthermore, 93 percent of these individuals are adults, with the remaining 7% being minors. Furthermore, by 2050, it is expected that the number of persons with hearing loss would have increased to nearly 900 million. Although hearing loss is not a life-threatening condition, it can reduce a person's quality of life and place a major burden on families and society. Children with hearing loss in low- and middle-income areas have severe developmental delays in language acquisition as well as a lack of schooling.

Hearing loss can be caused by ear infections, noise, and chemical exposure, in addition to congenital causes. Notably, medication ototoxicity is one of the leading causes of hearing loss that may be avoided. An increasing amount of data suggests that ototoxic medicines primarily damage hair cells in the organ of Corti, which are surrounded by supporting cells. Hair cells in the cochlea are critical for translating mechanical sound waves into neural impulses for hearing, and because hair cells in adult mammals are terminally differentiated, they have little capacity to regenerate if injured or destroyed.

After systemic or intratympanic injection, ototoxic medicines can be delivered from the strial vessels or diffuse through the round window into the cochlear tissues. Different medications can harm many cells and tissues in the inner ear, including hair cells, supporting cells, spiral ganglion cells, and the auditory nerve, although hair cell destruction is the major consequence of ototoxicity. As a result, much study has been focused on the causes of hair cell loss induced by ototoxic medications, as well as potential treatment methods. For example, an older study established the time of ototoxic drug sensitivity, whereas a more current study found that overexpression of the X-linked inhibitor of apoptosis protein gene can prevent hair cell loss during this sensitive phase. We think that a better knowledge of ototoxicity will lead to new ideas for drug-induced hearing loss prevention and therapy.

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Cranial Nerve Stimulation's Central Effects

By Elizabeth Mills*

INTRODUCTION

Although cranial nerve stimulation is frequently used to treat the peripheral component of a nerve, many cranial nerve stimulators are known to have significant effects on the central nervous system (CNS) and have been studied especially for their central effects. Vagal nerve stimulation (VNS) for epilepsy therapy and cochlear nerve stimulation for hearing loss are two well-known instances. There are other reasons for cranial nerve stimulation, including the central effects of the stimulation, and commercially accessible devices. The potential to extend indications is being intensively studied due to well-known central effects. Despite the need to broaden indications, the mechanisms of action remain unknown. The existence of complex brain networks and linkages makes understanding the specific mechanism of these effects difficult. The notion that peripheral stimulation of cranial nerves causes central alterations that play a part in the underlying effects of these stimulators is introduced in this article.

Central Effects of Vagus Nerve Stimulation

At first look, it's difficult to see why a VNS, a peripheral nerve stimulator, would be used to treat a central condition like epilepsy, depression, or migraine headache. Nonetheless, there is strong evidence that the VNS is a valuable tool in these situations. It was first licenced for epilepsy, but as the benefits became clearer and the notion of CNS impact evolved, it was broadened to cover depression and migraine. With stimulation of the vagus nerve, a number of central changes occur, and quantifying the particular effect of each of these changes is challenging. Nonetheless, each of these effects is expected to play a role in the mechanism of VNS benefit in each situation.

The effects of VNS on norepinephrine and serotonin levels via the locus coeruleus and raphe nuclei have been proposed as the mechanism by which VNS affects mood in the context of depression. With VNS usage, brain imaging investigations using fMRI and PET have revealed different patterns of alterations in activation and blood flow. The bilateral orbito-frontal and parietooccipital cortex, the left temporal cortex, the hypothalamus, and the left amygdala, for example, were all activated in an fMRI research. At 3 months and 12 months after starting VNS therapy for depression, a longitudinal study looked at fludeoxylglucose-18 (FDG) avidity with PET scanning and found decreased right-sided dorsolateral prefrontal and cingulate cortical activity, followed by increased ventral tegmental area (VTA) activity. Because the VTA is the major brainstem location for dopamine, this might involve changes in dopamine levels in the mechanism of VNS for depression..

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Promulgate Your Proficiency in Scope of Hearing

By Elizabeth Mills*

INTRODUCTION

It is of immense delectation to share with our readers/authors/editors/reviewers that Journal of Canadian Hearing Report had successfully disseminated 15 Volumes worldwide through the journal platform. Currently, we are on the way to promulgate more updates in the field of Hearing aids and hearing disorders. We would like to acknowledge the contributors of our journal for their time-honoured support and cooperation in bringing and publishing the issues on journal website within time. With reference to the previous submissions received in the journal, we request the eminent authors to come up with their valuable submissions based on their recent on-going studies related to the Cochlear implant, presbycusis, hearing loss and other such topics that cover the journal scope.

ARTICLE TYPES

Article Type	**Word Count**
Research Article	1500-6000
Review Article	2000-7500
Original Article	2000-3000
Case/Brief Report	1000-1800
Research Poster	Informative & Captivating
Editorial Note	500-800
Commentary	500-750
Short Communication	900-1200
Perspective/Opinion/ Suggestions	900-1000
Letter to Editor	500-750

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SURVEY & FEEDBACK

Canadian Hearing Report is a growing journal and the journal team is regularly working to upgrade the journal and make it more captivating platform. To achieve the goals the journal circulates survey forms at planned time intervals to take suggestions/feedbacks from the audience of the journal and accordingly the work directions goes on.

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