

ORIGINAL ARTICLE

Acoustic shock injury (ASI)

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Abstract

Conclusion. The potential severity and persistence of ASI symptoms has significant clinical and medico-legal implications. With the rapid growth of call centres around the world, professionals providing tinnitus and hyperacusis therapy are increasingly likely to encounter some or all of the cluster of ASI symptoms in their clients. **Background.** Acoustic shock injury (ASI), occurring as a result of exposure to a sudden unexpected loud sound, has been observed to cause a specific and consistent pattern of neurophysiological and psychological symptoms. These include aural pain, tinnitus, hyperacusis/phonophobia, vertigo and other unusual symptoms such as numbness or burning sensations around the ear. A range of emotional reactions including trauma, anxiety and depression can develop. 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 sudden unexpected loud sounds randomly transmitted via the telephone line. **Discussion.** This paper presents an overview of a study of 103 people exposed to 123 acoustic incidents, and of the proposed neurophysiological mechanism of ASI, in particular tonic tensor tympani syndrome (TTTS). An understanding of TTTS has the potential to provide insight into the neurophysiological and psychological development of tinnitus and hyperacusis and the association with high levels of emotional trauma and anxiety. **Rehabilitation.** ASI rehabilitation is discussed.

Keywords: *Acoustic shock injury, tonic tensor tympani syndrome, hyperacusis, hyperacusis rehabilitation, tinnitus retraining therapy*

Introduction

Research into acoustic shock injury (ASI) in call centre workers has the potential to expand our understanding of the psychological and neurophysiological mechanisms underlying tinnitus and hyperacusis. The processes involved in the development of ASI can provide insight into the relationship between tinnitus and hyperacusis, as well as providing a stronger rationale for therapy.

Background

In the early 1990s, co-inciding with the rapid growth of call centres in Australia, increasing numbers of call centre employees were reporting an unusual cluster of symptoms following exposure to a sudden unexpected loud sound (acoustic incident) randomly transmitted via the telephone line [1]. A similar pattern was being noticed overseas [2,3]. These

neurophysiological and psychological symptoms were different to those occurring with traditional noise injury and include a shock/trauma reaction, pain in and around the ear, other sensations including burning/numbness/blockage in and around the ear, tinnitus, hyperacusis and vertigo.

The increased numbers of people reporting this specific and consistent pattern of symptoms led to further research [1,4], and the term acoustic shock injury (ASI) was developed to identify this symptom cluster.

Two international seminars on ASI were held in Australia in 2001: 'Risking acoustic shock', 18 and 19 September, Fremantle, WA and 'Beyond acoustic shock', 25 September, Sydney, NSW. Papers on ASI were given at the XXVI International Congress of Audiology 17–21 March 2002, Melbourne, Victoria.

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), to an acoustic incident, and the research on ASI to date has focused on this cohort. However, any clients who have developed tinnitus and hyperacusis, particularly following exposure to a sudden unexpected loud sound, are likely to have reported at least some of these symptoms.

An overview of ASI symptoms

Typical descriptions of acoustic shock include 'like being stabbed with an icepick in the ear', 'like being electrocuted in the ear'. For those using a headset, the immediate reaction is to pull it off.

The initial symptoms include a severe startle reaction with a head and neck jerk, in extreme cases, falling to the floor (Tullio's symptom); pain in the ear/neck/arm; tinnitus; hyperacusis; sensations of burning, numbness, tingling and feeling of fullness in the ear; vertigo/nausea; and a shock response with shaking, crying, disorientation, headaches and fatigue [1].

Milhinch and Doyle [1] carried out the first large-scale study of ASI, using data from a number of audiological clinics and from the files of a large call centre operator. The cases studied came from four states in Australia from a total population of 16 000 call centre workers. A total of 103 people, including 91 females (89%) and 12 males (11%), were exposed to 123 acoustic incidents in the period 1994–1999.

Pain was the most frequent symptom, reported by 95%. Of all reports of pain, more than 81% were of ear pain, which varied from stabbing pain to duller earaches, or sometimes a combination of these; 11% of pain reports were of pain in the neck or jaw, and 7% of pain reports were of facial pain.

