

Acoustic shock

D J McFERRAN, D M BAGULEY*

Abstract

Acoustic shock is a recently recognised clinical entity: following an abrupt, intense and unanticipated acoustic stimulus, usually delivered by a telephone handset or headset, some individuals report a symptom cluster that includes otalgia, altered hearing, aural fullness, imbalance, tinnitus, dislike or even fear of loud noises, and anxiety and/or depression. Symptoms start shortly after the triggering acoustic incident and can be short-lived or can last for a considerable time. If persistent, the condition can lead to significant disability. Proposed mechanisms include involvement of the tensor tympani muscle, hyperexcitability of central auditory pathways, and a precursive state of raised anxiety or arousal. A formal treatment programme has not yet been proposed, but the potential utility of modern therapeutic techniques for tinnitus and hyperacusis are considered. Given the large number of UK residents working in telephone call centres, this condition is of considerable clinical importance.

Key words: Occupational Noise; Hyperacusis; Tinnitus; Serotonin; Ear Diseases

Introduction

Over the last two decades, there has been a huge expansion in the number of people employed in call centres (contact centres) or similar workplace environments. Approximately 900 000 people (3 per cent of the working population of the United Kingdom) are now engaged in this type of employment (S Morrell, personal communication), making call centre work the most common job in the country. People employed in this fashion are frequently required to use telecommunications equipment with either headsets or telephone handsets for protracted periods of time. They may work in areas with poor acoustics and relatively high levels of ambient noise.

It has been recognised for many years that people who wear headsets at work may be subjected to spurious auditory signals. In 1976, a study of telephone operatives found 36 such signals of between 94 and 109 dB in 2000 hours of recording, or approximately one signal per operative per week,¹ although no significant audiological sequelae were reported after these exposures.

As the number of people employed in call centres has risen, so too has the number of reports of workers developing a pattern of both physical and psychological symptoms arising immediately after or soon after exposure to sudden, unexpected noise over their headset or handset. This symptom cluster occurring after noise exposure has become known as acoustic

shock syndrome, acoustic shock injury or simply acoustic shock.

Noises that generate acoustic shock do not have an intensity and duration profile that would be regarded as dangerous to the auditory system within the framework of existing workplace legislation. In this respect, it is important to distinguish acoustic shock from acute acoustic trauma that is experienced with exposure to extremely loud sounds, over 140 dB. Similarly, acoustic shock is unrelated to noise-induced hearing loss, in which repeated exposure to sounds of an intensity greater than 85 dB causes cochlear damage.

The majority of reports of acoustic shock have come from Denmark² and Australia,³ with a significant but smaller number arising from the United Kingdom.⁴ However, there is a dearth of reports from other countries with large numbers of call centre workers. This has led some researchers to question whether the syndrome is a genuine entity. One of the possible explanations for this seeming paradox is that, as an emerging syndrome, the condition often passes unrecognised and is almost certainly under-reported at present.

Although acoustic shock has become firmly associated with the use of telecommunications equipment, it is likely that exposure to other forms of sudden, unexpected sound can generate similar symptoms. There are anecdotal reports of people developing symptoms resembling acoustic shock after exposure

From the Department of Otolaryngology and Head and Neck Surgery, Essex County Hospital, Colchester, and the *Department of Audiology, Addenbrookes Hospital, Cambridge, UK.
Accepted for publication: 13 November 2006.

to noise from engineering equipment or from their personal stereo headphones. However, these types of exposure form a much more heterogeneous set and have largely been excluded from formal definitions of the syndrome.

Acoustic shock and post-traumatic stress disorder share many clinical features, including persistent avoidance of stimuli associated with the trauma, sleep disturbance, hypervigilance and exaggerated startle response.⁵ Parallels can also be drawn with other forms of altered sound tolerance, including hyperacusis and phonophobia. These observations have led some workers to question whether acoustic shock is a discrete entity or whether it is simply a subset of one of these conditions.

Much of the information pertaining to acoustic shock is located on websites, in conference proceedings, and in health and safety documents; little has yet reached peer-reviewed medical literature. This paucity is hampering both recognition of the condition and further research.

