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Brain pulsatility and pulsatile tinnitus: clinical types

The purpose of this commentary is to share our evolving clinical experience with the tympanic membrane displacement (TMD) analyzer which has focused on brain pulsatility in nonpulsatile, predominantly central-type severe disabling subjective idiopathic tinnitus (SIT) patients resistant to attempts for tinnitus relief with instrumentation or medication.

One outcome of this evolving TMD experience has been the establishment of a classification system for pulsatile tinnitus and its clinical translation for diagnosis and treatment.

A new discipline, brain pulsatility, has been emerging in the last 50-60 years, the principles of which are based on physical principles of matter, volume, and pressure relationships in a confined area (i.e., brain) and find clinical translation to the ear and the discipline of tinnitology - in particular, pulsatile tinnitus. One clinical development for brain disease has been the application of advances in technology for the extracranial identification of alterations in intracranial pressure (ICP) as a marker of brain disease and function. Among these technological advances are transcranial Doppler ultrasonography, phase contrast magnetic resonance imaging (MRI) of brain, transorbital ultrasonography, and the tympanic membrane displacement test[1,2].

A systems analysis of the data (e.g., ICP), recognizing the multifactorial aspects of brain pulsatility as a reflection of a pulsatile system in brain, has added a new dimension to an analysis based predominantly on the raw data.

Principles of brain pulsatility continue to evolve, with concepts that are recommended to be identified for their clinical significance for inner ear function, dysfunction, and ear complaints, including pulsatile tinnitus which include: intracranial pulse pressure amplitude (IPPA), intracranial relationships of brain volume, pressure, and compartmentalization, cerebral compliance, cerebral flow, cerebral perfusion pressure, cerebral resistance (arterial, venous, cerebrospinal fluid), cerebral blood flow autoregulation (CBA), and idiopathic intracranial hypertension (IIH).

Since 2005, our team has successfully identified clinically normal and abnormal intracranial pressures with the recording of spontaneous IPPA waveforms transmitted to the inner ear and tympanic membrane with a probe placed into the external ear canal, a procedure known as the *tympanic membrane displacement test* (TMDT)Marchbanks[1,2]. The hypothesis for the TMDT is supported in the literature by animal and human studies ongoing since 1980. The raw data analysis of the IPPA wave in patients with nonpulsatile, predominantly central-type severe disabling SIT resistant to attempts for tinnitus relief with instrumentation or medication are published in this issue of the *International Tinnitus Journal* (see the article “The Tympanic Membrane Displacement Test and Tinnitus”).

In our experience, the extensive otologic, neurotologic, and neuroradiolgical workup for pulsatile tinnitus is frequently negative, and the etiology of the pulsation is a vascular hypothesis based predominantly on the clinical history and or physical examination. The establishment of an accurate diagnosis and treatment of pulsatile tinnitus has a satisfactory outcome to the patient and physician when a pathological process is identified in an extracranial or intracranial major blood vessel, artery, or vein or when the pathology is a dural arteriovenous fistula for which appropriate surgical treatment or instrumentation may result in tinnitus relief. In addition, a significant advance for the diagnosis and treatment of a pulsatile tinnitus has been identification of IIH with associated neurotologic symptoms, including sensorineural hearing loss, dizziness, and aural fullness (i.e., ear blockage)[3].

In our TMD report of nonpulsatile, predominantly central-type severe disabling SIT patients resistant to attempts for tinnitus relief with instrumentation or medication, who were preselected based on the clinical impression of the presence of IIH, spontaneous abnormal IPPA consistent with an elevated ICP and intracranial hypertension were identified in 10 of 12 patients. Our ongoing tinnitus experience since 1979 has focused on nonpulsatile SIT. The TMD data are teaching us (1) that analogous anatomic and physiologic relationships, in general, appear clinically to exist between ear and brain and underlie some clinical complaints of the inner ear, including hearing loss, tinnitus, vertigo, and ear blockage; (2) to review our knowledge and understanding of endolymphatic hydrops and cochleovestibular data consistent with the diagnosis of a secondary endolymphatic hydrops; and (3) to classify different clinical types of pulsatile tinnitus for improvement of an accuracy of the tinnitus diagnosis and efficacy of treatment. Brain pulsatility information may provide a marker of brain disease and improve the diagnosis and treatment of a particular pulsatile and or nonpulsatile tinnitus.

To date, the classification of pulsatile tinnitus includes the following different clinical types of pulsatile tinnitus which have been identified:

1. Pulsation limited to synchrony with cardiac heartbeat and radial pulse; no associated tone or noise
   - Carotid artery - dehiscence of the bony cover of the carotid artery in the hypotympanum; location ear with dehiscence; tinnitus quality a low-frequency noise; constant duration.
   - Internal jugular vein - tinnitus quality a low-frequency hum; constant duration; position influence; location ear and head elimination with digital palpation and pressure.
   - Arteriovenous dural fistula - tinnitus quality a low-frequency hum.
   - Communicating vessels head and neck - tinnitus quality a low frequency; duration intermittent or constant.
2. Pulsation synchrony with cardiac heartbeat and radial pulse; additional associated tone or noise (i.e., a subjective tinnitus)
   - Microangiopathy ear and/or brain - tinnitus quality a low-frequency noise; constant duration.
   - Brain and inner ear - localized inflammation hypothesized; tinnitus quality a middle- to high-frequency noise; constant duration; frequent clinical history of hypertension.
3. Pulsation with no synchrony cardiac heartbeat and radial pulse; with/without additional associated tone or noise (i.e., a subjective tinnitus)
   - Myoclonus middle ear or pharynx - tinnitus quality-staccato, with/without low-frequency noise; constant/intermittent duration.
4. Subclinical pulsation - preceding three types of reported subjective pulsatile tinnitus. Bruit positive identification with auscultation ear and or head.

Why some patients with hypertension and tinnitus report pulsation and others, as in our report, do not is a question that awaits results of investigation. What is evolving in the discipline of brain pulsatility is the introduction of technological advances, such as the TMDT, which provide objective recordings of physiologic markers that can identify ear as well as brain disease, thereby providing a basis for accurate pulsatile and nonpulsatile tinnitus diagnosis and an increased efficacy of tinnitus treatment.

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REFERENCES

1. Marchbanks RJ. A study of tympanic membrane displacement. Doctoral thesis, Brunel University, Uxbridge, Middlesex, UK (Br. Lib. Ref D34215/80).1980.
2. Marchbanks RJ, Reid A. Cochlear and cerebrospinal fluid pressure: Their interrelationships and control mechanisms. Br J Audiol. 1990;24:179-87.
3. Sismanis A, Hughes GB, Abedi E. Otologic symptoms and findings of the pseudotumor cerebri syndrome: A preliminary report. Otolaryngol Head Neck Surg. 1985;93:398-402.