

Acoustic Shock Injury (ASI)

Guide for Medical Practitioners

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Acoustic shock injury (ASI) is an involuntary trauma reaction, which can occur following exposure to a sudden unexpected loud sound, causing a specific and consistent pattern of neurophysiological and psychological symptoms.

The potential severity and persistence of ASI symptoms have significant medical, clinical and medico-legal implications. With the rapid growth of call centres, general practitioners, ENT specialists, occupational physicians, TMD specialists, neurologists, trauma psychologists/psychiatrists and audiologists are increasingly likely to encounter some or all of the cluster of ASI symptoms in their patients.

Background

Call centre staff using a telephone headset or handset are vulnerable to ASI because of the increased likelihood of exposure, close to their ear(s), of a sudden unexpected loud sound (acoustic incident) randomly transmitted via the telephone line. In the early 1990s, co-inciding with the rapid growth of call centres in Australia, increasing numbers of employees were reporting these symptoms. A similar pattern was being noticed overseas, and the term acoustic shock injury (ASI) was coined to identify this symptom cluster.

More generally, any patients who have developed tinnitus and hyperacusis (abnormal sound intolerance), particularly following exposure to a sudden unexpected loud sound or associated with a highly traumatic experience, may report at least some of these symptoms.

ASI symptoms

The neurophysiological and psychological symptoms of an ASI are different to those occurring with a traditional noise injury and include some or all of the following:

- a shock/trauma reaction. A severe ASI can lead to Post Traumatic Stress Disorder (PTSD)
- pain/blockage/pressure/tympanic fluttering in the ear
- subjective muffled/distorted hearing. ASI generally does not result in a hearing loss, although if present it tends not to follow the typical high frequency pattern of a noise induced hearing injury but affects low and mid frequency sensorineural hearing.
- other sensations including pain/burning/numbness around the ear/jaw/neck
- tinnitus, hyperacusis and phonophobia (abnormal fear of sound)
- mild vertigo and nausea
- headache

Typically, people experiencing an acoustic shock describe it as like being stabbed or electrocuted in the ear. The initial symptoms include a severe startle reaction, often with a head and neck jerk. The symptoms are involuntary, unpleasant, frightening and can be deeply traumatic; they can range from mild to severe; and be of short, temporary duration or persistent. If symptoms persist, a range of emotional reactions including trauma, anxiety and depression can develop.

What causes ASI symptoms?

Middle ear muscle contractions, particularly of the tensor tympani muscle, have been demonstrated to occur as part of the startle reflex. The physiological symptoms of an ASI are considered to be a direct consequence of excessive, involuntary middle ear muscle contractions caused by a strong startle response to an acoustic incident. However, in some cases, ASI symptoms can develop as a result of cumulative exposure to sustained headset use, without a specific acoustic incident being identified, apparently as a result of triggering the established protective function of the tensor tympani muscle.

Tonic tensor tympani syndrome (TTTS) has been proposed as the neurophysiological mechanism causing most of the persistent ASI symptoms. This is an involuntary condition where the centrally mediated reflex threshold for tensor tympani muscle activity becomes reduced as a result of anxiety and trauma, so it is continually and rhythmically contracting and relaxing. This appears to activate a series of physiological reactions in and around the ear, which can include: an abnormal stimulation of the trigeminal nerve innervating the tensor tympani muscle; alterations in ventilation of the middle ear cavity; muscular tightness around the ear potentially extending to the cervical/shoulder girdle and upper limb muscles, causing muscular trigger points which lead to CNS pain pathway sensitisation; and to a lesser extent abnormal stimulation of the nerves innervating the tympanic membrane and ossicular chain.

More generally, these symptoms have been observed in our clinic over many years in tinnitus and hyperacusis clients. A recent study at our clinic examining the incidence of symptoms consistent with TTTS in a sample of 51 tinnitus and 51 hyperacusis clients showed they were present in 37% of the tinnitus clients and 94% of the hyperacusis clients [1].

