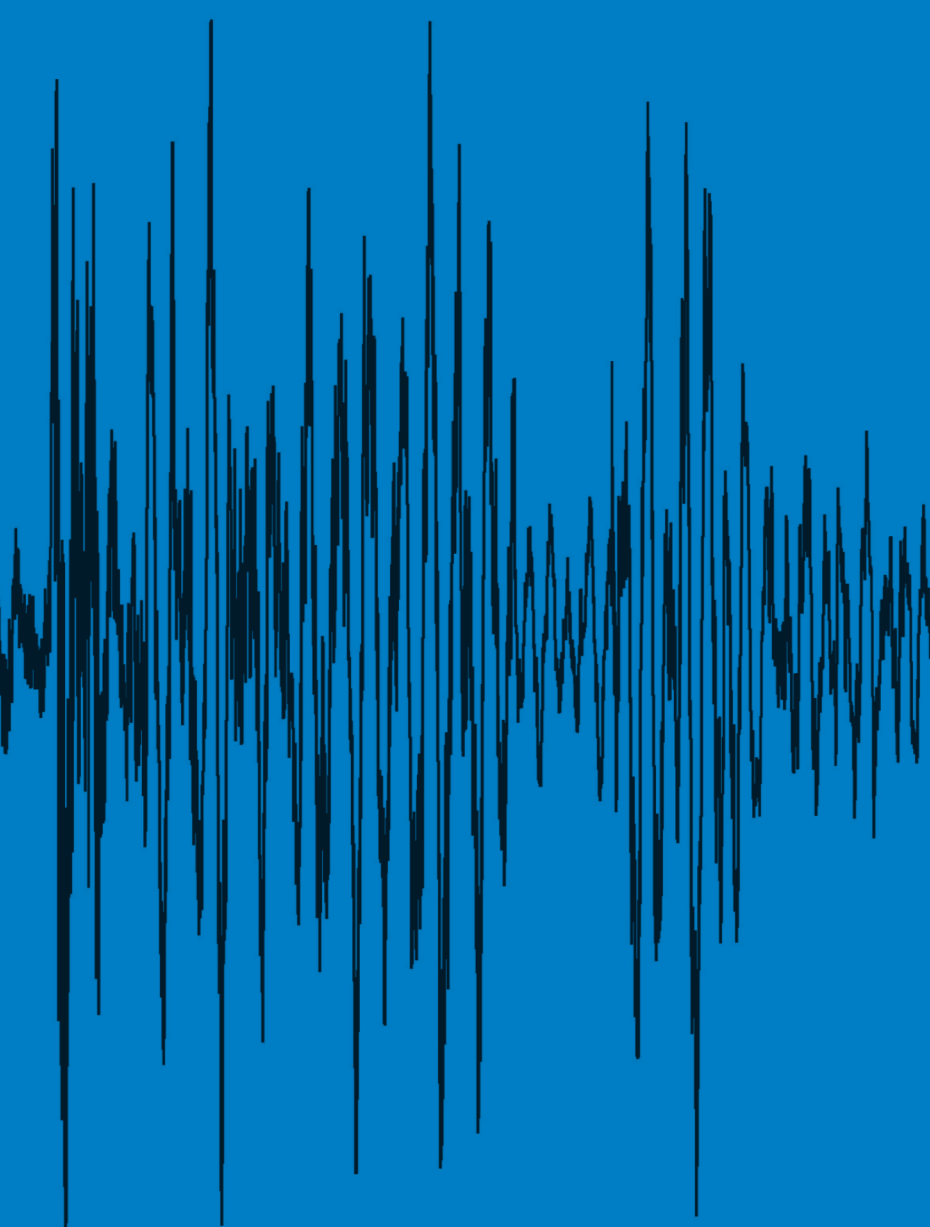


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Tonic tensor tympani syndrome in tinnitus and hyperacusis patients: A multi-clinic prevalence study

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Abstract

Tonic tensor tympani syndrome (TTTS) is an involuntary, anxiety-based condition where the reflex threshold for tensor tympani muscle activity is reduced, causing a frequent spasm. This can trigger aural symptoms from tympanic membrane tension, middle ear ventilation alterations and trigeminal nerve irritability. TTTS is considered to cause the distinctive symptoms of acoustic shock (AS), which can develop after exposure to an unexpected loud sound perceived as highly threatening. Hyperacusis is a dominant AS symptom. Aural pain/blockage without underlying pathology has been noted in tinnitus and hyperacusis patients, without wide acknowledgment. This multiclinic study investigated the prevalence of TTTS symptoms and AS in tinnitus and hyperacusis patients. This study included consecutive patients with tinnitus and/or hyperacusis seen in multiple clinics. Data collected: Symptoms consistent with TTTS (pain/numbness/burning in and around the ear; aural “blockage”; mild vertigo/nausea; “muffled” hearing; tympanic flutter; headache); onset or exacerbation from exposure to loud/intolerable sounds; tinnitus/hyperacusis severity. All patients were medically cleared of underlying pathology, which could cause these symptoms. 60.0% of the total sample (345 patients), 40.6% of tinnitus only patients, 81.1% of hyperacusis patients had ≥ 1 symptoms ($P < 0.001$). 68% of severe tinnitus patients, 91.3% of severe hyperacusis patients had ≥ 1 symptoms ($P < 0.001$). 19.7% (68/345) of patients in the total sample had AS. 83.8% of AS patients had hyperacusis, 41.2% of non-AS patients had hyperacusis ($P < 0.001$). The high prevalence of TTTS symptoms suggests they readily develop in tinnitus patients, more particularly with hyperacusis. Along with AS, they should be routinely investigated in history-taking.

Keywords: Acoustic shock, hyperacusis, tensor tympani, tinnitus, tonic tensor tympani syndrome

Introduction

The tensor tympani reflex is a startle reflex, which is exaggerated by high stress levels.^[1,2] The tensor tympani muscle contracts immediately preceding the sounds produced during self-vocalisation, suggesting it has an established protective function to loud sounds,^[3] assists in the discrimination of low frequency sounds,^[4] and is involved in velopharyngeal movements.^[3,4]

Tonic tensor tympani syndrome (TTTS) was originally

described by Dr. I. Klockhoff.^[5-8] TTTS is an involuntary condition where the centrally mediated reflex threshold for tensor tympani muscle activity becomes reduced, so it is continually and rhythmically contracting and relaxing.^[5-8] This appears to initiate physiological reactions in and around the ear without objectively measurable dysfunction or pathology.^[4,9] Symptoms consistent with TTTS can include: tinnitus; rhythmic aural sensations such as clicks and tympanic membrane flutter; alterations in ventilation of the middle ear cavity leading to a sense of aural blockage or fullness, a frequent aural “popping” sensation and mild vertigo;^[10,11] minor alterations in middle ear impedance^[12] leading to fluctuating symptoms of “muffled” and/or “distorted” hearing; irritation of the trigeminal nerve innervating the tensor tympani muscle, leading to pain, numbness and burning sensations in and around the ear, along the cheek, neck and temporomandibular joint (TMJ) area.^[9,10]

The specific and consistent cluster of physiological symptoms

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of acoustic shock (AS) is consistent with TTTS, without underlying aural or TMJ pathology.^[2,3,9] AS can occur involuntarily after exposure to a sudden unexpected loud sound perceived as highly threatening (acoustic incident). AS becomes an acoustic shock disorder (ASD) if symptoms persist.^[2,3,9] AS was originally identified in call center staff, who are vulnerable to AS because of the increased likelihood of exposure near the ear(s) to an acoustic incident transmitted via a telephone headset.^[2,3,9] The research on AS has focused on this cohort, however acoustic incidents can occur anywhere.

