Otoacoustic Emissions Testing (for Indiana Only)

Policy Number: CS323IN.03
Effective Date: October 1, 2021

Instructions for Use

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Related Policies
None

Application

This Medical Policy only applies to the state of Indiana.

Coverage Rationale

Neonatal hearing screening as a preventive service using otoacoustic emissions (OAEs) is proven and medically necessary for infants who are 90 days or younger.

Otoacoustic emissions (OAEs) testing as a diagnostic service is proven and medically necessary for the evaluation of hearing loss in one or more of the following:

- Infants over 90 days old and children up to 4 years of age who did not pass or receive an initial hearing screening
- Children and adults who are unable to cooperate with other methods of hearing testing (e.g., individuals with autism or stroke)
- Children with developmental or delayed speech or language disorders
- Individuals with, acoustic trauma, noise induced hearing loss, or sudden hearing loss
- Individuals with Auditory Neuropathy or auditory processing disorder (APD), also known as central auditory processing disorder (CAPD)
- Individuals with Sensorineural Hearing Loss confirmed by audiometry
- Individuals with abnormal auditory function studies or failed hearing exam
- Individuals who may be feigning a hearing loss
- Monitoring of ototoxicity in individuals before, during, and after administration of agents known to be ototoxic (e.g., aminoglycosides, chemotherapy agents)

Note: Otoacoustic emissions tests should not be offered as part of an investigation of tinnitus unless the tinnitus is accompanied by other symptoms and signs. (NICE guideline NG155, 2020)

Auditory screening or diagnostic testing using otoacoustic emissions (OAEs) is unproven and not medically necessary for all other populations and conditions due to insufficient evidence of efficacy.

Definitions

Auditory Neuropathy (AN): Occurs as hearing loss in which the outer hair cells within the cochlea are present and functional, but sound information is not faithfully transmitted to the auditory nerve and brain properly. Also known as Auditory Neuropathy/auditory dys-synchrony (AN/AD) or Auditory Neuropathy spectrum disorder (ANSD).

Degree of Hearing Loss:

Degree of Hearing Loss	Range (dbHL = decibels hearing level)
Normal hearing	-10 to15 dBHL
Slight Loss	16 to 25 dBHL
Mild Loss	26 to 40 dBHL
Moderate Loss	41 to 55 dBHL
Moderately Severe Loss	56 to 70 dBHL
Severe Loss	71 to 90 dBHL
Profound Loss	91 dBHL or more

(ASHA, Type, Degree and Configuration of Hearing Loss, 2015; Clark, 1981)

Sensorineural Hearing Loss (SNHL): Occurs when there is damage to the inner ear (cochlea), or to the nerve pathways from the inner ear to the brain. Most of the time, SNHL cannot be medically or surgically corrected. This is the most common type of permanent hearing loss. (American Speech-Language-Hearing Association (ASHA) Sensorineural Hearing Loss)

Applicable Codes

The following list(s) of procedure and/or diagnosis codes is provided for reference purposes only and may not be all inclusive. Listing of a code in this policy does not imply that the service described by the code is a covered or non-covered health service. Benefit coverage for health services is determined by the member specific benefit plan document and applicable laws that may require coverage for a specific service. The inclusion of a code does not imply any right to reimbursement or guarantee claim payment. Other Policies and Guidelines may apply.

Coding Clarifications:

- CPT code 92558 should be used for screening. CPT codes 92587 and 92588 are used for diagnostic evaluations to confirm the presence or absence of hearing disorders.
- For more information, refer to the following website: http://leader.pubs.asha.org/article.aspx?articleid=2280157 [American Speech-Language-Hearing, New and Revised Otoacoustic Emissions (OAE) CPT Codes for 2012] (Accessed March 19, 2021)

CPT Code	Description
92558	Evoked otoacoustic emissions, screening (qualitative measurement of distortion product or transient evoked otoacoustic emissions), automated analysis
92587	Distortion product evoked otoacoustic emissions; limited evaluation (to confirm the presence or absence of hearing disorder, 3-6 frequencies) or transient evoked otoacoustic emissions, with interpretation and report
92588	Distortion product evoked otoacoustic emissions; comprehensive diagnostic evaluation (quantitative analysis of outer hair cell function by cochlear mapping, minimum of 12 frequencies), with interpretation and report

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Diagnosis Code	Description
A17.0	Tuberculous meningitis
A39.0	Meningococcal meningitis