Definitions

As this is a new and developing field, there are still several slightly different definitions of acoustic shock, and global consensus has yet to be reached. One of the first definitions of acoustic shock was produced by the International Telecommunications Union and European Transmission Standards Institute, which defined the condition as 'any temporary or permanent disturbance of the functioning of the ear, or of the nervous system, which may be caused to the user of a telephone earphone by a sudden sharp rise in the acoustic pressure produced by it'.⁶

The Health Services Australia Group has produced a slightly different definition which states 'acoustic shock refers to the combination of exposure to a brief, sudden, unexpected, high frequency, high intensity sound emitted (the stimulus) and the subsequent symptoms (the response) which can develop'. This group also produced a definition for the causative sound or 'acoustic incident': 'acoustic incident refers to a sudden, unexpected, high-pitched sound of high intensity. It may be emitted from a headset or handset and is commonly reported as a 'shriek'.⁷

In the United Kingdom, the Acoustic Safety Programme has recently published the following definitions: 'an acoustic incident is a sudden, unexpected, noise event which is perceived as loud, transmitted through a telephone or headset' and 'acoustic shock is an adverse response to an acoustic incident resulting in alteration of auditory function'.⁸

Several other definitions exist with subtle variations on the above themes. All of the available definitions have advantages and disadvantages, and it seems likely that further honing of the definition will occur.

Acoustic incidents

Although the sounds that can generate acoustic shock are increasingly referred to as acoustic incidents, other terminology exists. The triggering

sounds have been described as acoustic shocks, audio shocks, acoustic shrieks, high-pitched tones, spikes, howls, screeches or squawks. There are many potential ways that such sounds may arise within a call centre workplace. There may be faulty telephone or headset equipment or transmission faults within the network. Positive feedback may occur with some cordless and mobile phones. Tones from facsimile machines or modems may be misdirected to an operative's earpiece. There have been occasions on which a customer has maliciously generated an acoustic incident by shouting or blowing a whistle into their telephone.

Although many disparate sound sources seem capable of generating acoustic shock, there are some common features in the characteristics of the sounds. A Danish study identified acoustic incidents featuring sounds of intensities varying from 56 to 108 dB, in the frequency range 100 Hz to 3.8 kHz.² Work in Australia by Milhinch³ suggested that the causative sound is often in the frequency range 2.3 to 3.4 kHz, with an intensity of 82 to 120 dB. The rise time of the sound is usually very short, varying between 0 and 20 milliseconds. The duration of exposure is very difficult to estimate because the natural response of the affected person is to remove the headset or handset from the affected ear(s). Because removing a headset from the head takes longer than moving a handset away from the ear, it seems likely that wearing a headset carries more risk of incurring acoustic shock.

Symptom profile

Although most people develop their symptoms immediately after exposure to the acoustic incident, there are a small number of people who develop their symptoms several hours after the event.

The largest systematic study into symptoms of acoustic shock was carried out in Australia between 1995 and 1999.³ One hundred and three patients who had been exposed to acoustic incidents were investigated. Of the symptoms seen immediately or soon after exposure to an acoustic incident, ear pain was the most common complaint, occurring in 81 per cent of cases. There were reports of pain in the neck or jaw in 11 per cent and of pain in the face in 7 per cent. Tinnitus was described in 50 per cent of cases and balance problems were present in 48 per cent. Other symptoms included a sensation of blockage or aural fullness, numbness or even collapse. Hearing loss was relatively uncommon, occurring in only 18.4 per cent of cases, and there was no statistically significant audiological difference between exposed and non-exposed ears except at a frequency of 1.5 kHz. Other symptoms took longer to emerge and included anxiety, depression, headache, sensitivity to previously tolerated sounds, hypervigilance and anger. Affected people developed a mean of 2.7 symptoms per shock. For many people, the symptoms resolved within hours or days, but for some the symptoms persisted in a chronic fashion.

These findings are in broad agreement with observations made in the United Kingdom⁹ and Denmark.²

Epidemiology

There are no reliable data available regarding the prevalence and incidence of acoustic shock.

A study in Denmark looked at reports of acoustic shock from 14 call centres.² Some centres reported no incidents, whereas one centre reported that 22 per cent of 90 operatives had experienced an acoustic shock. The study examined the characteristics of those who had experienced an acoustic shock and found a statistically significant correlation with stress, smoking, and neck and shoulder pain. Employees who had experienced more than one acoustic shock tended to report worse symptoms.

In an Australian study of 103 people reporting acoustic shock, 91 (89 per cent) were female and 12 (11 per cent) were male.³ This compares to a sex ratio of 74 per cent female to 36 per cent male in the call centre workforce in Australia.