Symptoms such as aural pain and a sensation of aural blockage/fullness, with no underlying aural or TMJ pathology, have been observed in tinnitus and hyperacusis patients. These symptoms have been linked to TTTS by Jastreboff and Hazell^[13] and Westcott.^[3] However, these symptoms have not been widely acknowledged or investigated in this patient population. TTTS has been more intensively investigated in temporomandibular disorder (TMD) research, with TTTS considered to be a secondary consequence of TMD and/or TMJ dysfunction, predominantly responsible for referred tinnitus, ear pain and other symptoms in and around the ear.^[4,10,11,14]

This multiclinic study aimed to investigate in a sample of tinnitus and hyperacusis patients the prevalence of:

- Symptoms consistent with TTTS
- Symptoms consistent with TTTS developing or being exacerbated by intolerable sound exposure
- AS aetiology triggering the onset of their tinnitus and/or hyperacusis.

Methods

The selection criteria for this study included all consecutive tinnitus and hyperacusis patients consulting a clinician (otolaryngologist or audiologist) specialising in and trained to provide tinnitus/hyperacusis therapy. The study was conducted simultaneously in eight clinics over the survey period (between April and November 2010): Five clinics in Australia/New Zealand, two clinics in Brazil, one clinic in Spain.

Despite some heterogeneity expected in the procedures adopted by each clinic, a detailed medical history, otoscopy and a hearing assessment were carried out on all patients.

The clinicians were asked to complete a spreadsheet recording the following information on each patient:

- Age and gender
- The presence of tinnitus and/or hyperacusis in each ear
- The degree of severity of tinnitus and/or hyperacusis in each ear. The participating clinics routinely used either the tinnitus reaction questionnaire (TRQ) or the tinnitus handicap inventory (THI) to evaluate tinnitus in their patients. Because different questionnaires were being used, each clinician was asked to categorize their

patient's tinnitus and hyperacusis as mild or moderate or severe. All the clinicians involved in this study were experienced in tinnitus/hyperacusis evaluation and therapy, so it was considered reasonable to assume there would be consistency in these severity judgements, and in most cases the TRQ/THI score was also provided.

- Whether the tinnitus/hyperacusis onset had been triggered by exposure to an acoustic incident (a loud/sudden/unexpected/intolerable sound perceived as highly threatening) and if so, whether the acoustic incident was near one or both ears.
- The presence of symptoms consistent with TTTS in each ear or side of the head (except for the symptoms of subjective vertigo and nausea, which could not be lateralized, and headache, if central):
 - o Sharp pain in the ear
 - o Dull ache in the ear
 - o A sensation of aural fullness or “blockage”
 - o A sensation of tympanic flutter
 - o A sensation of numbness around the ear
 - o A burning sensation around the ear
 - o Pain in the cheek
 - o Pain in the TMJ area
 - o Numbness/burning/pain along the side of the neck
 - o “Disordered” balance/mild vertigo (often described as ‘sway’– like being on a boat)
 - o Nausea
 - o “Muffled” hearing (when the patient reports a subjective, often fluctuating, hearing loss, but their test results indicate normal or stable hearing)
 - o Subjective “distorted” hearing (includes any fluctuating change in clarity of hearing, e.g., diplacusis)
 - o Headache.
- If symptoms consistent with TTTS were present, further data was recorded:
 - o Whether the symptoms were intermittent or constant
 - o Whether the symptoms developed with loud/intolerable sound exposure or, if constantly present, were exacerbated by loud/intolerable sound exposure.
- Hearing assessment results, recorded in each ear as a 4 frequency average of hearing thresholds at 500 Hz, 1 kHz, 2 kHz and 4 kHz. A hearing loss was considered to be present if the 4 frequency average exceeded 20 dB. A hearing loss was further categorized as sensorineural, conductive, or due to noise damage.

All patients were medically cleared of any outer ear, middle ear, inner ear, retrocochlear or TMJ dysfunction or pathology which could have caused these symptoms. Because of the possibility of an underlying pathology, those patients whose only symptoms were headache or disordered balance/vertigo or nausea or “muffled/distorted” hearing were not considered to have symptoms consistent with TTTS, and their symptom data was excluded.

If the patient had a hearing loss, “distorted” and/or “muffled

hearing” was excluded as a symptom consistent with TTTS unless it developed or was exacerbated by intolerable sound exposure.

Results

Epidemiological data

The total sample consisted of 345 patients, ranging in age from 11 to 97, with a mean age of 50.9. 179 of the total (51.9%) were male and 166 (48.1%) were female. 179 patients were from Australia/New Zealand (5 clinics), 113 from Brazil (2 clinics) and 53 from Spain (1 clinic).

Prevalence of tinnitus and hyperacusis

In the total sample:

- 49.3% (170/345) of patients had tinnitus only (T group)
- 42.3% (146/345) of patients had tinnitus plus hyperacusis (T + H group)
- 8.4% (29/345) of patients had hyperacusis only (H group).

Therefore, 91.6% (316/345) of patients in the total sample had tinnitus (T, T + H groups) and 50.7% (175/345) had hyperacusis (T + H, H groups).

Of those patients with tinnitus:

- 46.2% (146/316) had tinnitus plus hyperacusis (T + H group)
- 53.8% (170/316) had tinnitus only (T group).

Of those patients with hyperacusis:

- 83.4% (146/175) had tinnitus plus hyperacusis (T + H group)
- 16.6% (29/175) had hyperacusis only (H group).

Prevalence of acoustic incident trigger for tinnitus and/or hyperacusis onset

24.1% (83/345) patients reported having initially developed their tinnitus and/or hyperacusis following an acoustic incident trigger. Of those, 18.1% (15/83) patients had no symptoms consistent with TTTS, and 81.9% (68/83) patients had one or more symptoms consistent with TTTS [Figure 1]. Patients whose tinnitus/hyperacusis onset was triggered by an acoustic incident were highly statistically significantly more likely to develop symptoms consistent with TTTS ($P < 0.001$).

Of the 75.9% (262/345) patients who did not develop their tinnitus and/or hyperacusis following an acoustic incident trigger, 46.9% (123/262) patients had no symptoms consistent with TTTS, and 53.1% (139/262) patients had one or more symptoms consistent with TTTS.

Patients who developed symptoms consistent with TTTS were highly statistically significantly more likely to have had an acoustic incident triggering their tinnitus/hyperacusis onset than those who did not ($P < 0.001$) [Table 1].

