

# Acoustic Shock Disorder (ASD) and Tonic Tensor Tympani Syndrome (TTTS)

## Guide for Medical Practitioners

Dr Ross Dineen  
BBSc, PhD  
MAudSA(CCP), MAAAPP

Ms Myriam Westcott  
B.Sc, GradDipAud  
MAudSA(CCP), MAAAPP

Ms Mary Leung  
B.Sc, GradDipAud  
GradDipPsych  
MAudSA(CCP)

Ms Kate Moore  
B.Sc, GradDipAud  
MAudSA(CCP)

**Acoustic shock (AS)** 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. AS symptoms are usually temporary. The term **acoustic shock disorder (ASD)** has been developed if AS symptoms persist.

The potential severity and persistence of ASD 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 ASD symptoms in their patients.

### Background

Call centre staff using a telephone headset or handset are vulnerable to ASD 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-occurring 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 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.

### ASD symptoms

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

- a shock/trauma reaction. A severe ASD can lead to Post Traumatic Stress Disorder (PTSD)
- sensations of pain/blockage/pressure/tympanic fluttering in the ear
- subjective muffled/distorted hearing. ASD 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 ASD 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 AS 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, ASD 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 ASD 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 more frequently contracting and relaxing, aggravated by exposure to sounds perceived as difficult to tolerate. This appears to activate a series of physiological reactions in and around the ear, which can include: alterations in tympanic membrane tension; 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, with the development of muscular trigger points consistent with 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 patients. I have recently co-ordinated a multi-clinic TTTS prevalence study of 345 tinnitus/hyperacusis patients attending ENT and Audiology clinics in Australia, Spain, Brazil and New Zealand. Data was collected on all consecutive patients seen in the eight participating clinics over a six month survey period, including tinnitus/hyperacusis severity, onset from an acoustic incident trigger, the presence of symptoms consistent with TTTS and whether these symptoms were exacerbated by exposure to loud/intolerable sounds. All patients were medically cleared of underlying pathology that could have caused the symptoms. 61% of the total sample reported at least one TTTS symptom; 49% reported 2 or more symptoms. Hyperacusis was present in 51% of the total sample. If hyperacusis was present, 81% of the sample reported at least one TTTS symptom; 66% reported 2 or more symptoms. 23% of the total sample had ASD, of those, 82% had hyperacusis. These results confirm that symptoms consistent with TTTS can readily develop in patients with tinnitus, and more particularly in those with hyperacusis; that hyperacusis incidence is significant in tinnitus patients; and that ASD is a universal phenomenon. This study was presented orally and in poster form at the Xth International Tinnitus Seminar, Florianopolis, Brazil March 2011, where it was selected as the best poster out of 95, winning the inaugural Jack Vernon Award for originality, innovation, study design, analysis and ultimate impact on the field of tinnitus.

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 ASD 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 patients with severe ASD, 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 patients with severe ASD symptoms. Any suprathreshold audiological testing, including loudness discomfort testing, and in particular acoustic reflex testing due to the volume levels required, is contraindicated. Some ASD patients have unfortunately had their symptoms permanently exacerbated as a result of a traumatic response to acoustic reflex testing.

### **Differential Diagnosis of ASD**

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.

ASD symptoms can be mistakenly diagnosed as due to jaw joint dysfunction. Jaw joint dysfunction can potentially lead to temporomandibular disorder (TMD). TMD research has implicated TTTS as a major cause of the referred ear pain and other symptoms in and around the ear, with aural symptoms shown to be present in about 40% of patients with TMD. When TTTS is a secondary consequence of TMD and/or jaw joint dysfunction, the TTTS symptoms do not tend to escalate and hyperacusis is not usually present. With TTTS associated with ASD/hyperacusis/tinnitus, the primary cause is central, related to a trauma response to sound and the symptoms can readily escalate. Severe ASD can lead to TMJ disorder and/or TMD as a secondary consequence.

On examination of the affected ear, the ear canal and tympanic membrane generally appear healthy and normal. ASD 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 ASD patients, in my experience. Most patients are bewildered and frightened by their symptoms and desperate to recover.

### **Management of ASD**

The most persistent ASD 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 ASD, output limiters in headset equipment have been developed to restrict maximum volume levels transmitted down a telephone line. However, ASD 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 ASD appear related to the sudden onset, unexpectedness and impact quality of loudish sounds, outside the person's control, heard near to the ear(s), rather than to high volume levels alone.

### **Return to work**

An ASD patient 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. After seeing this occur in a number of ASD patients with persistent symptoms, I now consider the ASD patient 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 ASD and TTTS after evaluating and providing therapy for over 100 ASD patients. 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](http://www.dineenandwestcott.com.au)).**

**Contact Ms Westcott for a copy of her more recent publication "Acoustic Shock Disorder": invited speaker at Tinnitus Research Initiative Asia and Pacific Tinnitus Symposium, Auckland 11 – 12 September 2009. Conference Proceedings published in the New Zealand Medical Journal.**