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Review Article

The relationship between obstructive sleep apnea with hearing and balance: A scoping review

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ABSTRACT

Background: Obstructive sleep apnea (OSA) has been linked to multiple co-morbidities, and some research points to a potential relationship between OSA and hearing and balance dysfunction, and the benefits of continuous positive airway pressure (CPAP) treatment.
Objective: The present study was undertaken as a scoping review of research on OSA and its impact on hearing and balance function in adults and children and whether the treatment of CPAP would affect hearing and balance function.
Method: Online databases were used to identify 45 papers published that used hearing and balance assessments concerning OSA and CPAP therapy as a primary outcome. The secondary outcome was the subjective perception through validated questionnaires.
Results: Whilst the data on the effect of OSA on middle ear function remains inconclusive, most papers demonstrate an increase in hearing thresholds, an absence of otoacoustic emissions (OAE) and delayed auditory brainstem response (ABR) in adults with OSA. Nystagmus and abnormal vestibular evoked myogenic potentials (VEMPs) were observed in the small number of papers. The positive pressure from CPAP significantly and transiently increases middle ear pressure, however, its effects on other auditory regions and the vestibular system remains inconclusive. Research on hearing and balance function in children with OSA is limited.
Conclusions: Narrow assessments of hearing and balance are not sufficient to understand the nature of hearing and balance function in OSA patients and the effect of CPAP therapy. More comprehensive assessments are necessary to observe peripheral and central changes in the auditory and vestibular pathways.

1. Introduction

Obstructive sleep apnea (OSA) is the most common sleep-disordered breathing condition and is characterized by a partial or complete collapse of the upper airway. When this occurs, the air is unable to pass efficiently through the upper airway, resulting in a decrease in blood oxygen saturation. Due to this intermittent hypoxia, the central nervous system responds, and the patient wakes up to resume normal breathing. Therefore, the patient has fragmented sleep, which can have significant chronic health effects. OSA is a prevalent global health issue affecting approximately 13–33% of adult men and 6–19% of adult women [1]. The

Abbreviations: ABR, Auditory brainstem response; AHI, Apnea hypopnea index; BRIEF-P, Behavioural rating of executive function - preschool questionnaire; CMEDHQ, Childhood middle ear disease and hearing questionnaire; CPAP, Continuous positive airway pressure; cVEMP, Cervical VEMPS; DPOAE, Distortion product OAEs; ETDQ-7, Eustachian tube dysfunction questionnaire-7; ETS-7, Eustachian tube scores; GERD, Gastroesophageal reflux disease; MEP, Middle ear pressure; MoCA, Montreal cognitive assessment; OAE, Otoacoustic emissions; OSA, Obstructive sleep apnea; oVEMP, Ocular VEMPs; PTA, Pure-tone audiometry; SDQ, Strength and difficulties questionnaire; SNR, Signal-to-noise; SPT, Static posturography; SSNHL, Sudden sensorineural hearing loss; TEOAE, Transient-evoked OAEs; VEMP, Vestibular evoked myogenic potentials; VHIT, Video head impulse test; VNG, videonystagmography.

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consequences of having OSA are well documented with a strong association to excessive daytime sleepiness [2], increased incidence of hypertension [3], increased mortality [4] and risk of stroke [5]. The severity of OSA is determined by the apnea hypopnea index (AHI) which is the number of times the patient stops breathing per hour during sleep.

Despite the connection between OSA and multiple co-morbidities, there is limited research that evaluates the relationship between OSA and hearing/balance function. Such effects could be a direct consequence of the anatomical connection between the nasopharynx and the middle ear via the eustachian tube, and, potentially due to an effect of hypoxia on sensory structures in the inner ear and auditory brain centres. The eustachian tube connects the middle ear cavity with the nasopharynx. The main function of the eustachian tube is to aerate the middle ear and equalize atmospheric pressure across the tympanic membrane. The nasopharynx and oropharynx are the most common areas in the airway to collapse with OSA, these areas are also highly important in maintaining the physiological function of the eustachian tube [6]. Hence, any anatomical or functional changes to the nasopharynx and oropharynx with OSA could potentially affect the eustachian tube and regulation of middle ear function and transfer of sound to the inner ear. Changes in middle ear pressure (MEP) also have been shown previously to affect the activity of the vestibular neurons thus impacting balance function [7]. Thus, changes in middle ear function with OSA can potentially affect the vestibular system.

Continuous positive airway pressure (CPAP) is the primary treatment for patients with OSA [8,9]. CPAP acts as a splint where the positive pressure opens the airway. CPAP has been shown to reduce the AHI, reduce daytime sleepiness, increase the quality of life, and decrease the incidence of motor vehicle accidents [10]. CPAP, with its positive pressure, could influence middle ear aeration through opening the eustachian tube or increasing oropharynx pressure thereby potentially affecting hearing and balance function.

Considering these aspects, this scoping review was untaken to systematically map the current research in the area of hearing and balance function in OSA patients and the effect of CPAP therapy on hearing and balance functions.

The review considered the following research questions:

- What is known from the literature about hearing function in adults and children with OSA, and specifically which region of the hearing system is affected?
- What is known from the literature about vestibular function in adults and children with OSA, and specifically which region of the vestibular system is affected?
- What is the effect of CPAP on hearing and balance function?

2. Materials and methods

2.1. Search methods

The PRISMA extension for scoping review methodology was followed in conducting this scoping review [11].

2.2. Eligibility criteria

To be included in this scoping review published studies involving hearing or balance tests on suspected OSA adults or children were included. Studies involving CPAP treatment with hearing and balance evaluations in patients with or without OSA were included as it is important to consider the impact of CPAP on hearing and balance function in general. However other treatments for OSA like uvulopalatopharyngoplasty, implants or mandibular advancement were excluded. Conference abstracts were excluded. All animal studies were excluded. Publications only in English were included.

2.3. Information sources

A search of the literature for the relevant studies was conducted using PubMed and Embase databases. The search was conducted for all years up to 15 December 2020. The final search results were exported into Excel.

2.4. Search

The term “Obstructive Sleep Apnea” AND.

- “Hearing”
- “Vestibular”
- “Eustachian Tube”
- “Middle Ear”

The term “Continuous Positive Airway Pressure” AND.

- “Hearing”
- “Vestibular”
- “Eustachian Tube”
- “Middle Ear”

Were used and the filter “Humans” and “English” was applied. The search was conducted for all years up to 15 December 2020.

The number of recorded, identified, included, and excluded studies were recorded in a PRISMA flow diagram [12].

2.5. Selection of sources of evidence

Articles were screened for eligibility based on the title and abstract. Full articles were retrieved only for relevant studies. One reviewer completed the search and screened the articles. Once the full articles were retrieved for relevant studies, two reviewers agreed on the articles that were appropriate to be included in the scoping review.

2.6. Data charting process

Data charting captured the relevant information on key variables to extract. One reviewer completed the data charting process independently. Key variables were based on the primary and secondary outcomes described in section 2.7.

2.7. Data items

Baseline characteristics (e.g country of origin, experimental groups, number of patients in each group) were collected and reported in Table 1. The primary outcomes were changes in hearing assessment through pure tone audiometry (PTA), speech audiometry, tympanometry/admittance, eustachian tube testing, otoacoustic emissions (OAE), auditory brainstem response (ABR); and balance assessments through videonystagmography (VNG), video head impulse test (vHIT), vestibular evoked myogenic potentials (VEMP) and positional testing concerning OSA or CPAP therapy. The secondary outcome was the subjective perception of hearing/balance function through validated questionnaires with OSA patients and the changes after CPAP therapy.

Table 1
Description of Studies Included in this Review.

Author	Year	Country	Study Design	Category	Adults or Children	Groups	Total Patients included	Subgroup Numbers	Males (%)	Age	Comorbidities
Martins CH et al. [15]	2011	Brazil	Prospective Cross-Sectional	Observational	Adults	- OSA - Snorers	66	- OSA - 54 - Snorers - 12	- OSA - 85.2% - Snorers - 50%	- OSA - 43.53 (range 24–59) - Snorers - 34.41 (range 22–54)	No (excluded conductive hearing loss, middle and external ear disorder, neurological disorders, dementia, diabetes)
Sangal RB and Sangal JM [16]	1997	US	Prospective Cross-Sectional	Observational/Treatment	Adults	- Severe OSA pre/post CPAP - Control	80	- Severe OSA pre/post CPAP - 40 - Control - 40	Did not mention	- Severe OSA pre/post CPAP - 52.7 ± 11.9 - Control - 49.8 ± 14.9	Did not mention
Leite Filho CA et al. [17]	2017	Brazil	Prospective Cross-sectional	Observational	Children	- OSA - Snoring - Control	37	- OSA - 13 - Snoring - 13 - Control - 11	- OSA - 53.8% - Snoring - 46.2% - Control - 66.7%	- OSA - 9.2 ± 2.1 - Snoring - 9.2 ± 2.1 - Control - 9.0 ± 2.2	No
Hill CM et al. [18]	2017	UK	Prospective Cross-sectional	Observational	Children	- OSA (Snoring) - Control (no-snoring)	58	- OSA - 37 - Control - 21	- OSA - 64.9% - Control - 57.1%	- OSA - 3.76 ± 0.80 - Control - 4.19 ± 0.03	Did not mention
Ekin S et al. [19]	2016	Turkey	Prospective Cross-sectional	Observational	Adults	- OSA - Snoring - Control	66	- OSA - 27 - Snoring - 18 - Control - 21	- OSA - 74.1% - Snoring - 50% - Control - 42.9%	- OSA - 41.56 ± 8.99 - Snoring - 37.28 ± 8.23 - Control - 39.14 ± 9.91	No (excluded diabetes, congestive heart failure, hypertension etc)
Vorlova T et al. [20]	2016	Czech Republic	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Severe OSA - Control	43	- Mild OSA - 11 - Severe OSA - 17 - Control - 15	- Mild OSA - 100% - Severe OSA - 100% - Control - 100%	- Mild OSA - 46.4 ± 9.2 - Severe OSA - 50.8 ± 10.2 - Control - 47.3 ± 8.0	No (excluded diabetes, congestive heart failure, hypertension etc)
Deniz M et al. [21]	2016	Turkey	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	160	- OSA - 120 - Control - 40	52.5% (total)	- Mild OSA - 42.64 ± 13.8 - Moderate OSA - 41.35 ± 13.7 - Severe OSA - 43.14 ± 11.6 - Control - 42.32 ± 12.4	No (excluded diabetes, congestive heart failure, hypertension etc)
Seo YJ et al. [22]	2016	South Korea	Retrospective Cross-sectional	Observational	Adults	- Severe OSA with HI - Severe OSA without HI	41	- Severe OSA with HI - 19 - Severe OSA without HI - 22	- Severe OSA with HI - 73.7% - Severe OSA without HI - 77.3%	- Severe OSA with HI - 61.5 ± 3.3 - Severe OSA without HI - 57.3 ± 1.6[a]	Yes - Tinnitus, Hypertension, Diabetes, Dyslipidaemia, Chronic Kidney disease
Martines F et al. [23]	2016	Italy	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	160	- Mild OSA - 58 - Moderate OSA - 18 - Severe OSA - 24 - Control - 60	– 64.37% (Total)	– 46.19 ± 7.01 (Total)	No (excluded diabetes, congestive heart failure, hypertension etc)
Robison JG et al. [24]	2012	US	Retrospective Cross-sectional	Prevalence	Children	- OSA - OSA with ETD - MTs	295	- OSA - 295 - OSA with ETD - 94 - MTs - 30	- OSA - 66.4% - OSA with ETD - 68.1% - MTs - 70%	Not stated - inclusion between 3 and 24 months	Yes
Hwang JH et al. [25]	2011	Taiwan	Prospective Cross-sectional	Observational	Adults	- OSA - Control	224	- OSA - 34 - Control - 190	- OSA - 82.4% - Control - 51.6%	- OSA - 62.2 ± 5.5 - Control - 61.1 ± 6.9	Yes - coronary artery disease, chronic kidney disease, hypertension, diabetes, dyslipidaemia
Casale M et al. [26]	2012	Italy	Prospective Cross-sectional	Observational	Adults	- Severe OSA - Control	39	- Severe OSA - 18 - Control - 21	- Severe OSA - 83.3% - Control - 76.2%	- Severe OSA - 31 - Control - 39	No (excluded diabetes, hypertension, hypercholesterolemia, mild or moderate OSA
	2006	Brazil		Observational	Children	- OSA oral breathers	30	- OSA oral breathers - 10			