It has been suggested that exposure to an acoustic incident does not automatically result in the development of acoustic shock symptoms, and that even when acoustic shock does result it is short-lived in the vast majority of cases. Whilst this may seem like common sense, it is not yet underpinned by evidence, highlighting the need for robust studies in this area.

Pathophysiology

The pathophysiological mechanisms underpinning acoustic shock remain obscure, and it is reasonable to assume that these will be complex and multifactorial. The symptom profile includes experiences that can variously be ascribed to middle ear, cochlear and central auditory pathway involvement. Additionally, the marked emotional impact of the acoustic shock experience leads one to consider a role for psychological mechanisms. Each of these will be considered in turn. It should be noted that such mechanisms are by no means mutually exclusive.

Middle ear

The ossicular chain has associated muscle and tendon structures. The role of the stapedius muscle in protecting the cochlea from intense sound exposure is well known. The function of the tensor tympani muscle is less well established; however, an involvement in setting the operating point of the eustachian tube has been proposed.¹⁰ Both of these muscles have been implicated in acoustic shock, with a particularly important role proposed for the tensor tympani muscle.^{3,11} A comparison has been made between acoustic shock injury and tensor tympani syndrome,¹² wherein spontaneous contractions of the tensor tympani (similar to blepharospasm) give rise to a fluttering or beating sensation. Additionally, there is evidence that middle-ear muscle function is influenced by the serotonergic system,¹³ and thus

there is a potential link between emotional state and middle-ear muscle contraction.

Cochlea

The high incidence of tinnitus in the acoustic shock population may cause one to consider cochlear dysfunction as a potential mechanism. However, the low incidence of frank hearing loss is at odds with such a proposal. In the few cases in which persistent hearing loss has been identified, there has been no characteristic pattern, contrasting with the typical audiometric findings seen in noise-induced hearing loss.³

Central auditory system

Various mechanisms involving the central auditory system have been suggested as causes of other forms of auditory hypersensitivity (such as hyperacusis and phonophobia); it is possible that these mechanisms may also be applicable to acoustic shock. It has been suggested that disturbance of central auditory serotonergic pathways may result in altered sound tolerance, without involvement of the middle-ear muscles. Certainly, auditory hypersensitivity is seen within several conditions that are thought to be due to abnormal function within serotonin pathways, including migraine, post-traumatic stress disorder and depression.^{14,15} Medial efferent system dysfunction has also been suggested as a possible cause of hyperacusis; nerve fibres from the medial efferent system terminate on the outer hair cells in the cochlea and this system has been proposed as being important in modulating auditory gain.¹⁶ Dysfunction of this system could result in the auditory system being kept in a state of abnormally high sensitivity.¹⁷ Such central auditory system mechanisms have the merit of suggesting how acoustic shock could arise without any evidence of accompanying peripheral auditory deficit.

Psychological mechanisms

The suggestion that some individuals who experience acoustic shock have a precursive state of anxiety, stress or arousal is of some interest. Auditory startle is potentiated by anxiety and arousal, and it may be that the onset of acoustic shock is triggered by a hyperintense startle to an unanticipated noise. Jastreboff¹⁸ (2000) suggested that aversive reaction to sound can occur, mediated by the limbic system and autonomic nervous system, and that this reaction is independent of the intensity of the sound. Work in the field of chronic pain¹⁹ has demonstrated that, in certain situations, a fear-avoidance pathway can be set up: pain produces fear which results in limitation of movement, and the subsequent inactivity causes further pain, setting up a vicious circle. Translating this observation to the auditory system, fear of sound can result in avoidance of sound, which in turn causes increased central auditory sensitivity, thereby enhancing the fear. Evidence supporting this hypothesis in acoustic shock is seen in the observation that if one person in a call centre suffers an

acoustic shock, there is increased risk of other operatives within that centre also developing symptoms. This process could also help explain the patchy distribution of the condition; it is relatively common in some workplaces and geographical locations and completely absent in others, despite similar working environments and similar or identical telecommunications equipment.