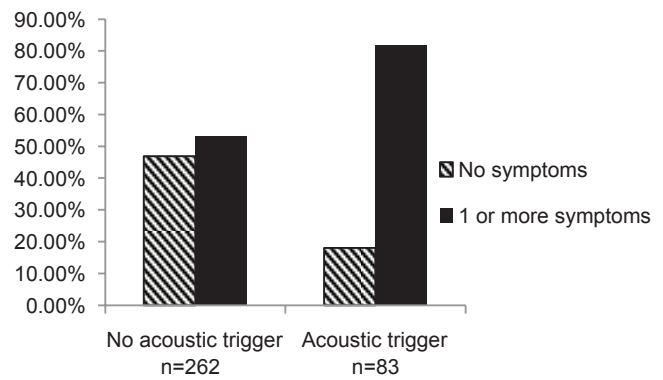


Figure 1: Prevalence of symptoms consistent with tonic tensor tympani syndrome in patients with and without an acoustic incident trigger

Table 1: Acoustic incident trigger and symptoms consistent with TTTS

	TTTS	No TTTS	Difference	95% CI for the difference		P value*
Acoustic incident	32.9%	10.9%	-22.0%	-30.2%	-13.7%	<0.001

*Test of equality of proportions. TTTS = Tonic tensor tympani syndrome, CI = Confidence interval

Acoustic shock

AS was defined as an acoustic incident trigger for tinnitus/hyperacusis onset plus the presence of one or more symptoms consistent with TTTS.

Prevalence of AS

AS was identified in:

- 19.7% (68/345) of patients in the total sample
- 26.3% (47/179) of patients in the clinics from Australia/New Zealand
- 13.2% (7/53) of patients in the clinic from Spain
- 12.4% (14/113) of patients in the clinics from Brazil.

Prevalence of hyperacusis in AS patients

83.8% (57/68) of AS patients had hyperacusis. 41.2% (114/277) of non-AS patients had hyperacusis.

Patients with AS were highly statistically significantly more likely to have hyperacusis in comparison to the non-AS patients ($P < 0.001$) [Table 2].

Table 2: Hyperacusis in AS and non-AS patients

	AS	No AS	Difference	95% CI for the difference		P value*
Hyperacusis (T+H/H groups)	83.8%	42.6%	-41.2%	-51.7%	-30.7%	<0.001

*Test of equality of proportions. CI = Confidence interval, AS = Acoustic shock, T+H = Tinnitus plus hyperacusis

Unilateral acoustic incident trigger in AS patients

33/68 of patients with AS reported a unilateral acoustic incident trigger (near to one ear, rather than both ears). Of those:

- 66.7% (22/33) had one or more unilateral symptoms consistent with TTTS only on that side
- 30.3% (10/33) had one or more bilateral symptoms to a greater degree of severity on that side
- 3.0% (1/33) had one or more bilateral symptoms of equal severity.

Therefore, 97% (32/33) of patients with a unilateral acoustic incident trigger for their AS had one or more symptoms consistent with TTTS to a greater degree on the affected side.

Patients with a unilateral acoustic incident exposure triggering their AS were highly statistically significantly more likely to have one or more symptoms consistent with TTTS to a greater degree on that side ($P < 0.001$) [Table 3]. The likelihood was not quite as high ($P < 0.080$) for patients with a unilateral acoustic incident exposure to only have one or more symptoms on the affected side [Table 4].

Table 3: AS patients with a unilateral acoustic incident trigger

	Bilateral symptoms greater on the affected side	95% CI for percentage	P value*
AS unilateral trigger	97.0%	84.2 99.9	<0.001

*Test of proportion equal to 0.5. CI = Confidence interval, AS = Acoustic shock

Table 4: AS patients with a unilateral acoustic incident trigger

	Symptoms only on the affected side	95% CI for percentage	P value*
AS unilateral trigger	66.7%	48.2 82.0	0.080

*Test of proportion equal to 0.5. CI = Confidence interval, AS = Acoustic shock

Prevalence of symptoms consistent with TTTS

Prevalence of symptoms consistent with TTTS in the whole sample

- 40.0% (138/345) of patients had no symptoms
- 12.5% (43/345) of patients had 1 symptom
- 47.5% (164/345) of patients had ≥ 2 symptoms.

Therefore, 60.0% (207/345) of patients in this sample had one or more symptoms consistent with TTTS [Figure 2].

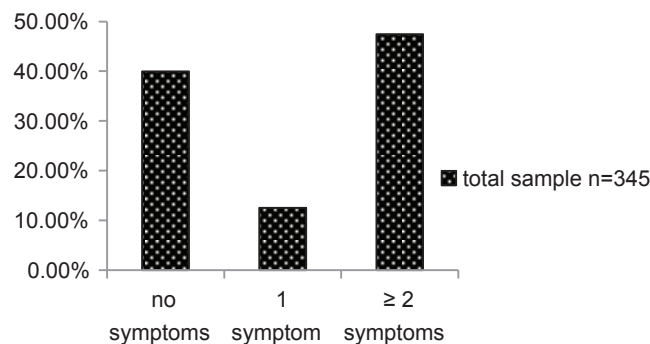


Figure 2: Prevalence of symptoms consistent with tonic tensor tympani syndrome in the total sample

Relationship between symptoms consistent with TTTS

A statistical test of independence of symptoms shows that the distribution of symptoms is significantly different from what would be expected if the symptoms were unrelated ($P < 0.001$). Patients with symptoms consistent with TTTS were significantly more likely to have ≥ 2 symptoms, rather than a single symptom [Table 5].

Table 5: Independence of symptoms

	Observed total	If independent expected total	P value*
No symptoms	138	115	<0.001
One symptom	43	126	
Two or more symptoms	164	104	
Total	345	345	

*Test of independence of symptoms

Prevalence of symptoms consistent with TTTS in the tinnitus only group (T group) compared to the hyperacusis groups (T + H, H groups)

In the tinnitus only patients (T group, $n = 170$):

- 59.4% (101/170) of patients had no symptoms
- 9.4% (16/170) of patients had 1 symptom
- 31.2% (53/170) of patients had ≥ 2 symptoms.

Therefore, 40.6% (69/170) of patients with tinnitus only (T group) had one or more symptoms consistent with TTTS [Figure 3].

In the hyperacusis patients (T + H, H groups, $n = 175$):

- 18.9% (33/175) of patients had no symptoms
- 15.4% (27/175) of patients had 1 symptom
- 65.7% (115/175) of patients had ≥ 2 symptoms.

Therefore, 81.1% (142/175) of patients with hyperacusis (T + H, H groups) had one or more symptoms consistent with TTTS [Figure 3].

Patients with tinnitus only (T group) were highly statistically significantly more likely to have no symptoms consistent

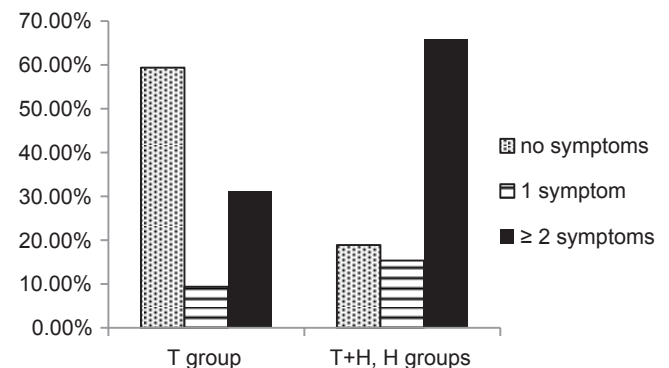


Figure 3: Prevalence of symptoms consistent with tonic tensor tympani syndrome in T group and tinnitus plus hyperacusis, H groups

with TTTS than patients with hyperacusis (T + H, H groups) [Table 6].

Patients with hyperacusis (T + H, H groups) were highly statistically significantly more likely to report one or more symptoms consistent with TTTS than patients with tinnitus alone (T group) $P < 0.001$ [Table 7].

