Involuntary Volume

Research is revealing more about our internal volume controls. Our emotional brain center, as well as exposure to artificial sounds, can influence sensitivity to sound.

By Kathi Mestayer

Hyperacusis and recruitment have a lot in common, at least on the surface. They both cause a perceived increase in volume triggered by an actual, external sound.

But that is where the resemblance ends. Recruitment is a sudden, rapid increase in volume, which can result in a startle reflex. It is always accompanied by sensorineural hearing loss. Hyperacusis, on the other hand, is a significantly more acute sensitivity to sound—often to the point of being physically uncomfortable and stressful.

“Hyperacusis is a condition that can have a variety of causes and can involve the brain’s central processing functions,” says Daniel Coelho, M.D., a co-director of the Virginia Commonwealth University Medical Center Cochlear Implant Center in Richmond, Va.

It is much more complex than recruitment, and more closely related to tinnitus, he says. In fact, tinnitus (a ringing in the ears that does not have an external sound source) and hyperacusis often occur together.

“Recruitment, in contrast, is a much better understood physiologic condition,” Coelho says. “It results from damage to the stereocilia in the cochlea, and as such, is always associated with sensorineural hearing loss.”

With recruitment, the death of stereocilia responsible for hearing loss results in neighboring cilia being “recruited” by the auditory cortex in an attempt to turn up the volume. It is often described as an abnormally rapid increase in loudness. Recruitment generally does not result in uncomfortable levels of sound, according to Coelho, but can narrow the comfort zone of volume for
people who have hearing loss.

Hyperacusis, by contrast, can occur along with a number of conditions, such as acute acoustic trauma, migraines, Bell’s palsy, temporomandibular (TMJ) syndrome, Ménière’s disease, tinnitus, and hearing loss. It is thought to involve the auditory central processing areas of the brain, and not just the cochlea. Recent experiments suggest that the limbic system, specifically the amygdala, may play a part.

Where it can become confusing is when trying to make fine distinctions between cause and effect. It’s even possible for one person to have hearing loss, recruitment, hyperacusis, and tinnitus. “In fact, the relationships between them are complicated and still incompletely understood,” Coelho says. “Recruitment from hearing loss can cause a perception of hyperacusis, though patients without hearing loss can also have hyperacusis.”

### HYPERACUSIS AND THE BRAIN

One of the leading researchers in this field is Richard Salvi, Ph.D., of the State University of New York at Buffalo’s Center for Hearing and Deafness, in Buffalo, N.Y. Salvi and his colleagues have been exploring the role of different brain regions in hyperacusis.

“So many of the big questions are: How does hyperacusis get going? Where is the brain in it happening?” Salvi says. “Our experiments explore what regions of the brain, other than the auditory pathway, might be involved.”

Salvi and his team have conducted experiments using positron emission tomography (PET) to image brain activity in humans. Their PET imaging studies revealed hyperactivity in portions of the limbic system associated with emotion and memory.

In more recent electrophysiological studies in rats, whose auditory systems are similar to humans, high doses of sodium salicylate (the active ingredient in aspirin) were used to temporarily induce a hyper-excited state, hearing loss, and tinnitus.

Electrophysiological recordings revealed sound-evoked hyperactivity in neurons located in the auditory cortex, where sounds are interpreted, and in the amygdala, the part of the limbic system that creates associations between certain sounds and emotions like fear, stress, and anxiety.

“It’s possible that the amygdala amplifies the emotional response to sound, creating more ‘volume’ in the process. That could be a factor in hyperacusis and tinnitus,” Salvi says. “Although the connection is still theoretical, this is the first experimental evidence of physiological activity in the amygdala under these conditions.”

Salvi’s research continues on the role of neurotransmitters in the hyperactive auditory brain. “You can think of the excitatory neurotransmitters as the accelerator pedal of a car, and the inhibitory neurotransmitters would be the brakes,” he says. “There are basically two possibilities: Excitatory neurotransmission (the accelerator) could be enhanced, or inhibitory neurotransmission (the brakes) could be reduced.”

He adds, “Either way, the sound-evoked neural activity would be amplified in the central nervous system, presumably causing the sounds to seem much louder than normal (i.e., hyperacusis). At this point, we’re focusing on the second scenario, reduced inhibition.”

Evidence from Salvi’s group and others indicates that inhibition in the brain is reduced (“less braking”), resulting in a more robust response to sound.

### TRIALS AND TREATMENTS

As the mechanisms and brain areas involved in hyperacusis emerge, research is also progressing on another front: How can we treat hyperacusis?

For several years Craig Formby, Ph.D., a University of Alabama distinguished graduate research professor in the Department of Communicative Disorders in Tuscaloosa, has been researching the nature of hyperacusis and the potential mitigation of its effects. In 2002 and 2003, Formby and colleagues at the University of Maryland Tinnitus and Hyperacusis Center conducted a small trial in which 16 people with normal hearing were fitted with either sound generators or earplugs for two weeks. The sound generators emit a soft sound, like that heard when cupping a seashell to the ear.

The hearing of test subjects was measured with audiograms for their lower threshold (the quietest signal that they could hear), and their perceptions of loudness were measured using a seven-category Contour Test of Loudness (CTL) scale. The CTL categories are: very soft, soft, comfortably soft, comfortable, comfortably loud, loud but OK, and uncomfortably loud.