Prevention

Output limiters

Circuitry to limit the acoustic output of telecommunications equipment is widely available and undoubtedly has a useful role in the prevention of noise-induced hearing loss. Equipment fitted in call centres in the United Kingdom does ensure that noise above 118 dB is not transmitted. A study of 15 call centres²⁰ showed that daily personal noise exposure was unlikely to exceed the 85 dB(A) level defined by the 1989 noise at work regulations.²¹

However, simply limiting the output seems less effective in preventing acoustic shock. The sounds that trigger acoustic shock are not necessarily loud by conventional definitions and would not be filtered by simple limiting devices. In 1991, Blumenthal and Goode²² demonstrated that startle responses could occur following exposure to sounds with levels as low as 60 dB SPL. Also, reducing the overall output level through a headset can reduce the intelligibility of speech, particularly within the sometimes noisy environments of call centres. This can result in operatives straining to hear and therefore increasing their central auditory gain. This could negate the intended protective function of the circuitry or possibly even render the operative more susceptible to acoustic shock. Therefore, any limitation circuitry should preserve speech clarity.

Acoustic incident filtering

There have been several attempts to design more sophisticated filtering equipment that can recognise and reject acoustic incidents while allowing normal speech to pass through in a largely unaltered state. Several such devices are commercially available, but firm scientific proof of their efficacy in preventing acoustic shock is still awaited.

The call centre environment

Because acoustic shock seems more common among operatives who have pre-existing high levels of stress, it would seem sensible to try to design low impact working environments and to utilise working practices that do not cause or exacerbate stress. Ensuring a quiet workplace with good acoustic properties should be beneficial, by reducing the operatives' need to strain to listen.

Staff education

There is no evidence that making staff aware of acoustic shock as a potential problem has any direct effect on the prevalence of the condition.

However, it would seem sensible to educate staff to understand and have confidence in their telecommunications equipment. They should also be instructed to set the output level of their headset to the lowest level commensurate with satisfactory speech intelligibility.

Treatment

Anecdotally, one of the common complaints of patients who have experienced an acoustic shock is that their symptoms are ignored or minimised by medical staff. Following normal audiological tests, many patients are simply reassured that no damage has been sustained and are dismissed.

Recognition of the condition and a sympathetic approach are simple and helpful measures. One of the first actions of many patients with all forms of loudness intolerance is to try to avoid sound by protecting their ears with plugs and muffs. However, this may be counter-productive, as by reducing the amount of incoming auditory information central auditory gain is increased, further increasing the hypersensitivity of the auditory system. Patients should be counselled to use ear protection measures only when they are in genuinely noisy environments.

Techniques used for mainstream hyperacusis and phonophobic patients, such as tinnitus retraining therapy^{17,23} and psychological therapies,^{24,25} have been used to treat patients with acoustic shock. However, no firm evidence of the efficacy of this approach has yet emerged. Westcott¹¹ reported four cases of acoustic shock which were treated with sophisticated in-the-ear digital hearing aids, set up to act as electronic filters and compress all sounds down to the range of conversational speech. The rationale for this approach was that it would protect against dangerous sound levels while avoiding the risk of overprotection. Three of the four patients treated in this fashion showed improvement, but it is difficult to draw firm conclusions from such a small study.

Conclusions

A symptom complex arising from exposure to sudden, unexpected sound has been recognised. This condition shares some features with other conditions such as hyperacusis. However, there are sufficient differences to warrant its recognition as a separate condition in its own right, rather than as a subsection of an existing condition. As an emergent condition, the terminology is still somewhat confused, but there does seem to be merit in simplicity, and it is suggested that the condition should be termed acoustic shock and the triggering sound should be termed an acoustic incident.

Further research in this area is strongly indicated. Suggested topics include: prevalence and incidence studies; longitudinal study of cohorts of call centre workers; interrelationships between symptoms; treatment efficacy; functional imaging of patients with acoustic shock; and more detailed audiometric testing of patients with acoustic shock, using techniques such as distortion product otoacoustic emissions, to detect subtle cochlear changes.

Otology and audiology departments need to increase their awareness of this condition, as it is currently under-recognised and, anecdotally, patients often complain of having their symptoms ignored.