Table 6: Symptom prevalence in T group and T+H, H groups

	T group	T+H, H groups	Difference	95% CI for the difference		P value*
No symptoms	59.4%	18.9%	-40.6%	-49.9%	-31.2%	<0.001

*Test of equality of proportions. CI = Confidence interval, T+H = Tinnitus plus hyperacusis

Table 7: Symptom prevalence in T group and T+H, H groups

	T group	T+H, H groups	Difference	95% CI for the difference		P value*
Any symptoms	40.6%	81.1%	40.6%	31.2%	49.9%	<0.001

*Test of equality of proportions. CI = Confidence interval, T+H = Tinnitus plus hyperacusis

Unilateral hyperacusis

In view of the finding that patients with hyperacusis were more likely to report symptoms consistent with TTTS, patients reporting unilateral hyperacusis were analysed.

29.7% (52/175) of hyperacusis patients had unilateral hyperacusis (T + H, H groups). Of those:

- 63.5% (33/52) of patients had one or more symptoms only on that side
- 21.2% (11/52) of patients had one or more bilateral symptoms to a greater degree on that side
- 7.7% (4/52) of patients had one or more bilateral symptoms equally in both ears
- 1.9% (1/52) of patients had one or more bilateral symptoms to a greater degree on the opposite side
- 5.8% (3/52) of patients had no symptoms.

Therefore, 84.6% (44/52) of patients with unilateral hyperacusis had one or more symptoms consistent with TTTS to a greater degree on the affected side.

Patients with unilateral hyperacusis were highly statistically significantly more likely to have one or more symptoms consistent with TTTS to a greater degree on that side ($P < 0.001$) [Table 8]. The likelihood was not quite as high for patients with unilateral hyperacusis to only have one or more symptoms on the affected side ($P = 0.070$) [Table 9].

Prevalence of specific symptoms consistent with TTTS in the total sample

The most common symptoms for the total sample were aural fullness (33.0%); headache (21.2%); disordered balance (20.3%); TMJ pain (20.0%); pain along the side of the neck (20.0%); tympanic flutter (19.1%); dull ache in ear (19.1%); sharp pain in ear (17.7%); “muffled” hearing (12.5%); and

Table 8: Unilateral hyperacusis patients

Bilateral symptoms greater on affected side %	95% CI for percentage		P value*
84.6	71.9	93.1	<0.001

*Test of proportion equal to 0.5. CI = Confidence interval

Table 9: Unilateral hyperacusis patients

Symptoms only on affected side %	95% CI for percentage		P value*
63.5	49.0	76.4	0.070

*Test of proportion equal to 0.5. CI = Confidence interval

“distorted” hearing (11.0%) [Table 10].

Prevalence of specific symptoms consistent with TTTS in the Tinnitus only (T group) compared to hyperacusis groups (T + H, H groups)

The most common symptoms for the tinnitus only patients (T group) were TMJ pain (14.7%); pain along the side of the neck (14.7%); headache (13.5%); aural fullness (12.9%); and disordered balance (11.8%) [Figure 4, Table 10].

The most common symptoms for the hyperacusis patients (T + H, H groups) were aural fullness (52.6%); headache (28.6%); dull ache in ear (28.6%); disordered balance (28.6%); tympanic flutter (28.0%); sharp pain in ear (27.4%); TMJ pain (25.1%); and pain along the side of the neck (25.1%) [Figure 4, Table 10].

Each symptom was statistically more likely to be present in those patients with hyperacusis (T + H, H groups), in comparison to those with tinnitus alone (T group). This difference was highly statistically significant ($P < 0.001$) for symptoms of: Aural fullness, headache, disordered balance, tympanic flutter, dull ache in the ear and sharp pain in ear [Table 10].

Prevalence of symptoms consistent with TTTS in relation to the degree of severity of tinnitus and hyperacusis

Each clinician was asked to categorise their patient’s tinnitus and hyperacusis as mild or moderate or severe. The sample was broken into two groups, with tinnitus and hyperacusis severity rated and analysed separately.

Tinnitus severity

The total number of patients with tinnitus (T, T + H groups) was 316/345. Tinnitus severity was rated as mild, moderate or severe.

Mild tinnitus: Total number of patients with mild tinnitus was 101/316 (32.0%):

- 57.4% (58/101) of patients had no symptoms
- 12.9% (13/101) of patients had 1 symptom
- 29.7% (30/101) of patients had ≥ 2 symptoms.

Therefore, 42.6% (43/101) of patients with mild tinnitus had one or more symptoms consistent with TTTS [Figure 5].

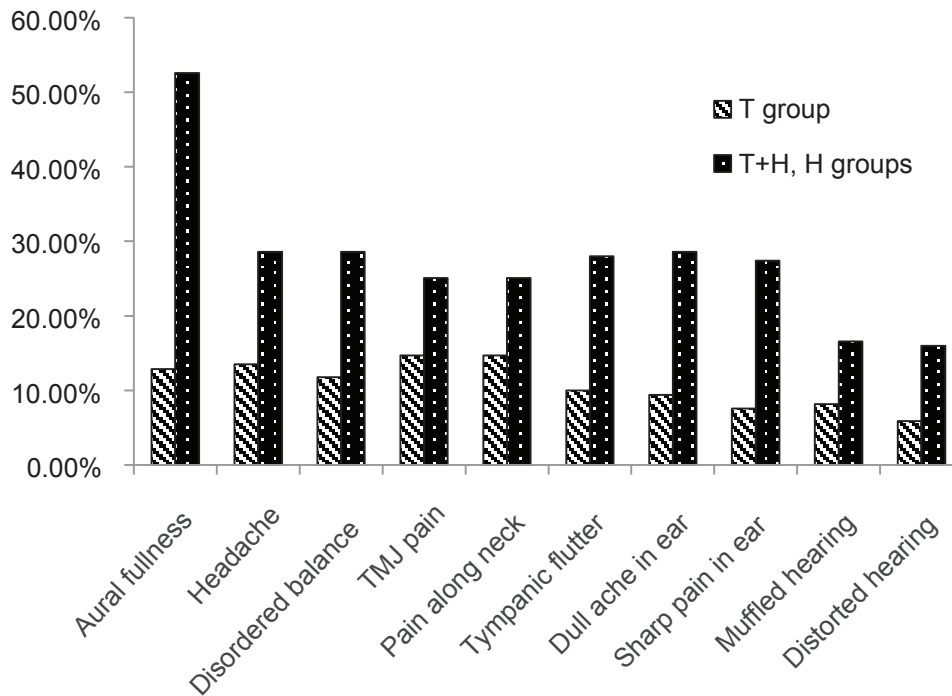


Figure 4: Individual symptom prevalence in T group compared to tinnitus plus hyperacusis, H groups