(*continued on next page*)

Table 1 (*continued*)

Author	Year	Country	Study Design	Category	Adults or Children	Groups	Total Patients included	Subgroup Numbers	Males (%)	Age	Comorbidities
Ziliotto KN et al. [27]			Prospective Cross-sectional			- Oral breathers - Nasal breathers		- Oral breathers - 10 - Nasal breathers - 10	- OSA oral breathers - 70% - Oral breathers - 60% - Nasal breathers - 50%	- OSA oral breathers - 7.6 - Oral breathers - 8.1 - Nasal breathers - 7.5	No (excluded facial malformation, genetic syndromes, neurological diseases
Thom JJ et al. [28]	2015	US	Prospective Cross-sectional	Observational and Treatment	Adults	- OSA pre/post CPAP	10	- OSA pre/post CPAP - 10	- OSA pre/post CPAP - 60%	- OSA pre/post CPAP - 57.8 (range 37.7 −74.7)	Not Mention
Micarelli A et al. [29]	2017	Italy	Prospective Cross-sectional	Observational	Adults	- Moderate-to-severe OSA - Control	64	- Moderate-to-severe OSA - 32 - Control - 32	- Moderate-to-severe OSA - 44.7% - Control - 56.3%	- Moderate-to-severe OSA - 51.4 ± 5.8 - Control - 52.7 ± 6.9	No (excluded neurologic, psychiatric, metabolic, cardiovascular or endocrine disorders)
Kayabasi S et al. [30]	2015	Turkey	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Moderate-to-severe OSA	50	- Mild OSA — 25 - Moderate-to-Severe OSA -25	— 68% (Total)	— 45 ± 11 (Total)	No (excluded neurologic, psychiatric, metabolic, cardiovascular or endocrine disorders)
Mutlu M et al. [31]	2015	Turkey	Prospective Cross-sectional	Observational	Adults	- Severe OSA - Snoring	54	- Severe OSA - 28 - Snoring - 26	Did not mention	— 46.5 ± 9.7 (Total)	No (excluded neurologic, psychiatric, metabolic, cardiovascular or endocrine disorders, older than 60, history of cervical surgery, BMI over 40)
Gallina S et al. [32]	2010	Italy	Prospective Cross-sectional	Observational	Adults	- OSA - Control	75	- OSA - 45 - Control - 30	- OSA - 68.9% - Control —66.7%	- OSA - 43.3 (range 24 —56) - Control - 41 (range 27 —54)	Did not mention
Urban PP et al. [33]	1996	Germany	Prospective Cross-sectional	Observational	Adults	- OSA	18	- OSA - 18	- OSA - 88.9%	- OSA - Range - 44-64	No mention
Wang W et al. [34]	2016	China	Prospective Cross-Sectional	Observational	Adults	- Moderate OSA - Severe OSA - Control	118	- Moderate OSA - 40 - Severe OSA - 44 - Control 34	- Moderate - 80% - Severe - 81.9% - Control - 64.7%	- Moderate 55.6 ± 13.2 male, 56.5 ± 4.9 female - Severe - 54.6 ± 9.6 male, 60.5 ± 8.2 female - Control - 53.3 ± 5.9 male, 58.2 ± 13.5 female	- No - excluded diabetes, hypertension/ cardiovascular disease, neurological deficits, any middle or inner ear pathology, Chronic respiratory disease
Matsumura E, Matas CG, Magliaro FCL et al. [35]	2018	Brazil	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	38	- Mild OSA - 11 - Moderate - 8 - Severe - 9 - Control - 10	— 100% (Total)	— 35.3 ± 7.1 (Total)	No - excluded BMI >40, Diabetes, Heart failure, Hypertension, Thyroid changes, Dyslipidemia, Stroke, hearing loss
Matsumura E, Matas CG, Sanches SGG et al. [36]	2018	Brazil	Prospective Cross-Sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	38	- Mild OSA - 11 - Moderate - 8 - Severe - 9 - Control - 10	— 100% (Total)	- Mild - 32.8 ± 2.9 - Moderate - 34.1 ± 6.8 - Severe - 41.2 ± 9.2 - Control - 33.6 ± 6.4	No - excluded BMI >40, Diabetes, Heart failure, Hypertension, Thyroid changes, Dyslipidemia, Stroke, hearing loss
Iriz A et al. [37]	2018	Turkey		Observational	Adults	- Moderate-to-severe OSA - Control	31	- Moderate-to-severe OSA - 21	- Moderate-to-severe OSA - 57.1%	- Moderate-to-severe OSA - 43.3 ± 8.2	No - excluded hypertension,

		Prospective Cross-Sectional					- Control - 10	- Control - 50%	- Control - 43.6 ± 3.8	diabetes, COPD, hearing loss
Lin FY et al. [38]	2012 US	Retrospective and prospective cross-sectional	Treatment	Adults	- Healthy + CPAP - Control	10 + 3066 tympanograms	- Healthy + CPAP - 10 - Control - 3066 tympanograms	- Healthy + CPAP - Not mention - Control - 49.5%	- Healthy + CPAP - Not mention - Control - 57	Did not mention
Aksoy F et al. [39]	2010 Turkey	Prospective Cross-sectional	Treatment	Adults	- OSA + CPAP - OSA	99	- OSA + CPAP - 51 - OSA - 48	- OSA + CPAP - 86.3% - OSA - 68.8%	- OSA + CPAP - 51.2 ± 9 - OSA - 36 ± 13.7[a]	Did not mention
Yung MW [40]	1999 UK	Prospective Cross-sectional	Treatment	Adults	- Atelectasis + CPAP - Control + CPAP	50	- Atelectasis + CPAP −30 - Control + CPAP −20	Did not mention	Did not mention	Did not mention
Aguilar F et al. [41]	2016 Spain	Prospective Cross-sectional	Treatment	Adults	- OSA + CPAP	36	- OSA + CPAP −36	- OSA - 86.1%	- OSA - 55.72 ± 9.6	Yes (but excluded patients with corticosteroids, anti-histamines, rhinitis-inducing medications, previous nasal pathologies, central sleep apneas)
Li J and Li K [42]	2016 China	Prospective Cross-sectional	Treatment	Adults	- Severe OSA + CPAP - Control + CPAP	100	- Severe OSA + CPAP - 50 - Control + CPAP - 50	- Severe OSA + CPAP - 100% - Control + CPAP - 100%	- Severe OSA + CPAP - 45.4 ± 6.4 - Control + CPAP - 24.4 ± 2.5[a]	Yes (but excluded Eustachian tube dysfunctions, other ear diseases, absence of acoustic stapedial reflex)
Akbulut S et al. [43]	2016 Turkey	Prospective Cross-sectional	Treatment	Adults	- Atelectasis + CPAP - Atelectasis + Placebo	47	- Atelectasis + CPAP - 24 - Atelectasis + Placebo - 23	- Atelectasis + CPAP − 41.7% - Atelectasis + Placebo − 52.2%	- Atelectasis + CPAP - 37.87 ± 10.83 - Atelectasis + Placebo - 36.03 ± 11.19	No − excluded snoring, daytime sleepiness, ESS >5, allergic, vasomotor rhinitis, sinonasal or nasopharyngeal pathology, air flights or diving during the following 6 months
Nakayama M et al. [44]	2015 Japan	Prospective Cross-sectional	Treatment	Adults	- OSA + Meniere's pre/post CPAP	20	- OSA + Meniere's pre/post CPAP −20	− 70%	− 60	Did not mention
Sheu JJ et al. [45]	2012 Taiwan	Retrospective Case-Control	Prevalence	Adults	- SSNHL - Control	19152	- SSNHL - 3192 - Control - 5960	- SSNHL - 53.9% - Control - 53.9%	- Unspecified	Yes - hyperlipidemia, diabetes, hypertension, coronary artery disease
Sivri B et al. [46]	2013 Turkey	Prospective Cross-sectional	Treatment	Adults	- Moderate/Severe OSA + CPAP - Control + CPAP	138	- Moderate/Severe OSA + CPAP - 78 - Control + CPAP - 60	− 69.6% (Total)	- Moderate/Severe OSA + CPAP - 47.38 ± 6.23 - Control + CPAP OSA - 46.10 ± 1.96	Yes
Kayabasi S et al. [47]	2019 Turkey	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	120	- Mild OSA - 33 - Moderate - 24 - Severe - 36 - Control - 27	- Mild - 57.6% - Moderate - 58.3% - Severe - 58.3% - Control - 51.9%	- Mild - 42 ± 8 - Moderate - 43 ± 6 - Severe - 42 ± 8 - Control - 43 ± 7	Yes (but excluded patients over 65 years, perforated tympanic membrane, otitis media, otosclerosis, Meniere's disease, otologic surgery, ototoxicity, noise-induced hearing loss)

