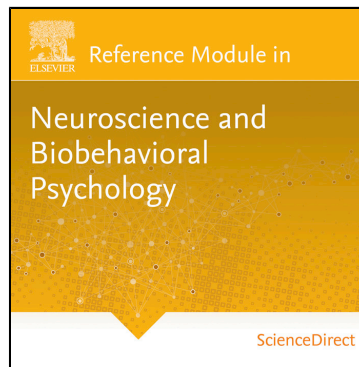


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# Exploding Head Syndrome<sup>☆</sup>

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## History and Nomenclature

Exploding head syndrome (EHS) is characterized by a momentary loud noise that patients usually experience during the early stages of sleep. Patients describe a sudden onset of “an explosion in the head,” enormous roar, bomb-like explosion, or lightening crack that awakens them from sleep. This is usually followed by a feeling of intense fear, terror, and/or palpitations. However, there is no headache or pain associated with the noise.

The syndrome was first named by Pearce in 1988 when he reported on 10 patients. However, Armstrong-Jones provided the first description of the syndrome as “snapping of the brain” in 1920. Weir Mitchell may have previously described the disorder in 1890. Pearce has reported the highest number of patients at 50.

## Demographics and Prevalence

EHS can occur at any age but is more common in patients older than 50 with a median age of 54 years (range 12–84) and a female:male ratio of 3:2. The prevalence is unknown as, anecdotally, patients may not report their symptoms. No large scale prevalence studies have been performed. However, EHS may not be that uncommon as a study of college students found a lifetime prevalence of 18% and 16.6% had recurrent cases with equal numbers of males and females.

Many physicians may not be familiar with the disorder. EHS may not be that rare.

There are numerous descriptions of EHS on the internet. For example, in response to an article on an MSNBC Website blog, there were many comments from readers describing their own EHS including comments such as the following: “Wow, it’s nice to know I’m not crazy!”; “I have had this since I was very young ... never knew it actually had a name or was a real condition. I am a 34 year old female.”; “I never reported it because it generally does not stick around and I’ve explained it away, appropriately it seems as being under stress or having a bad dream.”; and “I have seen two doctors who have never mentioned this. I did not think they believed me, they just ran test after test.”

## Onset, Ontogeny, and Clinical Course

Symptoms can arise from any stage of sleep, but primarily arise during stages 1 and 2. One study of nine patients indicated that symptoms correlated with an alert state or awakening on polysomnographic recordings. Attacks can occasionally occur as patients wake up following arousal and then get back to stage 1 sleep. The frequency is highly variable with a range from two to four attacks followed by prolonged or lifetime remission to seven attacks nightly for several nights each week.

## Etiology, Pathophysiology, and Pathogenesis

The etiology and pathophysiology are unknown although it is hypothesized that there is a delay in areas of the reticular activating formation in “turning off” the motor, sensory, visual, and auditory cortices.

<sup>☆</sup>*Change History:* January 2016. Randolph W Evans made some changes to the text and updated the Further Reading section.

### Associative, Predisposing, and Precipitating Factors

Fear, terror, palpitations, or a forceful heartbeat were reported as occurring after the loud noise in 47 of the 50 patients. Ten percent of patients described an associated flash of light and 6% reported a curious sensation as if they had stopped breathing and had to make a deliberate effort to breathe again—"an uncomfortable gasp." Occasionally, brief myoclonic jerks of the extremities or the entire body may follow. Psychological stress and tiredness may be triggers. Three patients out of 50 reported a positive family history.

EHS may be a migraine aura. Kallweit and colleagues reported a 54-year-old male with attacks of EHS followed by an exacerbation of his chronic migraine after each attack. In 2007, Evans reported a 26-year-old woman with a history of migraine without aura, with multiple episodes of EHS followed by brief sleep paralysis and then one of her typical migraine headaches.

### Complications and Consequences

The syndrome is benign except for anxiety produced by the symptoms and has no long term sequelae.

### Diagnosis

Diagnosis is made by the clinical history. A sleep study does not assist in diagnosis and it is not certain whether the study changes the method of management if the patient is found to have obstructive sleep apnea (see [Treatment](#)). The neurologic exam is normal. Imaging studies are not necessary although some patients may wish to be reassured that they do not have a tumor or an aneurysm.

### Treatment

After explanation and reassurance, most patients do not require medication. For those with frequent or disturbing symptoms, there are anecdotal reports of benefit of treatment with clomipramine, nifedipine, flunarizine, topiramate, amitriptyline, and use of an oral appliance for a patient with obstructive sleep apnea.

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