Table 10: Individual symptom prevalence in T group compared to T+H, H groups

Symptom prevalence in total sample	Prevalence		Difference %	95% CI for the difference %		P value*
	T group n=170%	T+H, H groups n=175%				
Aural fullness/blockage (114/345, 33.0%)	12.9 (22/170)	52.6 (92/175)	39.6	30.7	48.6	<0.001
Headache (73/345, 21.2%)	13.5 (23/170)	28.6 (50/175)	15.0	6.6	23.5	<0.001
Disordered balance (70/345, 20.3%)	11.8 (20/170)	28.6 (50/175)	16.8	8.5	25.1	<0.001
TMJ pain (69/345, 20.0%)	14.7 (25/170)	25.1 (44/175)	10.4	2.1	18.8	0.016
Pain along side of the neck (69/345, 20.0%)	14.7 (25/170)	25.1 (44/175)	10.4	2.1	1.8	0.016
Tympanic flutter (66/345, 19.1%)	10.0 (17/170)	28.0 (49/175)	18.0	10.0	26.0	<0.001
Dull ache in ear (66/345, 19.1%)	9.4 (16/170)	28.6 (50/175)	19.2	11.2	27.2	<0.001
Sharp pain in ear (61/345, 17.7%)	7.6 (13/170)	27.4 (48/175)	19.8	12.1	27.5	<0.001
Muffled hearing (43/345, 12.5%)	8.2 (14/170)	16.6 (29/175)	8.3	1.4	15.2	0.022
Distorted hearing (38/345, 11.0%)	5.9 (10/170)	16.0 (28/175)	10.1	3.6	0.2	0.003

*Test of equality of proportions. CI = Confidence interval, TMJ = Temporomandibular joint, T+H = Tinnitus plus hyperacusis

Moderate tinnitus: Total number of patients with moderate tinnitus was 115/316 (36.4%):

- 32.2% (37/115) of patients had no symptoms
- 13.0% (15/115) of patients had 1 symptom
- 54.8% (63/115) of patients had ≥ 2 symptoms.

Therefore, 67.8% (78/115) of patients with moderate tinnitus had one or more symptoms consistent with TTTS [Figure 5].

Severe tinnitus: Total number of patients with severe tinnitus was 100/316 (31.7%):

- 32% (32/100) of patients had no symptoms
- 10% (10/100) of patients had 1 symptom
- 58% (58/100) of patients had ≥ 2 symptoms.

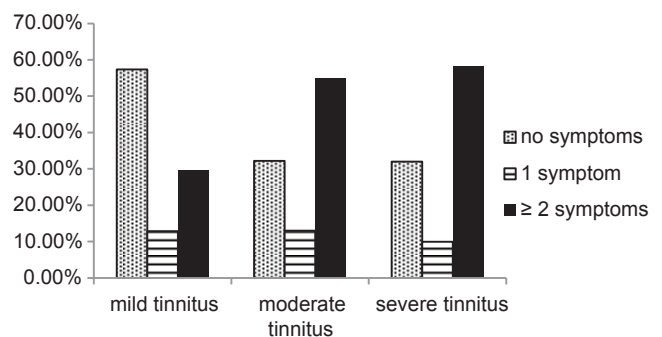


Figure 5: Effect of tinnitus severity on the prevalence of symptoms consistent with tonic tensor tympani syndrome

Therefore, 68% (68/100) of patients with severe tinnitus had one or more symptoms consistent with TTTS [Figure 5].

Patients with tinnitus were highly statistically significantly more likely to have two or more symptoms consistent with TTTS as their tinnitus severity increased ($P < 0.001$) [Table 11].

Table 11: Tinnitus severity and prevalence of symptoms consistent with TTTS

	Mild	Moderate	Severe	Total patients	<i>P</i> value*
No symptoms	58	37	32	127	<0.001
One symptom	13	15	10	38	
Two or more symptoms	30	63	58	151	
Total	101	115	100	316	

* χ^2 test of association. TTTS = Tonic tensor tympani syndrome

Hyperacusis severity

The total number of patients with hyperacusis (T + H, H groups) was 173/345 (the severity data had not been recorded on two patients, so they were omitted). Hyperacusis severity was rated as mild, moderate or severe.

Mild hyperacusis: Total number of patients with mild hyperacusis was 64/173 (37.0%):

- 26.6% (17/64) of patients had no symptoms
- 20.3% (13/64) of patients had 1 symptom
- 53.1% (34/64) of patients had ≥ 2 symptoms.

Therefore, 73.4% (47/64) of patients with mild hyperacusis had one or more symptoms consistent with TTTS [Figure 6].

Moderate hyperacusis: Total number of patients with moderate hyperacusis was 63/173 (36.4%):

- 17.5% (11/63) of patients had no symptoms
- 15.9% (10/63) of patients had 1 symptom
- 66.7% (42/63) of patients had ≥ 2 symptoms.

Therefore, 82.5% (52/63) of patients with moderate hyperacusis had one or more symptoms consistent with TTTS [Figure 6].

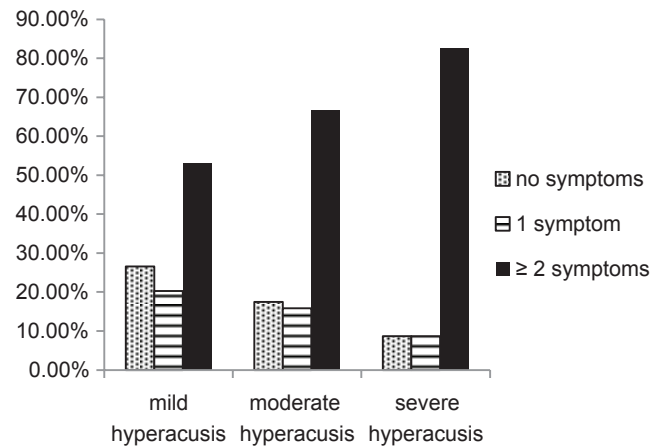


Figure 6: Effect of hyperacusis severity on the prevalence of symptoms consistent with tonic tensor tympani syndrome

Severe hyperacusis: Total number of patients with severe hyperacusis was 46/173 (26.6%):

- 8.7% (4/46) of patients had no symptoms
- 8.7% (4/46) of patients had 1 symptom
- 82.6% (38/46) of patients had ≥ 2 symptoms.

Therefore, 91.3% (42/46) of patients with severe hyperacusis had one or more symptoms consistent with TTTS [Figure 6].

The more severe the hyperacusis, the more likely the patient would have ≥ 2 symptoms consistent with TTTS. However, as the prevalence of symptoms consistent with TTTS is high for all hyperacusis patients, the differences between the mild, moderate and severe hyperacusis groups are less significant ($P = 0.032$) [Table 12].

Table 12: Hyperacusis severity and prevalence of symptoms consistent with TTTS

	Mild	Moderate	Severe	Total patients	<i>P</i> value*
No symptoms	17	11	4	32	0.032
One symptom	13	10	4	27	
Two or more symptoms	34	42	38	114	
Total	64	63	46	173**	

**Severity data not recorded on two patients so omitted. * χ^2 test of association. TTTS = Tonic tensor tympani syndrome

Symptom development and exacerbation from intolerable sound exposure

Figure 7 shows the prevalence of specific symptoms consistent with TTTS, which developed or, if frequently or constantly present, were exacerbated by loud/intolerable sound exposure, in tinnitus only patients (T group) compared to hyperacusis patients (T + H, H groups).

The most common symptoms in T group patients, which developed or were exacerbated by loud/intolerable sound exposure [Figure 7, Table 13]:

- “Distorted” hearing 80.0% (i.e. 80% of patients with “distorted” hearing reported this symptom developed or was exacerbated by loud/intolerable sound exposure)
- Tympanic flutter 35.5%
- Sharp pain in ear 30.8%
- Aural fullness 22.7%
- “Muffled” hearing 21.4%.

