

EXPLODING HEAD SYNDROME *by Regina Patrick RPSGT*



Exploding head syndrome (EHS) despite its somewhat humorous-sounding name does not leave its sufferers laughing but terrified. In EHS, a person suddenly hears a loud explosive noise just as they are going to sleep or awakening from sleep. The source of the explosive noise seems to be located just behind one's head or within one's head – hence, exploding head. The person may get out of bed to investigate the noise only to find out it has no source. This in turn may cause him to believe this may be a sign of an impending stroke, brain tumor, or other neurological problem. Scientists have been unable to explain why a person hears an explosive noise but have several theories.

The phenomenon of loud explosive noise occurring in association with sleep has been described in the medical literature since at least the late 19th century. American physician Silas Weir Mitchell in an 1890 article on sleep disorders discussed what he called "sensory shocks" which had been reported by several patients. His patients described feeling electric sensations followed by a violent explosion and a sensation of being struck. In 1920, British physician Robert Armstrong-Jones described a condition which he called "snapping of the brain." Patients who suffered "snapping of the brain" would complain of hearing a sudden loud or crashing noise when going to sleep or when awakening from sleep. Examinations by ear specialists could find nothing audiotologically wrong with the patients. In 1988, British physician J. M. S. Pearce first used the term

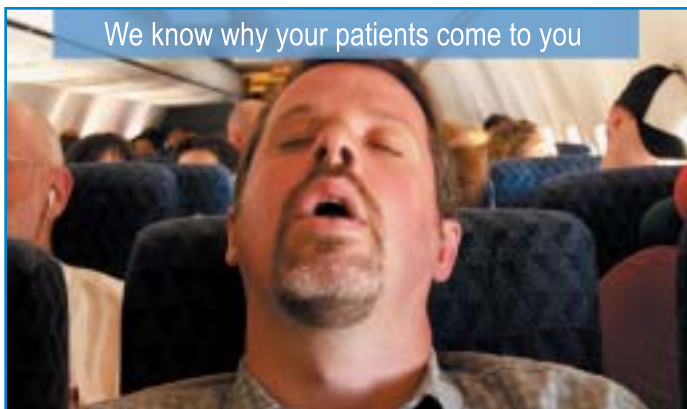
"exploding head syndrome" in the medical journal *The Lancet*. He described the cases of ten patients who complained of hearing a sudden loud noise upon going to sleep or just awakening. Three of the patients described the noise as "a frightening sense of explosion," "a thunderclap," and "something exploding, creating a loud bang in my head." Recent names for the phenomenon are sensory sleep starts and shocks.

Some scientists suspect that dysfunctional activity of the middle ear or inner ear structures may be the source of the sudden loud noise. Another possibility may lie in dysfunction of brainstem structures or pathways involved in hearing.

In the middle ear, explosive noise may come about due to altered activity of two middle ear muscles – the tensor tympani and stapedius muscle. These muscles attach to the malleus and stapes, respectively. (Due to their shapes, these bones are more commonly called the "hammer" and "stirrup." They are two of three ear bones that relay sound vibrations from the eardrum to the cochlea.) When activated by loud sounds, the tensor tympani muscle pulls on the malleus. Because the malleus is attached to the eardrum, the eardrum is pulled inward and becomes more tense. This action limits the eardrum's ability to vibrate thereby dampening the effect of loud noise on middle ear and inner ear structures. Also in response to loud sounds, the stapedius muscle pulls the foot of the stirrup away from the cochlea. This prevents the stirrup from pressing against the cochlea excessively which in turn protects nerve receptors in the cochlea from being overly stimulated and becoming damaged. In some people, the stapedius and tensor tympani muscles can suddenly spasm. When this occurs, a person may experience a roaring noise, a clicking sound, or a pop.

Another area in the middle ear which may be a source of a loud explosive noise is the Eustachian tube. The Eustachian tube is a narrow tube which extends from the lower portion of the middle ear to the throat. It allows air to quickly enter or exit the middle ear to accommodate pressure changes that occur during swallowing, talking, holding one's breath, etc. The openness (i.e., patency) of the Eustachian tube is affected by the activity of palatal muscles. For example, the tensor veli palatini muscle tenses the soft palate and opens the Eustachian tube during swallowing and the levator veli palatini muscle raises the soft palate while narrowing the opening of the tube. These muscles can spasm suddenly forcing the tube to remain open – a condition called patent Eustachian tube. The sudden opening can be experienced as popping noise and changes in hearing can occur.