Once TTTS has become established, the range of sounds that could elicit this involuntary response can increase to include everyday sounds, leading to the development and escalation of hyperacusis and phonophobia. An exaggerated startle is listed as one of the symptoms of PTSD (DSM-IV, D.5), and individuals with PTSD have been shown to produce heightened autonomic responses (eg increased heart rate) to acoustic stimuli that would not be expected to produce a startle response.

A subsequent acoustic incident exposure can therefore lead to a highly enhanced startle response, so that repeated acoustic incidents can significantly enhance ASI vulnerability. The more extreme the resultant middle ear reflex contractions, the more severe the TTTS symptoms can become, exacerbating the extent of the associated trauma reaction.

Hearing assessments

For clients with severe ASI, listening to sounds via headphones can be highly threatening and often leads to a significant increase in symptoms, which can persist for days. I consider that frequent audiological testing should not be carried out for clients with severe ASI symptoms. Any suprathreshold audiological testing, including loudness discomfort testing, and in particular acoustic reflex testing due to the volume levels required, is contraindicated. Some ASI clients have unfortunately had their symptoms permanently exacerbated as a result of a traumatic response to acoustic reflex testing.

Differential Diagnosis of ASI

TTTS symptoms can be readily confused with outer/middle/inner ear pathology, and an ENT specialist investigation is recommended to exclude this possibility. If severe vertigo is reported a perilymph fistula needs to be excluded.

ASI symptoms can be mistakenly diagnosed as due to temporomandibular joint disorder (TMD). TMD can produce TTTS, with referred ear pain and other TTTS symptoms present in about 40% of patients with TMD. With TMD, TTTS is a secondary consequence of physical dysfunction of the jaw joint; the TTTS symptoms do not tend to escalate and hyperacusis is not usually present. With TTTS associated with ASI/hyperacusis/tinnitus, the primary cause is central, related to a trauma response to sound and the symptoms can readily escalate.

On examination of the affected ear, the ear canal and tympanic membrane generally appear healthy and normal. ASI symptoms are subjective, so an experienced clinician makes a diagnosis on the basis of a thorough case history noting symptom onset, persistence and escalation; and their link with exposure to intolerable (or difficult to tolerate) sounds. If they have developed in association with acoustic incident exposure and/or hyperacusis is present, it is likely that they are a result of TTTS. The symptoms are remarkably consistent.

Significant malingering is rare in ASI clients, in my experience. Most clients are bewildered and frightened by their symptoms and desperate to recover.

Management of ASI

The most persistent ASI symptoms tend to be aural pain/blockage and hyperacusis. Sharp stabbing aural pain and numbness/burning in and around the ear are consistent with trigeminal nerve irritation. If pain levels are severe, treatment for trigeminal neuralgia and/or referral to a pain management clinic is recommended. Hyperacusis desensitisation therapy and massage of the muscular trigger points around the neck and shoulder will be of benefit in reducing TTTS symptoms, but progress can be slow once symptoms have become entrenched.

Output limiters

With the identification of ASI, output limiters in headset equipment have been developed to restrict maximum volume levels transmitted down a telephone line. However, ASI continues to occur despite their use. In my opinion they are of benefit primarily to help reduce the probability of an initial acoustic incident exposure. If TTTS develops, because of the vulnerability of further escalation at lower volume levels, it is impossible to give a 100% guarantee of protection. The dominant factors of an acoustic incident leading to ASI appear related to the sudden onset, unexpectedness and impact quality of loudish sounds outside the person's control near to the ear(s), rather than to high volume levels alone.

Return to work

An ASI client is vulnerable to a significant exacerbation of their symptoms should they be exposed to any unexpected, sudden onset, loud sound via a headset worn on either the affected ear or their other ear. I consider the ASI client should not return to headset or telephone duties until the symptoms have *fully* resolved. A graded return to work can then be carried out with *handset* use initially on the opposite ear.

**Ms Myriam Westcott developed this information on ASI after evaluating and providing therapy for over 65 ASI clients. This guide and a full copy of:
Westcott, M. "Acoustic Shock Injury." *Acta Otolaryngologica Supplement*, 556, 2006:
54-58 can be downloaded from our website (www.dineenandwestcott.com.au).**