Nociception in Pain Hyperacusis

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Research into the origins and mechanisms of hyperacusis has been elusive, especially for those associated with pain. In fact, the total number of articles with the search word "hyperacusis" on pubmed.gov included 1,002 articles since 1948, but for "pain hyperacusis," only 113 articles since 1976. In addition, until recently, the various perceptions of hyperacusis have not been well defined.

In 2016, Tyler and his colleagues studied the many different reactions and characteristics reported by patients with hyperacusis.[1] They concluded the features could be filtered into four different types: loudness, annoyance, fear, and pain. A patient may have more than one type, although loudness or pain hyperacusis appears the most offensive. Recognizing the differences between these groups is diagnostically significant and clinically valuable because of expanding choices for treatment and developing rehabilitation modes.

Fortunately, interest is gaining, and significant findings are starting to answer the most critical questions: What are the mechanisms behind pain hyperacusis, and are there pain receptors associated with the ear? A straightforward answer appears to lie in the feedback and modulating system of nociception, which is integral to the mammalian pain response. Nociceptive pain is one of the four types of pain; acute, chronic, and neuropathic pain are others. The cochlear organ of Corti, particularly the outer hair cells, can be damaged by noxious noise, but this does not innervate nociceptors because this type of pain loop is not found in the inner ear. Other forms of nociception appear to be at play, and until recently, research has been unable to identify potential mechanisms.

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According to Armstrong and Herr, "nociception provides a means of neural feedback that allows the central nervous system [CNS] to detect and avoid noxious and potentially damaging stimuli in both active and passive settings."[2] Noxious stimuli include mechanical force, chemical exposure, and extreme hot and cold. In addition, nociceptive pain occurs because of tissue damage resulting from physical or chemical insults, such as trauma, surgery, or chemical burns.

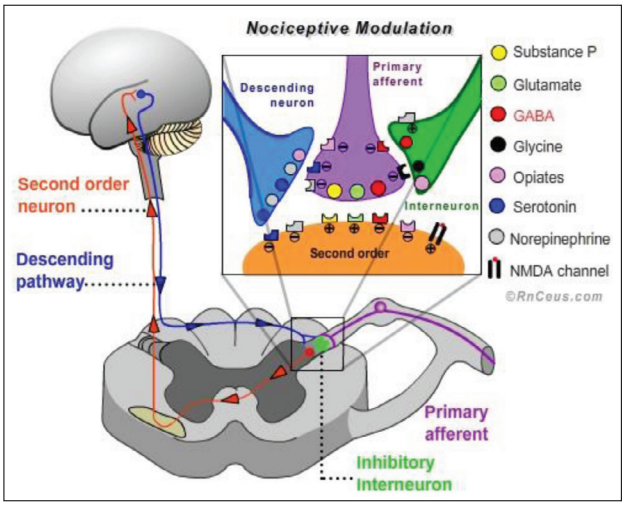
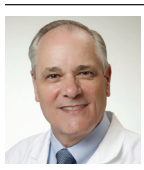


Figure 1. Nociceptive Modulation (with permission from RnCeus Interactive, LLC; https://www.rnceus.com/ages/nociceptive.htm)[4]

According to psychologist G.W. Turmen, pain presents a "complex constellation of unpleasant sensory, emotional, and cognitive experiences provoked by real or perceived tissue damage and manifested by certain autonomic, psychological, and behavioral reactions."[3] To the tinnitus and hyperacusis specialist, this definition also describes patients with pain hyperacusis. Unfortunately, in the past, pain hyperacusis has been homogenized with other forms of sound sensitivity, resulting in treatment recommendations for sound therapy, cognitive behavioral therapy, and speculative medical interdictions. Although these interventions are helpful, more attention to the pain component and comorbid headaches or migraines needs to transcend current dogma. Like tinnitus, hyperacusis is not always associated with clinical hearing loss, and to date, no one has been cured of pain hyperacusis. Further investigation into the nociception mechanism may reveal other opportunities for the future.

THE NOCICEPTION MECHANISM

Figure 1, Nociception Modulation, provides a basic example of the mechanism (RnCeus Interactive, LLC).[4] The nociception feedback loop starts with information gathered by the sensory terminals, such as temperature, resulting in an action potential. These signals are conducted via afferent fibers to the nociceptor cell body found in the dorsal root ganglia of the spinal nerve. When the receptors detect noxious stimuli that can cause tissue damage or the presence of toxic molecules and inflammation, the perception of pain is evoked.[5] Fibers from the nociceptor transverse over to the inhibitory interneuron at the junction of the first- and second-order neurons. The



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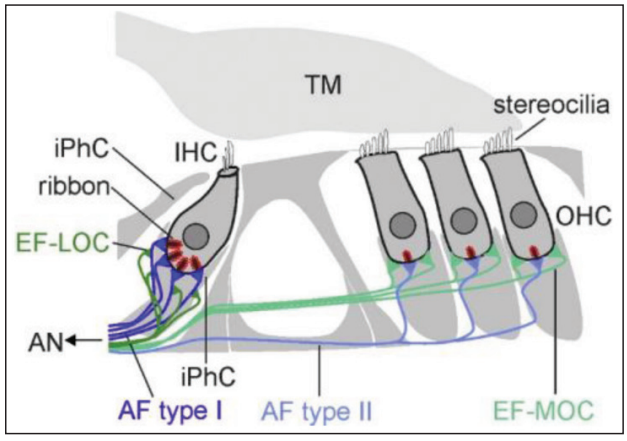