(*continued on next page*)

Table 1 (*continued*)

Author	Year	Country	Study Design	Category	Adults or Children	Groups	Total Patients included	Subgroup Numbers	Males (%)	Age	Comorbidities
Magliulo G et al. [48],	2018	Italy	Prospective Cross-Sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	70	- Mild OSA - 8 - Moderate - 14 - Severe - 18 - Control - 30	- OSA - 65% - Control - 56.7%	- OSA - 50.1 - Control - 47.1	Yes (but excluded middle ear, nose and oropharynx surgery)
Ungar OJ et al. [49],	2020	Israel	Prospective Cross-sectional	Observational	Adults	- OSA - Control	130	- OSA - 31 - Control - 99	- OSA - 61.3% - Control - 48%	- OSA - 43 (range 31–44) - Control - 55 (range 30–58)	Yes (but excluded ontological conditions, radiotherapy to the head/neck, connective tissue disorder)
Li X et al. [50],	2020	China	Prospective Cross-sectional	Observational	Adults	- OSA - Control	78	- OSA - 58 - Control - 20	– 79% (Total)	– 40.4 ± 11.3 (Total)	Yes (but excluded profound hearing loss, tinnitus, vertigo, history of ear surgery, family genetic history of deafness)
Xin Z et al. [51],	2018	China	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	85	- Mild OSA - 19 - Moderate - 20 - Severe - 21 - Control - 25	– 100% (Total)	- Mild - 32.1 ± 4.5 - Moderate - 33.5 ± 4.3 - Severe - 34 8 ± 4.9 - Control - 31.2 ± 4.9	No – excluded neurological or psychiatric diseases, recent use of drugs known to affect sleep or daytime sleepiness, brain traumatic injury, unstable high blood pressure and other diseases (e.g asthma, hypothyroidism, periodic leg-movement) causing daytime hypoxemia SaO_2 < 90%,
Gao T et al. [52],	2020	China	Prospective Cross-sectional	Observational	Adults	- Severe OSA - Control	80	- Severe - 38 - Control - 42	- Severe - 84% - Control - 83%	- Severe - 39.5 ± 8.3 - Control - 36.5 ± 9.6	No – excluded neurological diseases, middle or inner ear deficit, hearing threshold >30 db HL, diabetes, uncontrolled hypertension, cervical disease, BMI >32 kg/m^2
Ulusoy B et al. [53],	2020	Turkey	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	147	- Mild OSA - 40 - Moderate - 34 - Severe - 34 - Control - 39	- Mild OSA - 65% - Moderate - 70.6% - Severe - 85.3% - Control - 84.6%	- Mild - 46.75 ± 10.9 - Moderate 50.76 ± 12.2[a] - Severe - 49.29 ± 9.1 - Control - 41.33 ± 13.7	No – excluded hearing thresholds <25 dB, diabetes, uncontrolled hypertension, neurotological diseases, vascular diseases, acoustic trauma to the head, cervical or eye-related surgery, BMI >40, metabolic diseases, age >60
Alessandrini M et al. [54],	2019	Rome	Prospective Cross-sectional	Treatment	Adults	- Moderate/Severe OSA + CPAP	32	- Moderate/Severe OSA + CPAP - 32	Did not mention	Did not mention	No – excluded malignancy, head trauma, neuropsychiatric

											disorders; metabolic, cardiovascular, endocrine, infectious, neuro-otological illness
Cayir S et al. [55],	2020	Turkey	Prospective Cross-sectional	Observational + Treatment	Adults	- Mild OSA - Moderate OSA - Severe OSA without CPAP - Severe OSA with CPAP - Control	247	- Mild OSA - 42 - Moderate OSA -45 - Severe OSA without CPAP - 32 - Severe OSA with CPAP - 40 - Control - 88	- Mild OSA - 52.4% - Moderate OSA -60% - Severe OSA without CPAP -43.8% - Severe OSA with CPAP - 55% - Control - 47.7%	- Mild - 53 ± 9 - Moderate - 51 ± 8 - Severe OSA without CPAP - 55 ± 9 - Severe OSA with CPAP - 56 ± 8 - Control - 52 ± 9	Yes (but excluded ET dysfunction, reduced tympanic membrane compliance or middle ear volume, perforated tympanic membrane, otologic surgery, severe septal deviation, nasal polyposis, sinusitis, adenoidal hypertrophy)
Teklu M et al. [56],	2020	US	Prospective Cross-sectional	Observational	Adults	- OSA - Control	101	- OSA - 72 - Control - 26	- OSA - 58.3% - Control - 61.5%	- OSA - 48.2 ± 12.9 - Control - 41.3 ± 12.6	Yes (but excluded other sleep disorders and laryngopharyngeal reflux diseases)
Deniz M and Ersözlü T [57]	2020	Turkey	Retrospective Cross-sectional	Treatment	Adults	- OSA + CPAP - OSA without CPAP	22	- OSA + CPAP - 12 - OSA without CPAP - 10	- OSA + CPAP -58.3% - OSA without CPAP - 80%	- OSA + CPAP 57.33 ± 9.31 - OSA without CPAP - 56.30 ± 11.40.	- Unknown
Yan et al. [58],	2020	China	Prospective Cross-sectional	Observational	Adults	- OSA - GERD - OSA + GERD - Control	137	- OSA - 33 - GERD - 35 - OSA + GERD - 37 - Controls - 32	- OSA - 51.5% - GERD - 45.7% - OSA + GERD - 54.1% - Control - 53.1%	- OSA - 44.7 ± 10.2 - GERD - 47.1 ± 10.2 - OSA + GERD 41.1 ± 10.0. - Control - 47 ± 9.4	Yes (but excluded allergic or non-allergic rhinitis, previous history of head and neck surgery or radiation therapy, history of ear disease, use of medication that might alter the nasal mucosa, history of middle ear, nose or oropharynx surgery)
Birk et al. [59],	2020	Germany	Prospective Cross-sectional	Observational	Adults	- Mild OSA - Moderate OSA - Severe OSA - Control	56	- Mild OSA — 12 - Moderate OSA — 15 - Severe OSA — 19 - Control - 10	- Mild OSA - 58.3% - Moderate OSA - 80% - Severe OSA - 94.7% - Control OSA- 70.0%	- Mild OSA 51.08 ± 13.2 - Moderate OSA 57.07 ± 11.7 - Severe OSA 57.68 ± 11.57 - Control - 51.3 ± 13.15	- Yes (but excluded vestibular disorders, - profound hearing loss, patients who had a - poor range of motion in their neck, blind)

ETD: eustachian tube dysfunction; GERD: gastroesophageal reflux disease; HI: hearing impairment; MTs: ventilation tube placement; SSNHL: sudden sensorineural hearing loss.

[a] Statistically significant difference $P < 0.05$.

2.8. Critical appraisal of individual sources of evidence

Cross-sectional studies were assessed using the Newcastle–Ottawa scale adapted for cross-sectional studies [13] (see Table 2). Case-control studies were assessed using the Newcastle–Ottawa quality assessment scale for case–control studies [14] (see Table 3). Two reviewers independently critiqued the quality of the articles and resolved any disagreements through discussion and consensus.

2.9. Synthesis of results

The studies were grouped by the prevalence of OSA with hearing or balance dysfunction, specific changes in hearing assessments in either children or adults, changes in vestibular assessments in both children or adults and changes with CPAP concerning hearing and balance function.

3. Results

3.1. Selection of sources of evidence

See Fig. 1.

3.2. Characteristics of sources evidence

There were 45 studies included in the analysis; 44 were cross-sectional studies, and one was a case-controlled study. Descriptions of the included studies are shown in Table 1.

3.3. Critical appraisal within sources of evidence

Assessment of the quality of the studies are found in Tables 2 and 3

3.4. Synthesis of results

3.4.1. Prevalence

There were only two studies that examined the prevalence of concomitant hearing disorders with OSA [24,45], one of which included children [24]. No study evaluated the prevalence of balance disorders with OSA.

The prevalence of OSA and hearing disorder (sudden sensorineural hearing loss (SSNHL)) was evaluated in adults from a large nationwide population dataset in Taiwan [45]. From the dataset of 19152 patients, 3192 patients who were identified to have a sudden sensorineural hearing loss (SSNHL) were age and sex-matched with

Table 2
Methodologic quality assessment of cross-sectional studies based on the adapted Newcastle–Ottawa scale.

Study	Year	Selection	Comparability	Outcome	Overall Score (Out of 10)
Martins CH et al. [15]	2011	4	0	3	7
Sangal RB and Sangal JM [16]	1997	4	1	3	8
Leite Filho CA et al. [17]	2017	4	0	3	7
Hill CM et al. [18]	2017	4	2	3	9
Ekin S et al. [19]	2016	4	2	3	9
Vorlova T et al. [20]	2016	3	2	3	8
Deniz M et al. [21]	2016	4	1	3	8
Seo YJ et al. [22]	2016	3	1	3	7
Martines F et al. [23]	2016	4	1	3	8
Robison JG et al. [24]	2012	3	1	3	7
Hwang JH et al. [25]	2011	3	1	3	7
Casale M et al. [26]	2012	4	0	3	7
Ziliotto KN et al. [27]	2006	2	0	3	5
Thom JJ et al. [28]	2015	3	0	3	6
Micarelli A et al. [29]	2017	4	2	3	9
Kayabasi S et al. [30]	2015	4	2	3	9
Mutlu M et al. [31]	2015	4	2	2	8
Gallina S et al. [32]	2010	4	1	2	7
Urban PP et al. [33]	1996	3	0	2	5
Wang W et al. [34]	2016	4	1	3	8
Matsumura E et al. [35]	2018	3	2	3	8
Matsumura E et al. [36]	2018	3	2	3	8
Iriz A et al. [37]	2018	3	1	3	7
Lin FY et al. [38]	2012	3	0	3	6
Aksoy F et al. [39]	2010	4	0	3	7
Yung MW [40]	1999	3	0	2	5
Aguilar F et al. [41]	2016	4	0	3	7
Li J and Li K [42]	2016	2	0	3	5
Akbulut S et al. [43]	2016	4	2	3	9
Nakyama M et al. [44]	2015	4	0	3	7
Sivri B et al. [46]	2013	3	1	3	7
Kayabasi S et al. [47]	2019	4	2	3	9
Magliulo G et al. [48],	2018	4	2	3	9
Ungar OJ et al. [49],	2020	4	1	2	7
Li X et al. [50],	2020	4	0	3	7
Xin Z et al. [51],	2018	3	1	3	7
Gao T et al. [52],	2020	4	2	3	9
Ulusoy B et al. [53],	2020	5	1	3	9
Alessandrini M et al. [54],	2019	4	0	3	7
Cayir S et al. [55],	2020	4	0	3	7
Teklu M et al. [56],	2020	4	1	2	7
Deniz M and Ersozlu T[57]	2020	3	2	3	8
Yan S et al. [58],	2020	4	2	3	9
Birk R et al. [59],	2020	4	1	3	8

Table 3
Methodological quality assessment of case-control studies based on the Newcastle–Ottawa scale.