Tinnitus was the next most frequent symptom, reported by 50%. It was usually accompanied by other symptoms, but in 6% it was the only symptom. Loss of balance was reported by 48%. The most distressing and durable symptom tended to be hyperacusis, reported by 32%.

Vertigo was severe in a few cases, and associated with a suspected fistula.

Of particular interest were the other symptoms reported. These included headaches (32%), facial numbness (9%), a burning feeling in the ear or face (5%), tingling (3%), a feeling of pressure or fullness in the ear (11%), and an echo, or hollow feeling in the ear (18.4%). The symptoms typically did not include a hearing loss, although if present (18.4%), it may have been temporary or permanent, and did not follow the typical high frequency pattern of a noise-induced hearing injury but tended to affect low and mid frequency sensorineural hearing.

In some cases all symptoms resolved within a few hours or days. In other cases symptoms persisted for months or indefinitely. In the long term, for some people (10%) a range of emotional reactions developed including anxiety, depression, hypervigilance, anger and feelings of vulnerability.

Repeated acoustic incident exposure exacerbated an individual's vulnerability to ASI, as well as the degree and persistence of their injury. Pre-existing stress/anxiety, as well as fear of repeated incident exposure, also appeared to increase vulnerability to ASI, with a ripple effect observed among other staff members [5]. Call centre staff are therefore particularly vulnerable, as their workplace can be stressful – the job is often repetitive, monitored and competitive, and the calls they make are frequently unwelcome [5].

Acoustic incidents

Acoustic incidents through a telephone line can originate as feedback oscillation, fax tones, signalling tones, or even malicious whistle blowing by dissatisfied customers [6]. The typical call centre work environment is a large open plan office, with significant ambient noise from many voices making telephone calls. If the ambient noise level is high, operators need to turn up the volume of their headset, increasing their risk of exposure to an acoustic incident.

Available data suggest that the acoustic incident triggering ASI is frequently a tone of an intensity of 82–110 dB SPL at the tympanic membrane, in the frequency range from 2.3 to 3.4 KHz and with a rise time of between 0 and 20 ms and of varying duration [1]. The duration is typically brief, lasting until the operator is able to remove the headset from their ear. Therefore, the sound is rarely loud enough or present for long enough to cause the classic pattern of noise-induced high frequency sensorineural hearing loss or a fistula.

ASI symptoms can develop without specific acoustic incident exposure. I have evaluated a call centre workplace where the changeover to a new telephone system resulted in ASI symptoms in some staff members. The trigger appeared to be that the new system allowed incoming voices to be heard at an unpredictable and often high volume level, with no immediate volume control available to staff.

Discussion: a proposed mechanism of ASI – tonic tensor tympani syndrome

Patuzzi et al. [7,8] sought a physiological explanation of the more unusual symptoms, observing that they were consistent with a condition called tonic

tensor tympani syndrome (TTTS), originally described by Dr I. Klockhoff [7–12]. Their proposal attributed the primary cause of ASI to excessive middle ear muscle contractions (stapedius and tensor tympani), in particular tensor tympani contraction [7,8].

TTTS is a condition in which the tensor tympani muscle is spontaneously active, continually and rhythmically contracting and relaxing [5,11]. Klockhoff and Westerberg [5,11], on the basis of clinical observations and experiments, stated that the symptoms of this chronic activity include aural pain, aural fullness, tinnitus, distorted hearing (but with normal audiometry), vertigo and tension headache. Klockhoff [12] considered the vertigo to be of central (reticular formation) origin because vestibular tests were usually normal. Klockhoff and Westerberg noted that ‘elevated psychic tension seems to be the essential etiologic factor in almost all cases’ [cited in 5,8,13].

Djupesland [5,13,14] showed that in 70 of 75 subjects, the mere sight of an item that could emit a loud stimulus (a toy pistol) was enough to produce contraction of the middle ear muscles. When anxiety was reduced, the anticipatory muscle contractions subsided.