Diagnosis Code	Description
A52.13	Late syphilitic meningitis
A80.0	Acute paralytic poliomyelitis, vaccine-associated
A80.1	Acute paralytic poliomyelitis, wild virus, imported
A80.2	Acute paralytic poliomyelitis, wild virus, indigenous
A80.30	Acute paralytic poliomyelitis, unspecified
A80.39	Other acute paralytic poliomyelitis
A80.9	Acute poliomyelitis, unspecified
A87.0	Enteroviral meningitis
A87.8	Other viral meningitis
A87.9	Viral meningitis, unspecified
B02.1	Zoster meningitis
B26.1	Mumps meningitis
B45.1	Cerebral cryptococcosis
B83.2	Angiostrongyliasis due to Parastrongylus cantonensis
B91	Sequelae of poliomyelitis
F01.50	Vascular dementia without behavioral disturbance
F01.51	Vascular dementia with behavioral disturbance
F02.80	Dementia in other diseases classified elsewhere without behavioral disturbance
F02.81	Dementia in other diseases classified elsewhere with behavioral disturbance
F03.90	Unspecified dementia without behavioral disturbance
F03.91	Unspecified dementia with behavioral disturbance
F07.9	Unspecified personality and behavioral disorder due to known physiological condition
F09	Unspecified mental disorder due to known physiological condition
F44.6	Conversion disorder with sensory symptom or deficit
F45.8	Other somatoform disorders
F68.10	Factitious disorder imposed on self, unspecified
F68.12	Factitious disorder imposed on self, with predominantly physical signs and symptoms
F68.13	Factitious disorder imposed on self, with combined psychological and physical signs and symptoms
F71	Moderate intellectual disabilities
F72	Severe intellectual disabilities
F73	Profound intellectual disabilities
F78.A1	SYNGAP1-related intellectual disability
F78.A9	Other genetic related intellectual disability
F79	Unspecified intellectual disabilities
F80.1	Expressive language disorder
F80.2	Mixed receptive-expressive language disorder
F80.4	Speech and language development delay due to hearing loss
F80.82	Social pragmatic communication disorder
F80.89	Other developmental disorders of speech and language
F80.9	Developmental disorder of speech and language, unspecified
F84.0	Autistic disorder
F84.2	Rett's syndrome

Diagnosis Code	Description
F84.3	Other childhood disintegrative disorder
F84.5	Asperger's syndrome
F84.8	Other pervasive developmental disorders
F84.9	Pervasive developmental disorder, unspecified
F90.1	Attention-deficit hyperactivity disorder, predominantly hyperactive type
F90.2	Attention-deficit hyperactivity disorder, combined type
F90.8	Attention-deficit hyperactivity disorder, other type
F95.2	Tourette's disorder
G00.0	Hemophilus meningitis
G00.1	Pneumococcal meningitis
G00.2	Streptococcal meningitis
G00.3	Staphylococcal meningitis
G00.8	Other bacterial meningitis
G00.9	Bacterial meningitis, unspecified
G01	Meningitis in bacterial diseases classified elsewhere
G02	Meningitis in other infectious and parasitic diseases classified elsewhere
G03.0	Nonpyogenic meningitis
G03.1	Chronic meningitis
G03.2	Benign recurrent meningitis [Mollaret]
G03.8	Meningitis due to other specified causes
G03.9	Meningitis, unspecified
G04.2	Bacterial meningoencephalitis and meningomyelitis, not elsewhere classified
G20	Parkinson's disease
G21.0	Malignant neuroleptic syndrome
G21.11	Neuroleptic induced parkinsonism
G21.3	Postencephalitic parkinsonism
G21.4	Vascular parkinsonism
G21.8	Other secondary parkinsonism
G21.9	Secondary parkinsonism, unspecified
G23.0	Hallervorden-Spatz disease
G23.1	Progressive supranuclear ophthalmoplegia [Steele-Richardson-Olszewski]
G23.2	Striatonigral degeneration
G23.8	Other specified degenerative diseases of basal ganglia
G23.9	Degenerative disease of basal ganglia, unspecified
G30.0	Alzheimer's disease with early onset
G30.1	Alzheimer's disease with late onset
G30.8	Other Alzheimer's disease
G30.9	Alzheimer's disease, unspecified
G46.3	Brain stem stroke syndrome
G46.4	Cerebellar stroke syndrome
G46.5	Pure motor lacunar syndrome
G46.6	Pure sensory lacunar syndrome