Study	Year	Selection	Comparability	Outcome	Overall Score (Out of 8)
Sheu JJ et al. [45]	2012	3	2	3	8

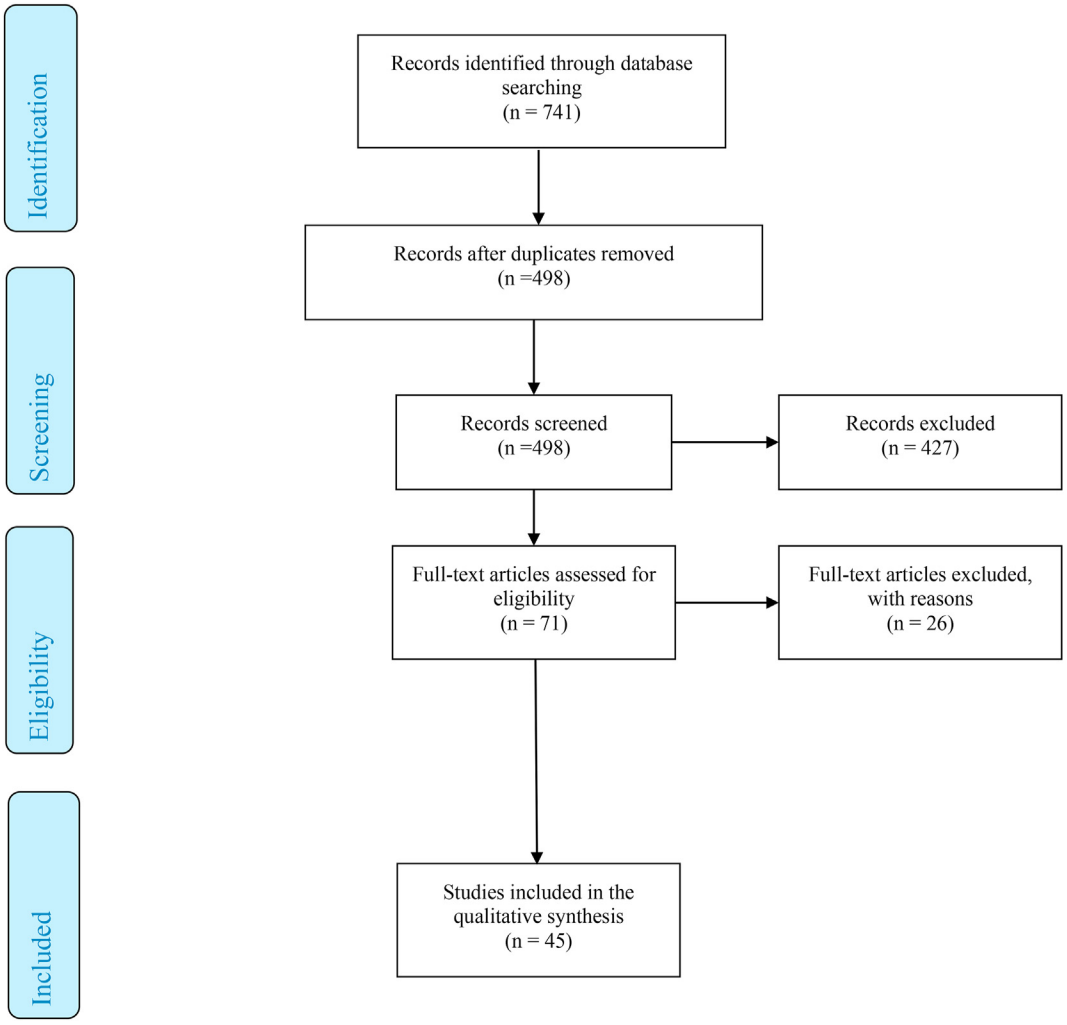

Fig. 1. Flow char of study selection.

15960 control patients (without SSNHL). 1.7% of the SSNHL patients had OSA while in the control group 1.2% had OSA. After adjusting for factors like income, diabetes, coronary heart failure, obesity, SSNHL patients are more likely to have prior OSA compared to the control (odds ratio 1.42; 95% CI, 1.05–1.93; p = 0.03). However, male patients with SSNHL had a higher proportion of prior OSA than controls (odds ratio 1.48; 95% CI, 1.02–2.16, p = 0.04) which was not observed in female patients with SSNHL. This large dataset highlights the issues that SSNHL patients might need to be evaluated for OSA and vice versa, especially in the male population.

In a retrospective review of medical records in children aged 3–24 months old diagnosed with OSA (AHI >1.5 events/hour), concomitant with eustachian tube disorder was in 94 of the 295 OSA children. The prevalence within this group was 31.9% which is significantly higher than the general paediatric population which would be between 4 and 7% [24] suggesting there may be a relationship between OSA and eustachian tube dysfunction but this paper was a retrospective study of medical records and cannot be applied to the general population.

3.4.2. OSA and hearing

The overall results relating to OSA and hearing in adults are found in Table 4.

3.4.2.1. Hearing thresholds. Twelve studies reported hearing thresholds through pure-tone audiometry (PTA) [19–21,23,25,26,30,32,35,37,47,50]. From that, nine studies reported an increase in hearing thresholds using pure-tone audiometry [19–21,23,25,26,32,47,50], especially at higher frequencies in moderate and severe OSA patients. The degree of hearing loss is within the mild hearing loss range (20 to <35 dB) at PTA frequency of 250–8000Hz [20,21,23,25,47,50] while moderate to moderately severe hearing loss (35 to <65 dB) were found with the extended high frequency (9000–16000Hz) [19,23]. However, despite

Table 4
Overview of studies on OSA and Hearing in Adults.

Study	Groups/Sample Size	Study Outcome						
		Pure Tone Audiometry (PTA)	Admittance	Otoacoustic Emission (OAE)	Auditory Brainstem Response (ABR)	Central Auditory processing (Behavioural)	Central Auditory processing (Objective)	Nasal and Eustachian tube Function
Martines F et al. [23]	- Mild OSA - 58 - Moderate OSA - 18 - Severe OSA - 24 - Control - 60	↑ 6000 - 16 000Hz (moderate and severe)		↓ 300–4000Hz TEOAE (severe)				
Hwang JH et al. [25]	- OSA - 34 - Control - 190	↑ 2000–8000Hz				↓ 880 and 1430Hz PPS		
Matsumura E, Matas CG, Magliaro FCL et al. [35]	- Mild OSA - 11 - Moderate - 8 - Severe - 9 - Control - 10	NC	NC (MEP, Ytm and acoustic reflexes)		↑ latencies Wave V (moderate OSA)			
Casale M et al. [26]	- Severe OSA - 18 - Control - 21	↑ 4000Hz		↓ 1000–4000Hz TEOAE ↓ 1000–6000Hz DPOAE	↑ latencies Wave I, III, V ↑ IPLS of I–V, III-V, I-III			
Martins CH et al. [15]	- OSA - 54 - Snorers - 12						↓ P300 amplitude. NC in P300 latency	
Matsumura E, Matas CG, Sanches SGG et al. [36]	- Mild OSA - 11 - Moderate - 8 - Severe - 9 - Control - 10	NC	NC (Tympanometric values)	↓ 1000–6000Hz DPOAE (severe)				
Ekin S et al. [19]	- OSA - 27 - Snoring - 18 - Control - 21	↑ extended high frequency 10 000–16 0000Hz (OSA and Snoring)						
Vorlova T et al. [20]	- Mild OSA - 11 - Severe OSA - 17 - Control - 15	↑ 4000 and 8000Hz (severe)		NC TEOAE	NC			
Deniz M et al. [21]	- Mild OSA - Moderate OSA - Severe OSA [Total OSA – 120] - Control –40	↑ Average (500, 1000, 2000Hz) (moderate and severe)		↓ 1000–4000Hz TEOAE (moderate and severe)				
Kayabasi S et al. [30]	- Mild OSA – 25 - Moderate-to-Severe OSA -25	NC						
Gallina S et al. [32]	- OSA - 45 - Control - 30	↑ Sensorineural Hearing Loss (4000–8000Hz)			↑ I–V wave latency			
Li J and Li K [42]	- Severe OSA pre/post CPAP –50 - Control pre/post CPAP –50		↑ MEP (before swallowing and after swallowing – before CPAP)					
Wang W et al. [34]	- Moderate OSA - 40 - Severe OSA - 44 - Control 34				↑ I, III, V wave latency			
Iriz A et al. [37]	- Moderate-to-severe OSA - 21 - Control - 10	NC ↓ Speech audiometry discrimination scores				↓ DPS, PPS		