While the stapedial reflex is an acoustic reflex triggered by high volume levels, the tensor tympani reflex is a startle reflex [5,8]. Additionally, there is evidence that the tensor tympani contracts immediately preceding the sounds produced during self-vocalization [15,16]. This pre-vocalization reflex, implicated as a mechanism to prevent over-stimulation of the cochlea by the sounds produced by the voice, suggests that the tensor tympani has an established protective function.

The initial symptoms of ASI such as sharp pain in and around the ear, sensations of burning, numbness and blockage in and around the ear, tinnitus, vertigo and headache are consistent with TTTS. These symptoms were proposed by Patuzzi to be due to TTTS causing an abnormal stimulation of the neurones of the branches of the facial, glossopharyngeal, vagus, trigeminal and cervical nerves innervating the tympanic membrane and ossicular chain [8].

Interestingly, many ASI symptoms mimic those of temporomandibular disorder (TMD). Anatomically, the tensor tympani and tensor veli palatini muscles have a role in mastication because they are modulated by motoneurons from the trigeminal motor nucleus (V3) [15]. TMD can produce an episodic or constant spasm of the tensor tympani muscle, with referred ear pain and other aural symptoms (of non-otologic origin) present in up to 42% of patients with

TMD [15]. This supports the proposal that ASI could be caused by TTTS.

The tensor tympani reflex has a variable threshold to sound, which can be reprogrammed downwards [8], can be triggered without sound but appears to be generally triggered by the anticipation and sub-conscious perception of sounds considered threatening. TTTS symptoms of varying degrees (e.g. aural fullness, tympanic fluttering) are regularly seen in tinnitus clients [17]. In my experience as an audiologist specializing in tinnitus and hyperacusis therapy, TTTS symptoms most frequently appear in clients whose anxiety appears to be focused on a need to protect their ears from sounds that could cause an escalation in their tinnitus and/or hearing loss and/or their TTTS symptoms. This could be consistent with the protective reflex function of the tensor tympani, and it could therefore be argued that TTTS provides a clear demonstration of the physiological manifestation of an anxiety state. In my experience evaluating and treating 34 ASI clients, hyperacusis is often part of the immediate symptom pattern. As the tensor tympani reflex threshold is centrally mediated, the trauma reaction and anxiety associated with ASI could rapidly lead to the development and escalation of hyperacusis and phonophobia. In my opinion, TTTS provides a plausible explanation of the aural pain following exposure to intolerable sounds commonly experienced by hyperacusis clients.

ASI symptoms can range from mild to severe [1]. In my experience, there appears to be no typical duration pattern of ASI symptoms, although milder symptoms are more likely to be temporary and respond more readily to rehabilitation, while severe symptoms are more likely to be persistent.

ASI/TTTS symptoms are involuntary and subjective. The unusual symptom cluster is readily misunderstood or not believed. As a result of an inadequate understanding of the symptoms, and if they persist or escalate, secondary and long-term psychological symptoms can develop [1,8]. In my experience, these can include phonophobia, auditory hypervigilance, anxiety, depression, post-traumatic stress reaction/disorder, fatigue and anger.

Post-traumatic stress disorder (PTSD) is marked by symptoms of persistently re-experiencing a trauma, persistent avoidance of stimuli associated with the trauma and hyperarousal. Hyperarousal includes the symptoms of hypervigilance and an exaggerated startle response. While the reaction to ASI may not develop into a full-blown PTSD, a post-traumatic stress reaction may include elements of these symptoms to varying degrees. When sounds are associated with the traumatic event, and more particularly if the traumatic event is a

sound, acoustic hypervigilance can become extreme as a means of limiting and controlling the acoustic environment [18]. This will exacerbate the exaggerated startle to impact or sudden unexpected sounds.

Malingering is rare in ASI clients, according to Patuzzi [8], and in my own experience. ASI clients are often desperate to recover from their symptoms, although, as with any injury, secondary gain can affect the commitment to rehabilitation.

Many of the long-term symptoms of severe ASI are consistent with severe hyperacusis, or category 4 as defined in tinnitus retraining therapy (TRT) [19,20]. Some of the most severe cases of hyperacusis seen in my clinic are clients with ASI. The validity of measuring loudness discomfort levels has not yet been convincingly proven [21]. As it is an ordeal for clients with severe hyperacusis, and can significantly aggravate their TTTS symptoms, it is not carried out with these clients in my clinic.