The most common symptoms in T + H, H group patients, which developed or were exacerbated by loud/intolerable sound exposure [Figure 7, Table 13]:

- “Distorted” hearing 78.6%
- “Muffled” hearing 75.9%
- Dull ache in ear 74.0%
- Sharp pain in ear 66.7%
- Tympanic flutter 59.2%
- Aural fullness 53.3%.

Patients with hyperacusis (T + H, H groups) with symptoms

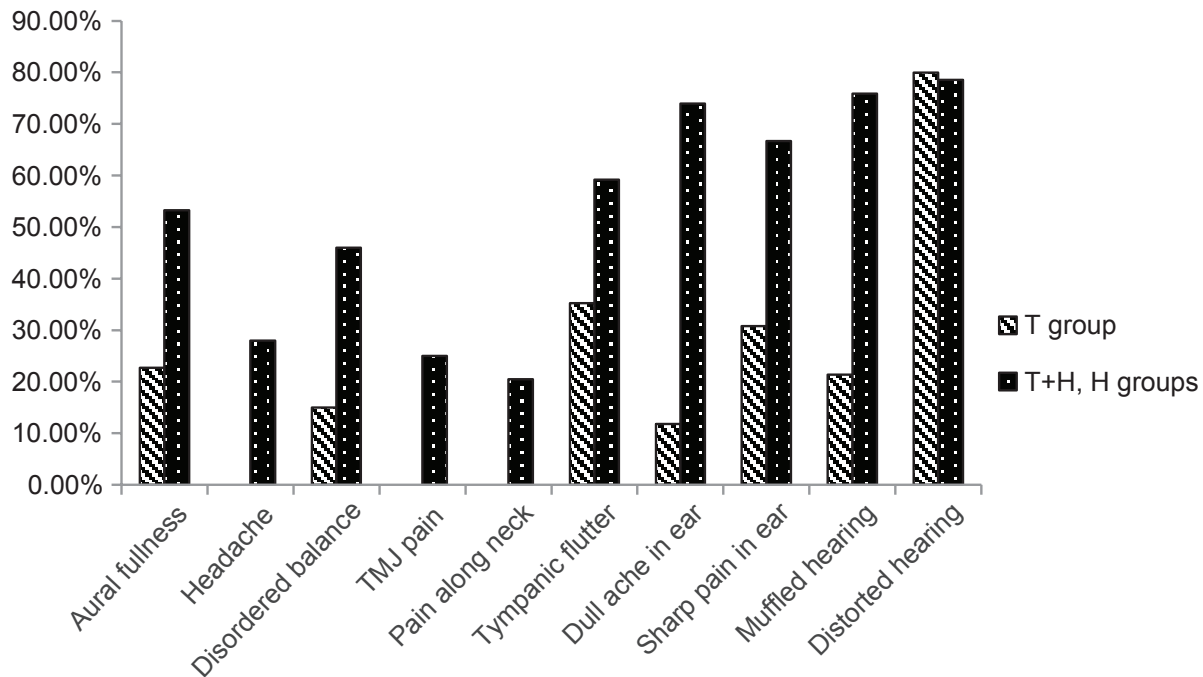


Figure 7: Patients with symptom, reporting development or exacerbation of the symptom from intolerable sound exposure

Table 13: Patients with symptom, reporting development or exacerbation of the symptom from intolerable sound exposure

Symptom	%age with development or exacerbation		Difference	95% CI for difference		P value*
	T	T+H, H				
Aural fullness/blockage	22.7%	53.3%	30.5%	10.3%	50.8%	0.016
Headache	0.0%	28.0%	28.0%	15.6%	40.4%	<0.001
Disordered balance	15.0%	46.0%	31.0%	10.1%	51.9%	0.027
TMJ pain	0.0%	25.0%	25.0%	12.2%	37.8%	0.005
Pain along side of the neck	0.0%	20.5%	20.5%	8.5%	32.4%	0.021
Tympanic flutter	35.3%	59.2%	23.9%	-2.7%	50.4%	0.1
Dull ache in ear	11.8%	74.0%	62.2%	42.7%	81.8%	<0.001
Sharp pain in ear	30.8%	66.7%	35.9%	7.5%	64.3%	0.027
Muffled hearing	21.4%	75.9%	54.4%	27.9%	81.0%	0.001
Distorted hearing	80.0%	78.6%	-1.4%	30.5%	27.7%	>0.9

*Test of equality of proportions, CI = Confidence interval, TMJ = Temporomandibular joint, T+H = Tinnitus plus hyperacusis

consistent with TTTS of: Aural fullness, headache, disordered balance, TMJ pain, pain along the side of the neck, dull ache in the ear and sharp pain in the ear were significantly more likely than patients with tinnitus only (T group) to have their symptoms develop or become exacerbated by intolerable sound exposure. This difference was highly statistically significant for symptoms of headache ($P < 0.001$), dull ache in ear ($P < 0.001$), muffled hearing ($P = 0.001$) and TMJ pain ($P = 0.005$) [Table 13].

Prevalence of symptoms consistent with TTTS in patients with normal hearing and with hearing loss

Normal hearing: Total number of patients with normal hearing was 173/345 (50.1%):

- 35.8% (62/173) of patients had no symptoms
- 11.6% (20/173) of patients had 1 symptom
- 52.6% (91/173) of patients had ≥ 2 symptoms.

Therefore, 64.16% (111/173) patients with normal hearing had one or more symptoms consistent with TTTS [Figure 8].

Hearing loss (>20 dB 4 frequency average): Total number of patients with a hearing loss was 172/345 (49.9%):

- 41.9% (72/172) of patients had no symptoms
- 12.8% (22/172) of patients had 1 symptom
- 45.4% (78/172) of patients had ≥ 2 symptoms.

Therefore, 58.1% (100/172) of patients with a hearing loss had one or more symptoms consistent with TTTS [Figure 8].

There was no significant difference ($P = 0.4$) in the prevalence of symptoms consistent with TTTS in patients with normal hearing and in those with a hearing loss [Table 14].

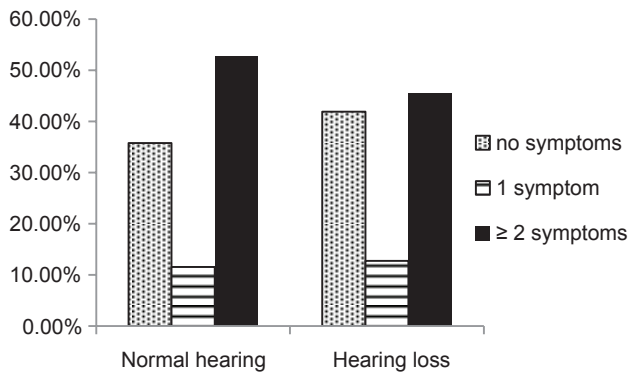


Figure 8: Prevalence of symptoms in normal hearing and hearing loss

Table 14: Prevalence of symptoms in normal hearing and hearing loss

	Normal hearing	Hearing loss	Total	<i>P</i> value*
No symptoms	62	72	134	0.4
One symptom	20	22	42	
Two or more symptoms	91	78	169	
Total	173	172	345	

* χ^2 test of association

Discussion

Tinnitus and hyperacusis prevalence

In this sample of tinnitus and hyperacusis patients attending the eight participating clinics, 49.3% of patients had tinnitus only, 42.3% of had tinnitus plus hyperacusis and 8.4% of patients had hyperacusis only.