Sangal RB and Sangal JM [16]	- Severe OSA pre/post CPAP - 40 - Control - 40					NC – P300 (Auditory amplitude, auditory latency, visual amplitude) ↑Visual Latency	
Urban PP et al. [33]	- OSA - 18				Normal range		
Thom JJ et al. [28]	- OSA pre/post CPAP - 10		↑MEP during sleep (before CPAP)				
Kayabasi S et al. [47]	- Mild OSA - 33 - Moderate - 24 - Severe - 36 - Control - 27	↑ 4000–8000Hz (moderate) ↑ 250–8000Hz (severe) ↑ Speech audiometry thresholds (severe) ↓ Speech audiometry discrimination scores (moderate and severe)					
Magliulo G et al. [48]	- Mild OSA - 8 - Moderate - 14 - Severe - 18 - Control - 30						↑ Rhinomanometry ↑ NMCT ↓ ETS-7
Ungar OJ et al. [49],	- OSA - 31 - Control - 99						↑ ETDQ-7
Li X et al. [50],	- OSA - 58 - Control - 20	↑ 4000–8000Hz		↓ 750–8000Hz DPOAE	↑ I, V wave latency NC - IPLS		
Xin Z et al. [51],	- Mild OSA - 19 - Moderate - 20 - Severe - 21 - Control - 25				↑ V and A wave latencies ↓ V/A wave Slope ↓ V and A wave Amplitude (note: Speech ABR)	↓ P300 amplitude. ↑P300 latency	
Cayir S et al. [55],	- Mild OSA - 42 - Moderate OSA -45 - Severe OSA without CPAP - 32 - Severe OSA with CPAP - 40 - Control - 88		↓MEP and ↑ Type B and C Tympanograms (Severe without CPAP)				
Teklu M et al. [56],	- OSA - 58.3% - Control - 61.5%						NC – ETDQ-7, SNOT-22, NOSE
Yan S et al. [58],	- OSA - 33 - GERD - 35 - OSA + GERD - 37 - Controls - 32						↑ ETS-7 and ETDQ-7 (OSA + GERD and GERD) ↑ Rhinomanometry (OSA and OSA + GERD)

Abbreviation: DPOAE – distortion product otoacoustic dmissions; DPS – duration pattern test; ETDQ-7 – Eustachian Tube Dysfunction Questionnaire-7; ETS-7 – Eustachian Tube Score-7; GERD – gastroesophageal reflux disease; IPLS – longer interpeak latency; NMCT – nasal mucociliary clearance time; NOSE – Nasal Obstruction Symptom Evaluation; MEP: middle ear pressure; PPS – pitch pattern sequence; SNOT-22 – Sino-Nasal Outcome Test; Ytm – admittance.

showing differences between OSA vs control, there were three studies where hearing thresholds is also within the abnormal range in the controls (thresholds >20 dB) [19,23,25] while one study showed a normal hearing range in the OSA group [26].

Comparatively, three studies revealed no change compared to the control [30,35,37]. The discrepancies between the studies could be due to uneven or small sample sizes. None of the papers justified the sample size.

3.4.2.2. Speech audiometry. Only two papers evaluated speech audiometry and one of these OSA patients had worse speech discrimination rates than the control, despite no changes in hearing thresholds [37]. In another study where hearing thresholds were worse in moderate and severe OSA patients, speech discrimination scores were also worse in moderate and severe OSA patients, with severe OSA patients also having higher speech recognition thresholds [47].

3.4.2.3. Middle ear function. Using tympanometry to assess middle ear function, one study indicated an increase in MEP [42], one study showed a decrease [55] while two studies revealed no change in middle ear pressure in OSA patients [35,36].

MEP also increases in OSA patients during sleep with the increased pressure proportional to the number of hours asleep [28]. However, this single study had a small sample size where MEP was measured in 10 suspected OSA patients only. This was due to having the tympanometry probe temporarily sealed into their ears overnight which was time-consuming and limited the participants to sleep in a supine position only. It would be valuable to investigate whether the rate increase in MEP during sleep was proportionally the same in OSA patients and healthy controls.

Only one study included acoustic reflexes and these did not change in OSA patients [36].

3.4.2.4. Eustachian tube function. The Eustachian tube score (ETS-7) assesses the functionality of the eustachian tube by testing 5 subjective/objective aspects involving eustachian tube opening at different pressure levels, objective Valsalva evaluation, tympanometry and subjective perception of sound during the Valsalva manoeuvre (blowing against a closed mouth and nostrils) and the Toynbee manoeuvre (swallowing while the mouth and nostrils are closed). With a total score of 14 points, an ETS $\leq$7 corresponds to a diagnosis of eustachian tube dysfunction. In one study, 20% of OSA had ETS $\leq$7 compared to only 3.3% in the control group [48]. Within the same study, the nasal function was shown to be worse where the was an increase in nasal resistance through rhinomanometry and an increase in mucociliary transport time [48]. However, no difference was found with ETS-7, nasal resistance and the mucociliary transport time between the mild, moderate and severe OSA groups. This could be due to a lower number of mild OSA patients recruited (n = 8) compared to moderate (n = 14) and severe (n = 18). Abnormal ETS-7 results were also found in OSA patients with gastroesophageal reflux disease (GERD) and patients with GERD only, however not in pure OSA patients [58]. Rhinomanometry were worse in OSA and OSA + GERD [58]. This indicates a complex relationship between GERD, eustachian tube function and OSA.

Using a 7 questions subjective questionnaire - the Eustachian tube Dysfunction Questionnaire-7 (ETDQ-7), where patients are asked if they experience symptoms like ear pain or pressure, one study reported statistically worse symptoms in OSA patients despite unequal sample size (31 OSA vs 99 healthy controls) [49], while another study revealed no difference in Eustachian tube dysfunction symptoms as well as nasal symptoms compared to the control [56].

3.4.2.5. Cochlear function. There were six studies where Otoacoustic Emissions (OAE) (transient-evoked OAEs (TEOAE) [20,21,23,26] and/or distortion product OAEs (DPOAE) [26,36,50]) were measured to assess the function of the cochlear outer sensory hair cells. In studies that evaluated TEOAE, three studies revealed a statistically significant decrease in signal-to-noise (SNR) ratio between the 1000−4000Hz in severe OSA patients compared to the control [21,23,26], while only one study found no difference in TEOAE with severe OSA patients [20].

With DPOAE, all three studies described a decrease in DPOAE amplitude in severe OSA patients between 1000 and 6000Hz [26,36,50].

3.4.2.6. Auditory brainstem function. The neuronal function of the auditory brainstem pathways can be evaluated through the auditory brainstem response (ABR). Three studies revealed a significant increase in ABR wave latencies (I, III, V) in OSA patients [26,32,34], one study showed increased latencies in both waves I and V^{50} while another showed an increase in wave V latencies in moderate OSA patients only [35].

One of the studies that show an increase in ABR wave latencies in OSA patients also found a difference in sex, where severe male OSA patients had longer III wave latencies compared to female [34]. This indicated that males had more impairment than females.

There was only one study that showed no change in ABR between OSA and the controls despite an increase in hearing thresholds [20]. This could be due to the severe OSA patients having only mild hearing loss (20 to <35 dB) which might not be severe enough to change ABR characteristics [20]. Another study also revealed normal ABR in OSA patients, however in that study, with no controls and no statistical analysis, the evidence is too weak [33].

Instead of clicks which are used in standard ABR, speech ABR uses verbal stimuli consisting of consonants and vowels. In the one study that used the technique of speech ABR, it was found that the wave latencies of W and A were increased in OSA patients [51]. The amplitude of waves V and A were also lower compared to the control as well as the V/A wave slope [51]. This was seen in all OSA severity compared to the control, however as this study recruited male participants only, it is uncertain whether there are any gender differences [51].

An auditory processing disorder can be assessed through behavioural tests like pitch pattern sequence and duration pattern test which measures the ability to distinguish frequency and time between sounds and the ability to sort the difference. In two studies, OSA patients have reduced abilities to process the sound in frequency as well as with time [25,37].

3.4.2.7. Cognitive function. The objective test measuring the auditory P300 is a good tool for assessing cognition and awareness of sound. Auditory P300 amplitudes decreased in OSA patients in 2 studies [15,51] while one study showed no difference [16]. An increase in P300 latencies was also observed in one of the studies [51], but there was no change in latency in two others [15,16]. With visual P300, this was tested in one study which revealed an increase in P300 visual latencies with no change with visual amplitude compared to the control [16].

Cognitive function assessed with the Montreal Cognitive Assessment (MoCA), decreased progressively with the increase in OSA severities [51]. Through regression analysis, the increase in P300 auditory latencies and the decrease in amplitude was significantly correlated with this decrease in cognition scores [51]. Additionally, the speech ABR wave V and A amplitude were negatively correlated with P300 and a positive correlation with cognition scores [51], indicating the impact OSA has on the auditory brainstem which in turn leads to cognitive dysfunction.

Table 5
Overview of the studies on OSA and Hearing in Children.

Study	Groups	Study Outcome							
		Pure Tone Audiometry (PTA)	Sound localization	Memory Test (Verbal)	Memory Test (Non-verbal)	Dichotic digit test	Gaps-in-noise (GIN)	Hearing questionnaire	Behavioural questionnaire
Ziliotto KN et al. [27]	- OSA oral breathers - 10 - Oral breathers - 10 - Nasal breathers - 10		NC	NC	↓	↓			
Hill CM et al. [18]	- OSA (Snoring) - 37 - Control (No Snoring) - 21	↑						↑ (CMEDHQ)	↑ (BRIEF-P), ↑ (SDQ)
Leite Filho CA et al. [17]	- OSA - 13 - Snoring - 13 - Control - 11						↓ GDP NC - GDT		NC - SAB but worse for snoring group

Abbreviation: BRIEF-P - The Behaviour Rating of Executive Function-Preschool Questionnaire; CMEDHQ - The Childhood Middle Ear Disease and Hearing Questionnaire; GDP - gap detection percentage; GDT - gap detection threshold; SAB - Scale of Auditory Behaviours; SDQ - Strength and Difficulties Questionnaire.

3.4.3. Hearing in OSA children

The overall results relating to OSA and hearing in children are found in Table 5. The definition of OSA severities in the paediatric population are different from adults in that mild is classified as AHI 1–5, moderate as AHI 5–10 and severe as AHI >10 [60]. The problem with the three paediatric studies is that each of the studies had different definitions of AHI thresholds to be classified as OSA or did not specify which threshold they used. Hence it is difficult to compare the three studies.

Pure tone audiometry was only completed in one study and in this, the hearing thresholds were higher in snoring children aged 3–5 years compared to the non-snoring control [18]. Snoring children also had higher noise exposure through the Childhood Middle Ear Disease and Hearing Questionnaire (CMEDHQ), greater executive function problems as measured through the Behavioural Rating of Executive Function – Preschool Questionnaire (BRIEF-P) and worse behaviour through the Strength and Difficulties Questionnaire (SDQ) [18]. However, when the snoring OSA children were separated by the OSA diagnostic threshold of either AHI $\geq$2 events/hour or AHI $\geq$5 events/hour, it was only children with AHI $\geq$5 events/hour who showed a higher SDQ behavioural difficulties score. There was no difference in hearing thresholds, hearing exposure or executive function. Hence snoring might be an important factor with a lesser effect from the AHI (an indicator of OSA severity).