References

- 1 Alexander RW, Koenig AH, Cohen HS, Lepo CP. The effects of noise on telephone operators. *J Occup Med* 1979;**21**:21–5
- 2 Hinke K, Brask K. An investigation of the telephone services of the call centre of TeleDanmark in Aabenraa [in Danish]. Haderslev: Milijomedicinsk Klinik, 1999
- 3 Milhinch JC. Acoustic shock injury: real or imaginary. *Audiology Online*. http://www.audiologyonline.com/articles/article_detail.asp?article_iol=351
- 4 *IndeGent Exposure*. London: Royal National Institute for the Deaf/Trades Union Council, 1999
- 5 *Diagnostic and Statistical Manual of Mental Disorders, DSM-IV-TR*, 4th edn. Washington DC: American Psychiatric Association, 2000:467–8
- 6 Vocabulary of terms on telephone transmission quality and telephone sets. In: *Telecommunication Standardization Sector (ITU-T) of the International Telecommunication Union Recommendation P.10 (12/98)*. Geneva: International Telecommunication Union, 1998
- 7 Trifiletti B. *Fact Sheet: Acoustic Shock*. Canberra: HSA Group, 2001
- 8 National Definitions of Acoustic Shock in Telephone and Headset Users 2006. Salisbury: Acoustic Safety Programme and National Physical Laboratory, 2006:20–2
- 9 Lawton BW. Audiometric findings in call centre workers exposed to acoustic shock. *Proc Inst Acoustics* 2003;**25**: 249–58
- 10 Kierner AC, Mayer R, v Kirschhofer K. Do the tensor tympani and tensor veli palatini muscles of man form a functional unit? A histochemical investigation of their putative connections. *Hear Res* 2002;**165**:48–52
- 11 Westcott M. Acoustic shock injury (ASI). *Acta Otolaryngol Suppl* 2006;**556**:54–8
- 12 Klockhoff I. Impedance fluctuation and a “tensor tympani” syndrome. In: Penha RL, Pizarro P, eds. *Proceedings of 4th International Symposium on Acoustic Impedance Measurements*. Lisbon: Universidade Nove de Lisboa, 1981:69–76
- 13 Thompson AM, Thompson GC, Britton BH. Serotonergic innervation of stapedial and tensor tympani motoneurons. *Brain Res* 1998;**787**:175–8
- 14 Katzenell U, Segal S. Hyperacusis: review and clinical guidelines. *Otol Neurotol* 2001;**22**:321–7
- 15 Marriage J, Barnes NM. Is central hyperacusis a symptom of 5-hydroxytryptamine (5-HT) dysfunction? *J Laryngol Otol* 1995;**109**:915–21
- 16 Sahley TL, Nodar RH, Musiek FE. *Efferent Auditory System: Structure and Function*. San Diego: Singular, 1997
- 17 Jastreboff PJ, Hazell JWP. A neurophysiological approach to tinnitus: clinical implications. *Br J Audiol* 1993;**27**:7–17
- 18 Jastreboff PJ. Tinnitus habituation therapy (THT) and tinnitus retraining therapy (TRT). In: Tyler RS, ed. *Tinnitus Handbook*. San Diego: Singular, 2000:357–76
- 19 Lethem J, Slade PD, Troup JDG, Bentley G. Outline of a fear-avoidance model of exaggerated pain perception – I. *Behav Res Ther* 1983;**21**:401–8
- 20 Patel JA, Broughton K. Assessment of the noise exposure of call centre operators. *Ann Occup Hyg* 2002;**46**:653–61
- 21 Noise at work regulations 1989, Statutory instrument 1989 no 1790. London: Health and Safety Executive, 1989
- 22 Blumenthal T, Goode C. The startle eyeblink response to low intensity acoustic stimuli. *Psychophysiology* 1991;**28**: 296–306
- 23 Hazell JWP. Support for a neurophysiological model of tinnitus. In: Reich GE, Vernon JA, eds. *Proceedings of the Fifth International Tinnitus Seminar 1995*. Portland: American Tinnitus Association, 1996:51–57
- 24 Hallam RS, Rachman S, Hinchcliffe R. Psychological aspects of tinnitus. In: Rachman S, ed. *Contributions to Medical Psychology*. Oxford: Pergamon Press, 1984;**3**: 31–53
- 25 Andersson G. A cognitive-affective theory for tinnitus: experiments and theoretical implications. In: Patuzzi R, ed. *Proceedings of the Seventh International Tinnitus Seminar*. Fremantle: University of Western Australia, 2002:197–200

Address for correspondence:

Mr Don McFerran,
Department of Otolaryngology
and Head and Neck Surgery,
Essex County Hospital,
Lexden Road,
Colchester CO3 3NB, UK.

Fax: +44 (0) 1206 744773

E-mail: donald.mcferran@essexrivers.nhs.uk

Mr D J McFerran takes responsibility for the integrity of the content of the paper.

Competing interests: Both authors are members of the Research Advisory Board of the Acoustic Safety Programme and are reimbursed for that activity. Additionally, both have acted as expert witnesses in legal cases involving tinnitus.