91.6% of patients in the total sample had tinnitus and 50.7% had hyperacusis. Of those patients with tinnitus, 46.2% had tinnitus plus hyperacusis and 53.8% had tinnitus only. Of those patients with hyperacusis, 83.4% had tinnitus plus hyperacusis and 16.6% had hyperacusis only.

These prevalence figures are consistent with the consensus that about 40% of people with troublesome tinnitus have some degree of hyperacusis,^[15] although estimates of the prevalence of hyperacusis in patients attending a tinnitus clinic with a primary complaint of tinnitus have varied from about 40% up to 79%.^[16] These figures are also consistent with previous research showing 86% of patients with hyperacusis report tinnitus.^[17]

Symptoms consistent with TTTS: Evidence for syndrome

In this study, statistical analysis of the symptoms consistent with TTTS supports their belonging to a cluster or syndrome [Table 5]. A statistical test of independence of symptoms shows that the distribution of symptoms consistent with TTTS is significantly different from what would be expected if the symptoms were unrelated ($P < 0.001$). Additionally, patients with these symptoms were significantly more likely to have ≥ 2 symptoms, rather than a single symptom.

Symptoms consistent with TTTS: Acoustic incident trigger and symptom prevalence

24.1% of the cohort reported having initially developed their tinnitus and/or hyperacusis following exposure to a sudden unexpected loud sound perceived as highly threatening (acoustic incident). Of those, 81.9% reported one or more symptoms consistent with TTTS [Figure 1].

Patients whose tinnitus/hyperacusis onset was triggered by an acoustic incident were highly statistically significantly more likely to develop one or more symptoms consistent with TTTS ($P < 0.001$). Conversely, patients who developed one or more symptoms consistent with TTTS were more likely to have had an acoustic incident triggering their tinnitus/hyperacusis onset than those who did not ($P < 0.001$) [Table 1].

Acoustic shock (AS) Prevalence and link with hyperacusis

AS was defined as an acoustic incident trigger for tinnitus/hyperacusis onset plus the presence of one or more symptoms consistent with TTTS. AS was identified in 19.7% (68/345) of this sample and in all the participating countries, indicating that AS is a universal phenomenon. Much of the AS research has been carried out in Australia, and the higher prevalence of AS in the clinics from Australia/New Zealand (26.3% of their total) in comparison to the clinic from Spain (13.2%) and the clinics from Brazil (12.4%) reflects an increased level of AS awareness, diagnosis and referral for evaluation/therapy among tinnitus/hyperacusis clinicians, the medical profession and in the community.

83.8% of AS patients had hyperacusis. 41.2% (114/277) of non-AS patients had hyperacusis. Patients with AS were highly statistically significantly more likely to have hyperacusis in comparison to the non-AS group ($P < 0.001$, Table 2).

97% (32/33) of patients with a unilateral acoustic incident trigger for their AS had one or more symptoms consistent with TTTS to a greater degree on the affected side ($P < 0.001$, Table 3).

Symptoms consistent with TTTS: Prevalence and link with hyperacusis

One or more symptoms consistent with TTTS were reported in 60% (207/345) of the total sample [Figure 2]. This is a substantial finding, at the very least reflecting the likelihood of symptom-related annoyance in many tinnitus and hyperacusis patients, and for a large number representing significant levels of suffering, anxiety and distress.

While a substantial proportion of the total sample report symptoms consistent with TTTS, these results indicate a particularly strong association between hyperacusis and the development of symptoms. One or more symptoms consistent

with TTTS were reported in 81.1% of those patients with hyperacusis and in 40.6% of those with tinnitus only [Figure 3, Tables 6 and 7]. This was a highly statistically significant difference between tinnitus only and hyperacusis patients ($P \leq 0.001$).

The more severe the tinnitus and hyperacusis, the more likely the patient would report symptoms consistent with TTTS [Figures 5 and 6, Tables 11 and 12]. Almost all patients (91.3%) with severe hyperacusis report at least one symptom consistent with TTTS, and 82.6% report ≥ 2 symptoms [Figure 6]. An interesting finding in this study was that patients with unilateral hyperacusis were highly statistically significantly more likely to have one or more symptoms consistent with TTTS to a greater degree on that side ($P < 0.001$, Table 8).

Many patients with symptoms consistent with TTTS, particularly those with hyperacusis, reported that their symptoms developed or, if frequently or constantly present, were exacerbated by loud/intolerable sound exposure [Figure 7, Table 13]. For example, for those patients with the most common symptom of aural fullness ($n = 114$ or 33.0% of the total sample), 22.7% of tinnitus only patients and 53.3% of hyperacusis patients reported that this symptom develops or is exacerbated by intolerable sound exposure.

People with moderate/severe hyperacusis are likely to find a proportion of everyday sounds unpleasant, difficult to tolerate or intolerable. Hyperacusis patients with symptoms consistent with TTTS of: Aural fullness, headache, disordered balance, TMJ pain, pain along the side of the neck, dull ache in the ear and sharp pain in the ear were significantly more likely than patients with tinnitus only to have their symptoms develop or become exacerbated by loud/intolerable sound exposure. This difference was highly statistically significant for symptoms of headache, dull ache in ear, muffled hearing and TMJ pain [Table 13].

A higher prevalence of aural pain (dull ache in ear 28.6%; sharp pain in ear 27.4%) was reported by hyperacusis patients in this sample [Table 10], compared to the 12.5% of persons with hyperacusis reporting pain in the prevalence study conducted by Andersson *et al.*^[16] The hyperacusis patients in this sample were sufficiently affected by their tinnitus and/or hyperacusis to seek therapy, whereas those in Andersson's sample had responded to an online questionnaire or were randomly contacted by post.

Subjective symptoms of “muffled” hearing (when the patient reports a fluctuating hearing loss, but their test results indicate normal or stable hearing); and “distorted” hearing (any fluctuating change in clarity of hearing) are likely to be due to the minor alterations in the middle ear impedance associated with TTTS.^[12] The results for hyperacusis patients show that 78.6% with “distorted” hearing and 75.9% with “muffled” hearing reported that these symptoms developed or

were exacerbated by intolerable sound exposure [Table 13]. While these symptoms were less common in patients with tinnitus only, 80.0% with “distorted” hearing reported that it developed or was exacerbated by intolerable sound exposure.

There is evidence that middle ear muscle function is influenced by the serotonergic system, consistent with a link between the emotional state and middle ear muscle contraction.^[16,18] The central mediation of the reflex threshold for tensor tympani muscle activity is further supported in this study by:

- The association demonstrated between hyperacusis and the high prevalence of symptoms consistent with TTTS
- The prevalence of symptom development/exacerbation following intolerable sound exposure, consistent with a stress/anxiety/threat response
- The unilateral preponderance of symptoms in patients with unilateral hyperacusis and those who reported exposure to a unilateral acoustic incident exposure triggering their AS. 84.6% of patients with unilateral hyperacusis had one or more symptoms consistent with TTTS to a greater degree on the affected side ($P < 0.001$) [Table 8]. 97.0% (32/33) of patients with a unilateral acoustic incident trigger for their AS had one or more symptoms consistent with TTTS to a greater degree on the affected side ($P < 0.001$) [Table 3].