There was no difference in sound localization ability between OSA patients compared to control in children aged between 7 and 8 years old [27]. This was similar to the memory test for verbal sounds where OSA children were able to repeat syllables in a sequence compared to the control [27]. However, with a memory test for non-verbal sound where children had to point out the order in which 3 musical instruments were played, the OSA children performed significantly worse than the control [27]. This was similar to the dichotic digits test where the children had lower performance when they had to repeat the four numbers that were presented to them [27].

With the gaps-in-noise test which is used for assessing auditory temporal resolution, children with OSA performed worse in terms of gap detection percentage where they were worse at hearing gaps between white noise compared to the control and snoring children [17]. However, the gap detection threshold remains the same as the other groups [17]. With the Scale of Auditory Behaviours (SAB) questionnaire which evaluates the occurrence of behaviours associated with hearing, reading and academic abilities, attention, memory and organization, OSA children were the same as the control, however, the snoring group appears to have worse behaviours than the others [17].

3.4.4. OSA and balance

The overall results relating to OSA and balance are found in Table 6.

In four studies, eye movement recorded through video-nystagmography (VNG) and video head impulse test (VHIT) showed that OSA patients have a higher number of nystagmus and irregular eye movement [29,30,32,59]. One study showed an increased nystagmus and canal paresis in the moderate/severe OSA group compared to the mild OSA group when VNG were recorded [30], however, as this study did not have healthy controls, it cannot be determined rather the increase in nystagmus and canal paresis is due to OSA. A higher number of abnormal caloric results were reported in the OSA group compared to the control in the study by Gallina S et al., 2010, with abnormal results observed in 34 out of the 45 OSA patients compared to 1 out of 30 controls [32]. Through VHIT, Micarelli et al., 2017 revealed a decay of vestibulo-ocular reflex (VOR) gain in moderate-to-severe OSA patients compared to the control [29], while Birk et al., 2020 found a higher number of abnormal VHIT results in 10 OSA patients compared to only 1 from the controls [59].

Otolith and vestibular nerve dysfunction can be measured through vestibular evoked myogenic potentials (VEMPs). Cervical VEMPs (cVEMPs) are recorded from the sternocleidomastoid muscles and ocular VEMPS (oVEMPS) are recorded from the extraocular muscles, mainly the inferior oblique [61]. The cVEMPs tests the function of the saccule and inferior vestibular nerve along with the descending tracts of the brainstem. The oVEMPs originate in the saccule and the superior vestibular nerve and reflect ascending pathways [62]. VEMPs responses are biphasic (positive-negative waves), with the first biphasic complex as p1-n1 waves while the second phase is n2-p2 [31].

The response rate of cVEMPs was decreased in two studies [31,53], with no change observed in another [52]. cVEMPs amplitude significantly decreased in all three studies [31,52,53], however, in one of the studies, the decreased amplitude was in the moderate and severe OSA group compared to the mild OSA group, rather than the control group [53]. Nevertheless, this decrease in cVEMPs amplitude with OSA was hypothesis to be hypoxic damage to the sacculocolic reflex in the brainstem [31] or a decrease in vestibular nerve sensitivity [52].

There appears to be less effect with cVEMPs wave latencies as well as waves intervals. There was no difference observed for P1 latencies [31,52,53], N1 latencies [31,52] nor with N2 latencies compared to the control [31]. P2 latencies were shown to only decrease with moderate OSA patients [53], with another study revealing no change [31]. With wave intervals, the P1N1 interval decreased with moderate OSA patients [53] with no change in

Table 6
Overview of the studies on OSA and Balance in Adults.

Study	Groups	Study Outcome					
		VNG	VHIT	VEMP	Positional test	Cerebellar examination	DHI
Kayabasi S et al. [30]	- Mild OSA – 25 - Moderate-to-Severe OSA -25	↑ Nystagmus/Canal Paresis (moderate-to-severe OSA)			NC (Romberg)	NC	↑ Physical and Total (Moderate-to-severe OSA)
Gallina S et al. [32]	- OSA - 45 - Control - 30	↑ Hyporeflexia Bilateral, Hyporeflexia Unilateral, Saccadic eye movement, smooth-pursuit movement					
Micarelli A et al. [29]	- Moderate-to-severe OSA - 32 - Control - 32		↑ Covert and overt saccade		↑ (Static Posturography Testing)		↑ (Physical, Emotional, Functional, Total)
Mutlu M et al. [31]	- Severe OSA - 28 - Snoring - 26			↓ cVEMP response rate ↓ cVEMP p1n1 amplitude ↓ cVEMP n2p2 amplitude NC - cVEMP latencies (p1, n1, n2, p2) or interval (p1n1, n2p2)			
Gao T et al. [52],	- Severe OSA - 38 - Control - 42			NC - cVEMP response rate NC – cVEMP p1 and n1 latency, ↓cVEMP p1n1 amplitude ↓ oVEMP response rate, ↑ oVEMP n1 latency NC – oVEMP p1 latency			
Ulusoy B et al. [53],	- Mild OSA - 40 - Moderate - 34 - Severe - 34 - Control - 39			↓cVEMP response rate (moderate and severe), ↓cVEMP n1 and p2 latency (moderate) ↓cVEMP p1n1 interval (moderate) ↓cVEMP n1p2 interval (moderate and severe vs mild), ↓cVEMP p1n1 amplitude (moderate and severe vs mild), ↓cVEMP n1p2 amplitude (moderate and severe vs mild), NC to cVEMP p1 latency. ↓oVEMP response rate moderate and severe, ↓ oVEMP n1p1 amplitude (mild and severe), NC to oVEMP n1 and p1 latency and interval			
Birk et al. [59],	- Mild OSA – 12 - Moderate OSA – 15 - Severe OSA – 19 - Control - 10		50 completed VHIT – 7 OSA vs 1 control had pathological VHIT results	NC in cVEMP or oVEMP Note: cVEMP – reliable in half the patients oVEMP – 10 patients			

Abbreviation: DHI – Dizziness Handicap Inventory; cVEMP – cervical vestibular evoked myogenic potentials; oVEMP – ocular vestibular evoked myogenic potentials; VEMP – vestibular evoked myogenic potentials; VHIT – video head impulse test; VNG - videonystagmography.

another paper [31]. N1P2 interval was significantly decreased in one study in moderate and severe OSA patients, however, this was compared to mild OSA patients instead of the control [53]. Lastly, no change was seen with N2P2 interval [31]. The discrepancies between the decrease in cVEMPs amplitude with no consistent change in wave latencies remain puzzling as other brainstem damage (e.g Parkinson's disease, brainstem stroke, multiple sclerosis) shows a combination of decreased wave response, low wave amplitude and prolongation of latencies [63–65].

With oVEMPs, which provide information on the saccule and ascending pathway of the brainstem, oVEMPs in OSA were evaluated in only 2 studies. The response rate was significantly decreased in moderate and severe OSA patients [52,53]. The N1 latencies were increased in one study [52], while another study showed no change [53]. No difference was found with P1 latencies [52,53]. No change was discovered for the P1N1 interval nor N2P2 interval [31,53]. Changes in oVEMPs response might be associated with demyelination of the axons in the vestibular nucleus or with the vestibular pathway however it is uncertain the extent of changes if latencies or intervals were not significantly affected.

cVEMPs and oVEMPs were not included in one study due to unreliable data as data were not obtained in half of the participants for cVEMPs and only in 10 out of 56 with oVEMP [59].

In measures of the positional tests, one study revealed no change in postural instability through the Romberg test [30] while another study revealed an increase in postural instability through static posturography testing (SPT) in OSA patients [29]. As the cerebellum coordinates motor control, a cerebellar examination is a series of tests to evaluate gait, head, arm and leg movement. In the one study that uses the cerebellar examination, it revealed no change in OSA patients with dysdiadochokinesia or dysmetria [30]. In terms of the effect of dizziness on the quality of life through the Dizziness Handicap Inventory questionnaire, two studies revealed a significant impact of dizziness on the quality of life in OSA patients [29,30].

3.4.5. CPAP and hearing

The overall results relating to CPAP are shown in Table 7.

In three out of five studies, CPAP has been shown to decrease the hearing thresholds in patients with OSA with Meniere's disease or patients with atelectasis [40,43,44]. Two studies showed no change in hearing thresholds with CPAP, and this was in purely OSA patients [41,57]. Word recognition was evaluated by speech audiometry in one study but there was no improvement with CPAP despite the decrease in hearing thresholds in Atelectasis patients [43].

Six out of seven studies showed an increase in MEP with CPAP therapy in OSA patients [28,42,46,55], atelectasis patients [40,43] as well as healthy volunteers [38]. Two studies exhibited reflation of the eardrums in atelectasis patients [40,43]. This effect did not only occur acutely [28,38,42] but this also lasted long-term with MEP remaining increased or with reinflated eardrums when measured at 6 months post CPAP [40,43,46,55]. Eustachian tube functions did not differ between OSA patients using CPAP and OSA patients without CPAP when the automatic Toynbee test was completed [39]. Additionally, no difference in nasal resistance through rhinomanometry was found pre and post CPAP [41].

Table 7
Overview of the studies on CPAP and Hearing in Adults.