Figure 2. Adult Organ of Corti. Tectorial Membrane (TM), Outer and Inner Hair Cells (OHC, IHC), Afferent Type I (AF - type I), Afferent Type II (AF type II), Efferent Medical Olivary Complex (EF-MOC), Efferent Lateral Olivary Complex (EF-LOC), Auditory Nerve (AN), Inner Phalangeal cells (iPhC).[8]

inhibitory interneurons are responsible for facilitating or inhibiting signals to the second-order neurons that ascend to the cortex.[6] From the cortex, efferent pathways send signals to the descending neurons, and together with the inhibitory interneurons, apply countermeasures in the form of cascading biochemical reactions. These include both excitatory and inhibitory neurotransmitters, pro-inflammatory molecules, or the release of ligands to reduce the perception of pain, such as opiates, serotonin, and norepinephrine. In doing so, the sensation of pain is realized and managed by the CNS. Recent research has shown a modulation method in the auditory system, explaining how the pain response to noxious sounds may be due to inappropriate inhibition.

AUDITORY NOCICEPTION

The neurophysiology of loudness and pain hyperacusis have been under considerable investigation in recent years. We know inner hair cells connect to the brain via myelinated type I fibers, and outer hair cells are connected to the brain by unmyelinated type II fibers, representing only 5% of all cell fibers. According to Charles Liberman,[7] the characteristics of the type II fibers have been challenging to assess and are not well understood. They do not directly respond to sound but resemble somatosensory neurons reporting unrepairable cochlear damage. Possibly, the pain response occurs because of an absence of normal control over the modulation system of which type II neurons act as the ear's nociceptor. According to Paul Fuchs, type II cochlear afferents represent a novel sensation coined as "auditory nociception."[7] Based on research, the medial olivocochlear (MOC) efferent fibers are involved, as seen in Figure 2.[8]

The MOC neurons arise from the superior olivary complex in the brainstem. They are the final stage of descending control over the peripheral auditory system through axonal projections into the cochlea.[9] Although the MOC is not well understood, it adjusts cochlear gain and frequency tuning and helps protect the ear against acoustic trauma. Dr. Liberman's research has also shown type II fibers can innervate Henson's cells in the apical turn of the cochlea, which lies adjacent to the outer hair cells. These cells collapse and recover when overstimulated by noise. This innervation further propagates the theory that a somatic-like system exists within the auditory system. Dr. Liberman suggests type I pathways may account for loudness discomfort, while type II fibers may underlie pain hyperacusis.

LOUDNESS VERSUS PAIN HYPERACUSIS

The difference in pathways may explain the different clinical profiles between loudness and pain hyperacusis patients. Williams, Woynaroski, and Suzman compared subjects with loudness or pain hyperacusis in a study using the multinational patient registry from Stanford University.[10] The findings reveal no significant group differences, although those with "… pain hyperacusis presented with a more severe clinical phenotype, reporting a higher frequency of temporary symptom exacerbations (i.e., setbacks), less perceive symptom improvement over time, more severe comorbid headache disorders, and reduced benefits from sound therapy."

For example, patients with pain hyperacusis exposed to high frequencies and higher peak values from clanging dishes or metal pots and pans will have instant ear pain and headaches/migraines, with a pain cycle lasting for hours to days.[11] This type of pain hyperacusis is life altering and requires considerable courage for those afflicted.

In some cases, pain hyperacusis occurs in only one ear due to inner-ear damage from an occurrence such as acoustic shock, viral, or vascular insults.[12] To the clinician, this suggests a different mode of counseling and therapy to address individual differences. For example, patients with tinnitus and pain hyperacusis without hearing loss do not perform well with occlusion. In these cases, an open fit system is indicated. How do we protect the ear and help patients return to a more satisfying lifestyle with plugged ears and their voice in their head? When there is hearing loss in the low frequencies, this is easier to accomplish with electronics. Other methods and devices are needed.

Pain hyperacusis is a debilitating condition that requires patients to become vigilant over their environments, apply hearing protection when needed, and help others, especially family members, understand their disorder. Some resort to isolation and the chronic use of hearing protection, neither of which are satisfactory treatment when considering the quality of life changes that occur.

Additionally, many affected with pain hyperacusis have some degree of psychopathology, as socialization is lost, and self-esteem and worthiness become potential targets. As soon as possible, audiologists should provide diagnostics to qualify the causation, type of hyperacusis, and comorbidities, followed by counseling on environmental triggers and devices, etc. Taking actions to control exacerbations and setbacks is crucial to future success.[11] Research investigations need to develop objective protocols to distinguish pain versus loudness hyperacusis, develop novel technologies for protection without isolation, and provide better and less invasive medical interventions.

References for this article can be found at http://bit.ly/HJcurrent.