Symptoms consistent with TTTS: Normal hearing and hearing loss

The results showed there was no significant difference in the prevalence of symptoms consistent with TTTS in patients with normal hearing and in those with a hearing loss [Figure 8, Table 14], so further analysis of sensorineural, conductive, or noise damage aetiology was not carried out. A hearing loss was considered to be present if the 4 frequency average of hearing thresholds at 500 Hz, 1 kHz, 2 kHz and 4 kHz exceeded 20 dB. While a hearing assessment typically includes obtaining hearing thresholds up to 8 kHz, this 4 frequency average is a standard method in clinical audiological practice for calculating the presence of a hearing loss. It is acknowledged, however, that tinnitus and hyperacusis patients with a hearing loss for the higher frequencies only will be missed using this criterion. Further studies investigating the presence of a hearing loss for the high frequencies in greater depth are recommended to fully explore any link between hearing loss and the presence of symptoms consistent with TTTS.

TTTS as a secondary consequence of TMD and/or TMJ dysfunction

Ramirez-Aristeguieta *et al.* have investigated the anatomical and physiological connections in TMD patients with secondary aural symptoms and the central and peripheral mechanisms involved.^[4,10,11] The authors carried out a meta-analysis of data from 12,436 patients in 49 peer-reviewed publications, proposing a range of muscular, bone communication and

neural scenarios to explain the relationship between aural symptoms (otalgia, tinnitus, vertigo, subjective hearing loss and aural fullness) exacerbated by dysfunctional mouth and jaw dynamics.^[10] They placed a strong emphasis on tensor tympani muscle involvement and associated trigeminal nerve dysfunction.

The tensor tympani muscle is innervated by the motor portion of the mandibular branch of the trigeminal nerve, and the authors consider that TTTS can lead to, and in an efferent pathway be caused by, an abnormal stimulation of the trigeminal nerve,^[10] This can lead to a chronic irritation of the trigeminal nerve, as well as other cranial and cervical sensory nerves of the ear and periauricular region.^[10] Central sensitization can develop from the resultant chronic pain, leading to an expansion of the perceived peripheral pain and resulting in the typical symptoms of severe TMD.^[9]

Clinical implications: TTTS as a primary or secondary phenomenon

The presence of symptoms consistent with TTTS, including TMJ pain, does not imply TMJ dysfunction, as shown in this patient sample who were medically cleared of aural and TMJ pathology or dysfunction. These findings suggest an alternative etiologic pathway triggering TTTS as a primary phenomenon in tinnitus, hyperacusis and ASD patients in comparison to secondary TTTS in patients with TMD and/or TMJ dysfunction.

When TTTS is a secondary consequence of TMD and/or TMJ dysfunction, the TTTS symptoms do not tend to escalate and hyperacusis is not usually present. When TTTS is a primary phenomenon in tinnitus/hyperacusis/ASD patients, clinical observation supported by the results obtained implies a relationship between TTTS and a trauma response to sound, with the symptoms escalating in association with an escalation in tinnitus, hyperacusis and anxiety.^[9]

Conversely, severe persistent TTTS symptoms in ASD patients can lead to TMD as a secondary consequence of central sensitisation from chronic trigeminal nerve pain.^[9,19]

Clinical, medico-legal and military implications of AS

The potential severity and persistence of symptoms consistent with TTTS in ASD patients has significant clinical and medico-legal implications.^[9] The global growth of call centres can be expected to lead to an increase in ASD prevalence.

Hearing loss, tinnitus and post-traumatic stress disorder (PTSD) are the most prevalent service-connected disabilities among U.S. veterans.^[20] ASD development from exposure to threatening, unexpected, loud sounds in close proximity, such as explosions and weapon fire, can be expected to occur in active service, either with or without noise damage causing hearing loss. Apart from persistent TTTS symptoms, severe

ASD almost always includes hyperacusis (as shown in this study), often includes tinnitus, and can lead to PTSD.^[9]

Tinnitus and hyperacusis therapists as well as general medical practitioners, otolaryngologists, occupational physicians, TMD specialists, neurologists and trauma psychologists/psychiatrists are therefore increasingly likely to encounter some or all of the cluster of symptoms consistent with TTTS as a result of ASD in their patients.^[9]

Clinical implications: Management of symptoms consistent with TTTS

Andersson and Baguley postulated that fear of the pain experience is one of the factors playing a significant role in explaining avoidance of sounds by people with hyperacusis.^[16] Their observation that some of their hyperacusis clients suffer tremendous fear of any situation where they risk sound-induced pain will be familiar to clinicians providing hyperacusis therapy.^[16,21] However, Andersson and Baguley did not offer an explanation for the physiological basis of this pain.^[16] In this sample, 74.0% of hyperacusis patients with the symptom of dull ache in the ear and 66.7% of hyperacusis patients with the symptom of sharp pain in the ear reported these symptoms developed or were exacerbated by loud/intolerable sound exposure [Table 13]. These patients can readily believe that their ears are no longer able to physically tolerate these sounds, or that these sounds are causing damage to their ears or hearing, and should be avoided.^[22] This unpleasant consequence of exposure to sounds perceived as intolerable and the threat of further exposure is likely to play a significant role in tinnitus escalation and the development, persistence and escalation of hyperacusis.

Medical investigation in patients with symptoms consistent with TTTS is likely to be clear of underlying pathology in view of their subjective nature. If they are not given an explanation of the likely TTTS aetiology of their symptoms, the resultant bewilderment, anxiety and distress can play a role not only in tinnitus and hyperacusis escalation but also in limiting the degree of efficacy of therapeutic intervention. An explanation of TTTS is a powerful therapeutic tool, providing reassurance and can help limit tinnitus/hyperacusis escalation.^[9,22] Effective hyperacusis desensitisation therapy reduces the frequency and severity of symptoms consistent with TTTS.^[9,22]

Loudness discomfort levels (LDLs) are in general use as a measure of hyperacusis as well as tracking the efficacy of hyperacusis desensitisation therapy. For patients with moderate/severe hyperacusis, exposure to sounds at their loudness tolerance limits is threatening and unpleasant. This can affect the accuracy of the results obtained, as well as potentially kindling an increase in their tinnitus, hyperacusis and symptoms consistent with TTTS. Documenting the presence of symptoms consistent with TTTS and tracking a

reduction in symptoms over time offers a less invasive method than LDL testing for assessing hyperacusis severity and evaluating the efficacy of hyperacusis desensitisation therapy.

Conclusion

The high prevalence of symptoms consistent with TTTS in this sample suggests they can readily develop as a primary phenomenon in patients with tinnitus, and more particularly in those with hyperacusis. These results support a central relationship between tinnitus, hyperacusis and TTTS, with further research indicated to explore this relationship and the efferent pathway triggering TTTS.

TTTS offers an explanation for the aural pain reported by many hyperacusis patients, often triggered or aggravated by intolerable sound exposure. Symptoms consistent with TTTS are subjective and can cause high levels of anxiety. This can lead to tinnitus escalation, the development and escalation of hyperacusis, and limit the efficacy of tinnitus/hyperacusis therapy. These symptoms should be routinely evaluated in history taking, de-mystified to patients to provide reassurance, and treated accordingly.

These results indicate that AS is a world-wide phenomenon, with significant clinical, medico-legal and military diagnostic/rehabilitation implications. It is recommended that evaluation of an acoustic incident at the time of tinnitus/hyperacusis onset is routinely carried out in history taking with tinnitus and hyperacusis patients.

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