Study	Groups	Study Outcome			
		Pure Tone Audiometry (PTA)	Admittance	Central Auditory processing (Objective)	Nasal and Eustachian tube Function
Li J and Li K [42]	- Severe OSA + CPAP - 50 - Control + CPAP - 50		↑ MEP NC - Acoustic Reflex		
Lin FY et al. [38]	- Healthy + CPAP - 10 - Control - 3066 tympanograms		↑MEP (healthy)		
Thom JJ et al. [28]	- OSA pre/post CPAP - 10		↑ MEP		
Aksoy F et al. [39]	- OSA + CPAP - 51 - OSA - 48		NC − MEP NC - Compliance		NC (Automatic Toynbee test)
Sivri B et al. [46]	- Moderate/Severe OSA + CPAP - 78 - Control + CPAP - 60		↑ MEP (shift to Type A Tympanogram)		
Nakayama M et al. [44]	- OSA + Meniere's pre/post CPAP −20	↓ 500–2000Hz			
Yung MW [40]	- Atelectasis + CPAP -30 - Control + CPAP −20	↓	↑ ear drum reinflated		
Aguilar F et al. [41]	- OSA + CPAP −36	NC			NC (Rhinomanometry)
Akbulut S et al. [43]	- Atelectasis + CPAP - 24 - Atelectasis + Placebo - 23	↓ (average of 125–8000Hz) NC − Air bone gap NC - Word recognition score (Speech Audiometry)	↑ MEP ↑ ear drum reinflated		
Sangal RB and Sangal JM [16]	- Severe OSA pre/post CPAP - 40 - Control - 40			NC (Visual latency remained longer compared to pre CPAP treatment)	
Cayir S et al. [55],	- Mild OSA - 42 - Moderate OSA -45 - Severe OSA without CPAP - 32 - Severe OSA with CPAP - 40 - Control - 88		↑ MEP and shift to Type A tympanogram (Severe + CPAP post 6 months)		
Deniz M and Ersozlu T [57]	- OSA + CPAP - 12 - OSA without CPAP - 10	NC (2 years)			

Abbreviation: MEP − middle ear pressure; PTA − pure tone audiometry.

Table 8
Overview of the studies on CPAP and Balance in Adults.

Study	Groups	Study Outcome			
		VNG	VHIT	Positional Test	DHI
Nakayama M et al. [44],	- OSA + Meniere's pre/post CPAP - 20	NC (caloric test)			↓ (All)
Alessandrini M et al. [54],	- Moderate/Severe OSA + CPAP - 32		NC	↓ Static Posturography Testing	↓ (All)

Abbreviations: DHI – Dizziness Handicap Inventory; VHIT – video head impulse test; VNG - videonystagmography.

Objective testing of the auditory central processing with P300 revealed no change in auditory amplitude, auditory latency or visual amplitude; with visual latency remaining prolonged similar to before CPAP treatment [16].

3.4.6. CPAP and balance

The overall results relating to OSA and balance are found in Table 8.

In a cross-sectional study, CPAP did not affect nystagmus in patients with OSA + Meniere's disease as measured through the caloric test however it improved quality of life in all aspects (functional, emotional, physical) as presented with a decrease in the DHI [44]. In the only one study with purely OSA patients, CPAP did not improve VOR gain through the VHIT test, however, improvement with postural instability was revealed through a decrease with static posturography testing as well improvement in the quality of life through the DHI questionnaire [54].

3.4.7. Factors contributing to hearing and balance disorders

The overall results of factors with an association in hearing and balance in OSA patients are found in Table 9.

Increased AHI and lower oxygen saturation are factors that show the strongest association with hearing disorders. Increased AHI is linked to increased hearing loss in three out of four studies

Table 9
Factors associated with changes in hearing and balance in OSA patients.

	Average O_2	Lowest O_2	Duration of <90% O_2 saturation	Oxygen Desaturation Index (ODI)	Snoring Index	Total Sleep Time	BMI	ESS	AHI	AI	Gender
Ekin S et al. [19]	↑high-frequency hearing loss		↑ high-frequency hearing loss								
Vorlova T et al. [20]		↑high-frequency hearing loss	↑ high-frequency hearing loss	↑high-frequency hearing loss			↑high-frequency hearing loss		↑high-frequency hearing loss		
Seo YJ et al. [22]	– hearing loss	↑ hearing loss	– hearing loss	– hearing loss	– hearing loss	– hearing loss	– hearing loss	– hearing loss			
Martines F et al. [23]									↑high-frequency hearing loss		
Casale M et al. [26]	– hearing loss – OAE – ABR								– hearing loss – OAE – ABR		
Micarelli A et al. [29]	↑ VOR Gain (VHIT) ↑ Static Posturography body oscillations										
Wang W et al. [34]	↑ ABR	↑ ABR	↑ ABR	↑ ABR					↑ ABR	↑ ABR	↑ ABR in male
Kayabasi S et al. [47]		↑ hearing loss (250–8000Hz) ↑ Speech recognition threshold ↓ Speech discrimination score							↑ hearing loss (250–8000Hz) ↑ Speech recognition threshold ↓ Speech discrimination score		
Li X et al. [50],		– ABR							– ABR		
Ulusoy B et al. [53],									↓ cVEMP N1P2 interval and P1N1 amplitude – oVEMP		
Alessandrini M et al. [54],								↑ Static Posturography body oscillations	↑ Static Posturography body oscillations		
Birk et al. [59],	– VHIT – cVEMP – oVEMP	– VHIT – cVEMP – oVEMP	– VHIT – cVEMP – oVEMP	– VHIT – cVEMP – oVEMP					– VHIT – cVEMP – oVEMP		

Abbreviations: ABR – auditory brainstem response; AHI – apnea hypopnea index; AI – arousal index; BMI – body mass index; cVEMP – cervical vestibular evoked myogenic potentials; oVEMP – ocular vestibular evoked myogenic potentials; ESS – Epworth Sleepiness Scale; OAE – otoacoustic emissions, VHIT - video head impulse test; VOR – vestibulo-ocular reflex.
↑ increase, – No change. ↓ decrease.

[20,23,26,47], while the lowest oxygen saturation was linked to increased hearing loss in three out of three studies [20,22,47]. Speech recognition thresholds increased with a rise in AHI and oxygen saturation, while the speech discrimination score decreased, indicating a worsening ability to detect speech, however, this was completed in only one study [47].

The only study that showed an association with an increase in wave latencies of ABR with factors like oxygen saturation and AHI was Wang W et al. [34], the other two studies revealed no association [26,50], hence it remains inconclusive whether these factors influence changes in ABR. This is similar to OAE with only one study evaluating the association, where it showed no change in OAE with AHI nor oxygen saturation [26].

The evidence of factors associated with balance disorders in OSA patients is very limited. cVEMPs and oVEMPs were evaluated in only one study, where an increase in AHI was associated with a decrease in cVEMPs interval and amplitude, however, no association with oVEMPs was found [53]. The lowest average oxygen saturation was associated with irregular eye movement and postural instability in one study [29]. An increased in AHI as well as an increase in subjective perspective on sleepiness was also associated with postural instability, however, this was completed in one study [54].

4. Discussions

4.1. Summary of evidence

This review is the first to cumulatively evaluate the relationship between OSA with hearing and balance disorders and the effects of CPAP on these functions. There is evidence that OSA, a disease previously associated with hypertension and stroke, is also associated with hearing and balance disorders.

With two epidemiological studies completed, one in adults and one in children, the chances of having OSA was significantly higher in adults with sudden sensorineural hearing loss than in the general population (odds ratio of 1.43) [45]. In children with OSA, the association with eustachian tube disorder was approximately 4.5% higher than in the general population without OSA. However, as the study completed in children was a retrospective review of medical records, it only captured a small incidence in one hospital location, hence it cannot be representative of the general population. Regardless, patients with sudden sensorineural hearing loss or eustachian tube disorders should be examined for OSA and vice versa.

In audiology, it is important to evaluate the level of hearing loss as well as identify the site and cause of the hearing loss. Hence a battery of audiological tests is important to identify the site-of lesion or the regions involved within the auditory system. PTA is essential to assess the level of hearing loss, however, both air-conduction and bone-conduction audiometry are important to determine if it is conductive or sensorineural. It is also important to consider middle ear function when measuring OAE (an indicator of cochlear function), as abnormal MEP could reduce the transmission of sound through the middle ear and impact the amplitude of OAE.

Only one paper completed the battery of audiological assessments involving PTA, tympanometry and OAE [36]. There was no difference in hearing thresholds or tympanometry, however, DPOAE amplitude was significantly lower at 1000–6000Hz in severe OSA patients compared to the control. This is a possible indicator that OSA affects the cochlea, rather than the middle ear. However, as the sample size was small ($n = 38$) and involved only male participants, its generalisability needs to be furthered study.

When evaluating individual audiological assessments, OSA patients appear to have an increased hearing threshold at the higher frequencies, which is more evident in severe OSA patients [19–21,23,25,26,32,47,50]. At the hearing frequency between 250 and 8000Hz, the degree of hearing loss is only within the mild range (20 to < 35 dB) [20,21,23,25,47,50], however at extended high frequency (9000–16000Hz), OSA patients have moderate-to-moderately severe hearing loss (35 to < 65 dB) [19,23]. As there was a significant difference in hearing thresholds in OSA patients which was observed in 9 out of 12 papers, this provides strong evidence that OSA patients have worse hearing compared to the control. This is reflected in speech audiometry results, which evaluates speech recognition rather than tones, with both studies reporting worse speech discrimination scores in OSA patients [37,47]. However, the reason for this hearing loss cannot be determined until the battery of audiological assessments are completed to assess which region of the auditory system is being affected.

The middle ear gas composition and pressure are regulated by several factors, including gas exchange through the mucosal lining, the inner ear via the round window, the environment via diffusion across the tympanic membrane; and the nasopharynx via transfers across the eustachian tube [66]. With the eustachian tube connecting the middle ear to the nasopharynx, it is anatomically plausible that the collapse of the nasopharynx in OSA could ultimately affect middle ear function through the eustachian tube. However, the current literature on the relationship between OSA and middle ear function remains inconclusive, with two studies revealing no change in MEP with tympanometry or acoustic reflexes [35,36], another showing an increase in MEP [42] and conversely one showing a significant decrease in MEP [55].

Sleep can also independently increase the MEP. This has been previously shown in healthy controls [67] and this review indicates it also occurs in OSA patients during sleep as well [28]. Due to the natural suppression of respiratory drive during sleep [68], $PaCO_2$ would be elevated while PaO_2 decreases. The build-up of $PaCO_2$ could induce a net transport of CO_2 into the middle ear mucosa and transport of O_2 out from the mucosa, hence leading to an increase in middle ear pressure [66]. With the nature of OSA and its associated recurrent hypoxia, it could have an additive effect on CO_2 transport into the mucosa and alter MEP. However, this proposed mechanism of gas exchange through the mucosa during sleep is only one theory [67], an alternative mechanism is due to the reduction in tubal ventilation due to a decrease in swallowing during sleep, hence MEP was unable to equalized [69]. Nevertheless, since only one study with a sample size of only 10 participants evaluated MEP during sleep, it is important to expand the research with more participants and with different OSA severities.