“Our findings were intriguing,” Formby says. “The people with earplugs became more sensitive to the loudness of sounds, while those with sound generators became less sensitive. The degree of difference, for both groups, was about one category—plus or minus—on the CTL scale, which is equal to about 7 to 8 decibels.”

There was no change in either group’s lower loudness threshold. Formby says the experiment showed that
“It’s possible that the amygdala amplifies the emotional response to sound, creating more ‘volume’ in the process. That could be a factor in hyperacusis and tinnitus.”

Loudness judgments are “plastic,” or changeable, and suggested that it might be altered by either soft sound exposure or partial sound deprivation (i.e., silence).

“At that point, I was curious whether the practice of fitting patients with sound generators to enhance sound tolerance, plus the addition of specialized counseling, might work for hearing-impaired people with hyperacusis,” Formby says. Since people who have both hearing loss and hyperacusis already experience uncomfortable sound levels, sometimes they are not willing to use hearing aids to amplify sound.

With support from the National Institute for Deafness and Other Communication Disorders, part of the National Institutes of Health (NIH), Formby’s team attempted to address this conundrum. The specialized trial used four groups of nine people each, all of whom had both mild to moderate hearing loss and hyperacusis at a level that made them unwilling to wear hearing aids.

Participants in two groups received a sound-generating device, and the other two groups received a placebo device. Two groups received counseling about the nature and mechanisms of hearing, hearing loss, and hyperacusis, as well as the objectives of the study. The other two groups did not receive counseling.

Each of the four groups had a different combination of sound-generator/placebo, and counseling/no counseling. Audiologist and speech pathologist Susan Gold, formerly with the University of Maryland Medical Center, provided the counseling component, which was specifically targeted to hyperacusis and hearing loss. (Gold and Formby are collaborating on the NIH-funded Tinnitus Retraining Therapy Trial; see “A Landmark Trial,” in the Summer 2012 issue, at www.hearinghealthmag.com.)

After six months, changes in the study subjects’ sound tolerance were measured. The group that had received both sound-generating devices plus counseling had, on average, improved sound tolerance at a statistically significant level of about one loudness category (or 7 to 8 decibels). “After the trial, people whose sound tolerance had improved to normal levels no longer needed the sound generators,” Gold says. “So we fit them with hearing aids, which they could then wear comfortably.”

A New Literature Review

A new effort, with the joint support of Hearing Health Foundation and Hyperacusis Research, will be to conduct a comprehensive literature review of the research on exacerbated responses to sound (hyperacusis).

This will involve three broad components: loudness-hyperacusis (moderately intense sounds are perceived as very loud); annoyance-hyperacusis (sounds are unusually annoying); and fear-hyperacusis (unusual fear of sounds, also known as phonophobia).

The project’s objectives include providing definitions for those conditions, comprehensive background material with examples, and proposed mechanisms or demonstrated causes to the extent possible. The principal investigator is Richard S. Tyler, Ph.D., a professor in the Departments of Communication Sciences and Disorders and of Otolaryngology, at the University of Iowa in Iowa City. This work is especially important since it will provide a reference point for ensuring that future research efforts are productive, focused, and do not cover ground others have already investigated.

The understanding of the basic causes and connections between hearing loss, tinnitus, and hyperacusis is proceeding on a parallel track with advances in treatment. As researchers chip away at this complex set of problems, it appears that the solutions quickly go beyond the mechanical workings of the ear—and require that both sensory and broader cognitive brain functions are understood and engaged.

“Hearing loss, hyperacusis, and tinnitus are every bit as complicated as pain, memory, and other neurological processes,” Salvi says. By employing animal models, testing on humans, and a comprehensive review and definition of terms, the fact that “we hear with our brains, not with our ears” becomes ever more true with every advance in this multidisciplinary field.

A staff writer, Kathi Mestayer is on the advisory committee of the Greater Richmond, Va., Hearing Loss Association, and serves on the Advisory Council for Virginia Relay, which provides communications access for the deaf, hearing impaired, deaf and blind, and speech disabled.
TROUBLE-FREE.
WORRY-FREE.
BATTERY-FREE.

Discover the most advanced rechargeable hearing instrument line-up. Worrying about maintenance and batteries are a thing of the past.

NEW SLIM
Behind the Ear
Exceptional versatility, reliability and range in a sophisticated, slim design.

X-MINI
Receiver In Canal
Easy-to-use, exquisite design with a smooth response.

CUSTOM
In The Ear
Cosmetically appealing, ultra discrete. It's the simplest, most convenient custom hearing instrument on the market.

Award-winning Charging System
Simply drop into the docking station for up to 20 hours per charge. You'll never think about batteries again.

AQ
FAMILY OF RECHARGEABLES. ONE TO FIT ANY LIFESTYLE.

Now there's even more to love about HANSATON's award-winning rechargeable lineup. More features. More benefits. And now, more styles. It's time you tried the industry's best rechargeable hearing system with 20 hours per charge and a 5-year battery guarantee.

For more information about the AQ rechargeable visit www.hansaton-usa.com or contact your hearing healthcare professional.