In the inner ear (cochlea, semicircular canals, and vestibule), alterations in fluidic flow can result in the perception of a loud noise. The inner ear is filled with a fluid called perilymph. Nestled within the inner ear structures is a series of ducts and sacs which are filled with a different fluid called endolymph. Endolymph and perilymph normally do not mix. However, disease, trauma, or surgery can result in an intermixing of the two fluids or result in perilymph leaking out of the inner ear. For example, in Meniere's disease, breaches occur in the membrane (Reissner's membrane) which would normally act as a barrier between endolymph and perilymph. The change in chemical composition of endolymph



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resulting from the intermixing in turn damages cochlear receptors and results in impaired hearing. In a perilymph fistula, perilymph leaks out of the cochlea. Typically, the fluid leaks out through the oval or round window. This fluid loss results in hearing changes. Typical hearing changes resulting from intermixing of perilymph and endolymph or from leaking of perilymph out of the inner ear are hearing loss, tinnitus, or sense of fullness in ear. However, some people have complained of hearing sounds as excessively loud (i.e., hyperacusis), a roaring noise, or a popping sound.

In the brainstem, areas that may be involved in the perception of a loud noise in EHS are the reticular formation, the cochlear nuclei, and the vestibular nuclei. The reticular formation is located centrally throughout the midbrain, pons, and medulla. The cochlear and vestibular nuclei are located in the junction between the pons and medulla. The cochlear and vestibular nuclei have connections with neurons of the reticular formation. It may be that impaired activation of the reticular formation during sleep could be playing a role in the perception of a loud bang in EHS. In 2006, researcher Randolph W. Evans reported the case of a woman who would hear a sudden loud noise while lying down relaxed but not yet asleep. Moments later, she would be unable to move although awake and aware. This paralysis lasted for about six seconds and was followed by a throbbing migraine headache which could last up to 12 hours. Evans believes that areas activated in the reticular formation that resulted in her atonia may have also played a role in her hearing a loud noise.

Some studies suggest that altered sound perception (e.g., tinnitus, hyperacusis) can occur as a result of dysfunction in efferent auditory pathways such as the olivocochlear tract (which relays signals from the olivary body to the cochlea). Although no studies yet exist that specifically focus on efferent auditory pathway function in EHS sufferers, some research supports the possibility that these pathways may be contributing to the perception of a loud explosive noise in EHS.

The efferent auditory system brings signals from the auditory cortex (located on the temporal lobe) through the olivary body (located in front of the medulla) and finally to the cochlea. Scientists believe efferent auditory system activation stimulates the outer hairs (a type of nerve receptor) of the cochlea to generate otoacoustic emissions (OAEs). OAEs manifest soon after the cessation of an auditory stimulus. OAEs can range from 10 to 30 decibels but they are not normally perceived by a person.

An OAE that is elicited by auditory stimulation is called a transient otoacoustic emission (TOAE). On an audiogram, TOAEs appear as a series of sinusoidal waves of different amplitudes. Amplitude can be affected by cochlear damage (damage to outer hairs) or by a neurological problem such as an acoustic tumor.

Normally, exposing one ear to white noise decreases the amplitude of TOAE waves produced by the opposite ear. The converse occurs in people who have problems in the efferent system such as damage to olivocochlear tract. Israeli researchers Joseph Attias et al.⁸ used this characteristic to investigate the function of the efferent auditory system in subjects with brain injury who also had problems with hyperacusis (an oversensitivity to sound) or tinnitus (perception of sound which has no external source). They found that indeed TOAE waves had a higher-than-normal amplitude in people with brain damage who also had hearing problems. This suggests that a dysfunction in the efferent auditory system can play a role in hearing problems.

Because sufferers of EHS have described hearing the noise in the twilight stage between sleep and wake, scientists for a long time thought the disorder was a hypnagogic phenomenon. Polysomnography (PSG) suggests it is not.

In a 1991 PSG study, Swedish researchers Charlotte Sachs and Eva Svanborg found that EHS episodes were occurring while a sufferer was fully awake rather than when arousing from or going to sleep. They studied nine EHS subjects by PSG. The electroencephalograph (EEG) portion of the PSG revealed that none of the subjects were asleep at the time they indicated hearing an explosive noise. This meant that the attacks were occurring when

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the subjects were relaxed but fully awake. Sachs and Svanborg surmise that EHS sufferers may believe they are being awakened by a noise since an episode occurs during a time the subject happens to be temporarily awake during a sleep period.

EHS is listed in the International Classification of Sleep Disorders (ICSD) as a parasomnia. Besides hearing a sudden explosive noise in association with sleep, EHS also has these features: it is usually painless; episodes of exploding noise may occur for one night or for several nights at a time then resolve for weeks to months at a time or permanently; it tends to occur during times of high stress; it may be accompanied by flashes of light; a person may be so frightened on suddenly hearing a loud bang that he suffers other aspects of "flight or fight" response such as awakening with labored breathing or in a cold sweat; the disorder is more common in women and the average age of onset is about 58 years; it is not related to nocturnal headache (although some people have reported feeling stabbing pain with an episode) nor is it a seizure disorder. EHS does not always match these criteria. For example, one EHS patient described the sound as a musical twang¹ rather than a loud explosion.

Exploding head syndrome while frightening is a benign condition. No treatment exists although drug therapy (such as antidepressants) may help to reduce episodes in people with frequent attacks. Until more answers are known, sleep workers can best help patients who feel "weird" or frightened by hearing non-existent loud sounds during sleep by reassuring them of the benign nature of the disorder.

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