Rehabilitation

Understanding TTTS potentially provides a rationale and framework for rehabilitation. When symptoms are present, TTTS can be included in an explanation of the peripheral and central auditory pathway, using Jastreboff's neurophysiological model and following the TRT protocol [20,22,23].

The judicious use of solid and/or musician's filtered ear protection frequently assists in maintaining or facilitating the expansion of lifestyle horizons, with encouragement and support given to gradually reduce their use.

Environmental sound enrichment strategies are recommended to promote hyperacusis desensitization [24]. TRT category 4 clients with severe hyperacusis, whether ASI-based or not, are in my experience rarely able to tolerate ear level sound generators. The proximity of the generators appears to escalate their subconscious need to protect their ears or hearing to maintain their current tinnitus/TTTS levels. Despite careful reassurance and counselling, the subsequent and involuntary increase in anxiety appears to increase their TTTS symptoms, and rarely permits sustained use of the devices. An alternative is to use a portable CD or MP3 player producing white noise or a stable, neutral environmental sound presented via headphones worn around the neck, rather than over the ears.

Based on Vernon's approach to hyperacusis management [25], four of my clients have been fitted with sophisticated digital signal processing in-the-ear hearing aids set up to act as electronic filters, by using compression to reduce volume levels of all

sounds down to a normal conversational speech volume range. This avoids over-protection of the ears while at the same time providing effective protection against loud sounds. When anxiety levels permit, there is opportunity for desensitization by gradually increasing the upper volume limit of the hearing aids. The three successful fittings were clients with a hearing loss as well as hyperacusis. The benefit appeared to reflect that their need for an improvement in hearing conversation, particularly in competing sound, over-rode the strength of the anxiety leading to TTTS. The other ASI client rejected them for the same reasons given above for ear level sound generators. Despite the protection against loud and moderate volume sounds provided by the hearing aids, she found the proximity of even soft sounds intolerable because they were perceived as right next to the ears, rather than in their correct position. She doggedly persevered for several weeks, but could not cope with the continued increase in her TTTS symptoms.

Without evaluating and addressing the psychological factors underpinning TTTS/ASI, rehabilitation is likely to be limited. Cognitive behavioural therapy strategies to reduce auditory hypervigilance and phonophobia are required. These include cognitive distraction strategies to manage auditory hypervigilance, and active stress and sleep management strategies. Specific phonophobia strategies may need to be individually developed for each client. Referral for psychological/psychiatric treatment for anxiety, depression and trauma needs to be carried out when indicated. This is particularly required if pre-existing anxiety and depression were reported, which are likely to have exacerbated the level of emotional reaction subsequent to the ASI.

Call centres in Australia are starting to become aware of the risk of ASI and the need for ASI workplace management. Rapid referral of affected staff can help to contain the anxiety leading to an escalation of symptoms [1]. While output limiters have been developed to restrict maximum volume levels transmitted down a telephone line, in my opinion they are of benefit primarily to help reduce the probability of an initial acoustic incident exposure. If TTTS develops, there is a vulnerability to further escalation at lower volume levels [1], and it is impossible to give a 100% guarantee of protection. The workplace cited earlier indicates that ASI can develop without specific acoustic incident exposure. The dominant factors leading to ASI appear to be related to the sudden onset, unexpectedness and impact quality of loudish sounds outside the person's control, rather than to high volume levels alone.

Conclusion

The potential severity and persistence of ASI symptoms have significant clinical and medico-legal implications. With the rapid growth of call centres around the world, professionals providing tinnitus and hyperacusis therapy are increasingly likely to encounter some or all of the cluster of ASI symptoms in their clients.

More generally, the presence of TTTS symptoms in tinnitus and hyperacusis clients suggests a promising direction for further research to explore the emotional response to tinnitus and the development of hyperacusis. This could include the development of an effective, practical measurement tool to validate the presence and document the incidence of TTTS.

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