As both the upper airway and the eustachian tube are collapsible tubes that connect, it is important to evaluate eustachian tube function in OSA patients. Only two papers evaluated eustachian tube function through the ETS-7. In one study there were higher numbers of eustachian tube dysfunction diagnoses in the OSA patient group which did not differ between OSA severities [48]. In a different paper, comorbidity of OSA with GERD resulted in a higher number of eustachian tube dysfunction, however, this was not observed in purely OSA patients [58]. Both studies also indicated an increase in nasal resistance along with the eustachian tube dysfunction [48,58]. The current data on eustachian tube function in OSA patients remains complex and inconclusive.

Within the battery of audiological tests, OAE are important to assess the status of the cochlea. The majority of the studies showed a significant decrease in OAE amplitudes in OSA patients [21,23,26,36,50], indicating, a potential decline in cochlear function. The measure of OAE is through a click or tone that is presented to the ear which travels through the middle ear to stimulate the cochlea, then the response from the cochlea will travels back through the middle ear for it to be detected in the ear canal. Hence OAE

measurements can be impacted also by the middle ear status [70]. Since only one study recorded middle ear status (through tympanometry) with their OAE measurements [36], it becomes difficult to determine if the OSA is affecting the cochlea or rather OSA is changing the middle ear status and thus the stimulation to the cochlea is being impaired.

ABR is a recording of the response from the auditory nerve and brainstem activity after a tone or a click stimulus. Changes in the different waves recorded can be used to differentiate between conductive, cochlear or retro-cochlear loss. Four studies demonstrated an increase in I - V latencies in OSA patients [26,32,34,50], indicative of conducting hearing loss. However, one study demonstrated an increase in wave V latencies only (indicative of retro-cochlear hearing loss) in moderate OSA patients [35], while another indicated no change in ABR in OSA patients compared to the control [20]. Similar to OAE, the benefit of evaluating middle ear function as a complementary test with ABR allows the ability to confirm if OSA patients are experiencing conductive hearing loss. Additionally, with a simulated model of long-term mild hypoxia in an animal model, it appears that ABR thresholds and amplitude are reduced [71], hence, it remains unknown rather the decrease in ABR observed in OSA patients are related to obstruction of sound entering into the inner ear, or due to recurrent hypoxia of OSA.

Speech ABR differs from standards ABR in that speech stimuli are presented instead of clicks. This is an important tool as speech is a complex signal and a non-speech stimulus would not provide an insight into the processing of speech sounds at the brainstem level [72]. Only one study in this review utilize speech ABR, and there appears to be an increase in latency, decrease in amplitude and decrease in interpeak of wave V and A in OSA patients, indicating an abnormality in the acoustic representation of speech sound from the auditory brainstem [51]. The change in speech ABR amplitudes for waves V and A was correlated to changes in MoCA scores, indicating worsening of cognition in severe OSA patients [51].

The changes within the auditory nerve and brainstem could affect the ability to process auditory information through the central nervous system. OSA patients show impaired ability to process the sound in frequency as well as in time with pitched pattern sequence and duration pattern test [25,37]. However, since both the pitched pattern sequence and duration pattern test are subjective tests and there appear to be inconclusive results in objective test through the measurement of P300 [15,16,51], relatively little is known about the relationship between OSA and central auditory processing. Additionally, the sample size in both the pitched pattern sequence and duration pattern sequence studies were disproportionate between OSA and controls in both studies hence it is difficult to decipher rather this effect was legitimate [25,37].

The main cause of OSA is different in children compared to adults. The cause of OSA in children is commonly due to adenotonsillar hypertrophy [73] while the risk factors for adults is obesity and craniofacial and upper airway abnormalities [74]. Concerning hearing, further research is essential as there was only one study that completed PTA where children who snored had higher hearing threshold as well as more behavioural issues through BRIEF- P and SDQ questionnaires [18]. Besides, the different battery of tests completed in adults has not been performed in children, with no literature evaluating middle ear status through tympanometry nor cochlear hair cells through OAE. Only auditory processing has been evaluated, where the memory for non-verbal sound and numbers were worse [27] in OSA children, as well as the ability to detect gaps between silence within an auditory stimulus [17]. Similar to hearing thresholds, worse behavioural scores were observed in children who snores [17,18], but not necessarily in the snoring children who had a diagnosis of OSA. Hence snoring might play a stronger role in hearing impairment and behavioural issues rather than OSA. One major limitation of the studies was that the diagnostic criteria of OSA differed between all three studies hence it is difficult to compare the results collectively.

With balance disorders, adults with OSA have a higher number of nystagmus [29,30,32,59] through VNG and VHIT, which could potentially lead to postural instability. Nevertheless, with one study showing increased instability [29] and another showing no change [30], further research is required. Previous research has shown a discordance between static posturography testing and VOR testing [75], where the number of patients identified as having an abnormal vestibular function is different for each test. However, there is still merit in performing both tests as it provides different information. It is undeniable that there is a significant impact of dizziness on the quality of life in OSA patients with it affecting all aspects including emotional, physical and functional [29,30].

VEMPs can determine any changes in the otolithic organs in the inner ear, the vestibular nerve and the central connections of the brainstem. A sound stimulus is applied to one ear which evokes a response within the sternocleidomastoid muscles (cVEMPs) or extraocular muscles (oVEMPs) which are recorded with surface electrodes [76]. cVEMPs and oVEMPs' response and amplitude have been shown to decrease in OSA patients [31,52,53]. The decrease in VEMPs highlights that either the brainstem or vestibular nerve are impacted by OSA. It is hypothesized that the nature of hypoxia with OSA could be responsible for damaging the vestibular nerve and brainstem, however with only one study revealing an inverse relationship between cVEMPs with AHI [49], it remains to be verified. It is also important to note that VEMPs results could diminish with conductive hearing loss as the sound stimulus must activate the saccule in the inner ear before cVEMPs response can be recorded. Hence the battery of audiological assessments is equally important when evaluating vestibular function and should be performed before VEMPs [77].

CPAP with its positive pressure is the gold standard treatment for OSA. Positive airway pressure is applied through a mask where the pressure splints the upper airway open. With the eustachian tube connecting the middle ear to the nasopharynx in the upper airway, CPAP could alter hearing function through this anatomical connection. With hearing thresholds, improvement in hearing was only observed in OSA patients with Meniere's disease [44] and patients with atelectasis alone [40,43]. Two studies that evaluated purely OSA patients indicated no change in hearing thresholds with long-term CPAP treatment [41,57], however, the OSA patients in one of the studies had normal hearing thresholds before CPAP [41] so it would be difficult to see improvements, while the other study reported hearing thresholds changes from baseline rather than the actual hearing thresholds [57] hence it is unknown rather those patients also had normal hearing thresholds before CPAP. The majority of studies have shown an increase in MEP with CPAP [28,38,42,43,46,55] and reflation of ear drums [40,43], with a greater effect with a higher CPAP pressure. This was observed in OSA patients, healthy volunteers as well as patients with atelectasis. With no testing completed with OAE or ABR, as well as limited literature on speech audiometry, auditory central processing and eustachian tube testing, more research is required to assess the impact of CPAP on hearing. However, the mechanism of CPAP appears to be through the middle ear with a significant increase in middle ear pressure.

There is limited research on CPAP and its effects on balance with only two studies available.

One cross-sectional study with OSA patients with Meniere's disease showed no effect with CPAP with nystagmus [44], while the other study that evaluated purely OSA patients also revealed no improvement with nystagmus with CPAP through VHIT testing

despite improvement with postural stability through SPT [54]. Nevertheless, CPAP improved the quality of life through the dizziness handicap inventory in both of the studies hence there could be certain benefits with CPAP on vestibular functions [44,54].

The Meniett device, is a market release positive pressure device that delivers a repeated pressure pulse of 12cmH_20 directly applied in the middle ear through a ventilation tube. This device has been shown to reduce episodes of vertigo, dizziness, aural pressure and tinnitus, with long-term benefits [78]. This was also associated with an improvement in hearing [78,79]. Hence, it is plausible that CPAP, with its positive pressure, could improve symptoms of vestibular disorders. However, since CPAP is applied through a mask on a patient's face rather than directly into the middle ear through a ventilation tube, its effect on middle ear pressure remains inconclusive and cannot be concluded that the CPAP device would produce similar results. It is believed that the pressure through the Meniett device applied in the middle ear is increasing the perilymphatic pressure which boosts the flow of the endolymphatic fluid within the vestibular organs [79].

With the recurrent hypoxia nature of OSA, because the patients stop breathing intermittently throughout the night, lower oxygen saturation in multiple studies was found to be associated with hearing loss, especially at higher frequencies [20,22,47]. The number of times the patient stops breathing, as indicated by AHI, was also linked to increased hearing loss in three out of four studies [20,23,26,47]. It would be beneficial to know whether peripheral or central hypoxia in OSA is affecting hearing. Additional research is required the see if there is a link between oxygen saturation and AHI with changes to the cochlea or brainstem as currently, the research is inconclusive. This is similar to the association with balance disorders.

4.2. Limitations

The limitations to this scoping review were that the search was completed by one researcher who screened all the publications through the eligibility criteria. However eligible studies were independently assessed by two reviewers with any disagreements resolved through discussions between the two reviewers. The literature search covers any research from the inception of time up to 15 December 2020, hence it covers the depth of research available.

4.3. Conclusions

Overall, there is sufficient evidence to demonstrate that OSA patients experience hearing deficits. However, few studies have conducted a comprehensive assessment and so the location of the hearing loss and the cause cannot be clearly defined. Research in children with OSA are also limited but it appears to affect hearing thresholds and auditory processing abilities. The limited data available with vestibular testing in OSA patients indicate signs of increased abnormality in vestibular function which can affect their quality of life. However, the mechanisms by which OSA affects hearing and balance and the extent to which this occurs requires more research.

The positive pressure from CPAP has been shown to increase MEP, in not only patients with OSA, but also patients with atelectasis and even healthy participants. This could be the mechanism by which hearing thresholds improve in patients with atelectasis and OSA patients with Meniere's disease. Nevertheless, a battery of audiological testing is required to confirm whether the changes in hearing thresholds is due to CPAP's effects on MEP. There are no data currently on how CPAP could affect the cochlea or the higher auditory pathways. CPAP's effects on the vestibular function also remain to be clarified.

This scoping review advocates for further quality research involving a more comprehensive approach and a battery of audiological and vestibular testing with different severities of OSA.

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Declaration of competing interest

There is no conflict of interest.

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Appendix A. Supplementary data

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