Diagnosis Code	Description
G46.7	Other lacunar syndromes
G46.8	Other vascular syndromes of brain in cerebrovascular diseases
G52.7	Disorders of multiple cranial nerves
G60.8	Other hereditary and idiopathic neuropathies
G72.3	Periodic paralysis
G80.0	Spastic quadriplegic cerebral palsy
G80.1	Spastic diplegic cerebral palsy
G80.2	Spastic hemiplegic cerebral palsy
G80.3	Athetoid cerebral palsy
G80.4	Ataxic cerebral palsy
G80.8	Other cerebral palsy
G80.9	Cerebral palsy, unspecified
G83.81	Brown-Sequard syndrome
G83.82	Anterior cord syndrome
G83.83	Posterior cord syndrome
G83.84	Todd's paralysis (postepileptic)
G83.89	Other specified paralytic syndromes
G83.9	Paralytic syndrome, unspecified
G90.09	Other idiopathic peripheral autonomic neuropathy
G90.3	Multi-system degeneration of the autonomic nervous system
G93.1	Anoxic brain damage, not elsewhere classified
H83.01	Labyrinthitis, right ear
H83.02	Labyrinthitis, left ear
H83.03	Labyrinthitis, bilateral
H83.09	Labyrinthitis, unspecified ear
H83.3X1	Noise effects on right inner ear
H83.3X2	Noise effects on left inner ear
H83.3X3	Noise effects on inner ear, bilateral
H83.3X9	Noise effects on inner ear, unspecified ear
H90.3	Sensorineural hearing loss, bilateral
H90.41	Sensorineural hearing loss, unilateral, right ear, with unrestricted hearing on the contralateral side
H90.42	Sensorineural hearing loss, unilateral, left ear, with unrestricted hearing on the contralateral side
H90.5	Unspecified sensorineural hearing loss
H90.6	Mixed conductive and sensorineural hearing loss, bilateral
H90.71	Mixed conductive and sensorineural hearing loss, unilateral, right ear, with unrestricted hearing on the contralateral side
H90.72	Mixed conductive and sensorineural hearing loss, unilateral, left ear, with unrestricted hearing on the contralateral side
H90.8	Mixed conductive and sensorineural hearing loss, unspecified
H90.A11	Conductive hearing loss, unilateral, right ear with restricted hearing on the contralateral side
H90.A12	Conductive hearing loss, unilateral, left ear with restricted hearing on the contralateral side
H90.A21	Sensorineural hearing loss, unilateral, right ear, with restricted hearing on the contralateral side

Diagnosis Code	Description
H90.A22	Sensorineural hearing loss, unilateral, left ear, with restricted hearing on the contralateral side
H90.A31	Mixed conductive and sensorineural hearing loss, unilateral, right ear with restricted hearing on the contralateral side
H90.A32	Mixed conductive and sensorineural hearing loss, unilateral, left ear with restricted hearing on the contralateral side
H91.01	Ototoxic hearing loss, right ear
H91.02	Ototoxic hearing loss, left ear
H91.03	Ototoxic hearing loss, bilateral
H91.09	Ototoxic hearing loss, unspecified ear
H91.20	Sudden idiopathic hearing loss, unspecified ear
H91.21	Sudden idiopathic hearing loss, right ear
H91.22	Sudden idiopathic hearing loss, left ear
H91.23	Sudden idiopathic hearing loss, bilateral
H91.8X1	Other specified hearing loss, right ear
H91.8X2	Other specified hearing loss, left ear
H91.8X3	Other specified hearing loss, bilateral
H91.8X9	Other specified hearing loss, unspecified ear
H93.011	Transient ischemic deafness, right ear
H93.012	Transient ischemic deafness, left ear
H93.013	Transient ischemic deafness, bilateral
H93.019	Transient ischemic deafness, unspecified ear
H93.211	Auditory recruitment, right ear
H93.212	Auditory recruitment, left ear
H93.213	Auditory recruitment, bilateral
H93.219	Auditory recruitment, unspecified ear
H93.221	Diplacusis, right ear
H93.222	Diplacusis, left ear
H93.223	Diplacusis, bilateral
H93.229	Diplacusis, unspecified ear
H93.231	Hyperacusis, right ear
H93.232	Hyperacusis, left ear
H93.233	Hyperacusis, bilateral
H93.239	Hyperacusis, unspecified ear
H93.241	Temporary auditory threshold shift, right ear
H93.242	Temporary auditory threshold shift, left ear
H93.243	Temporary auditory threshold shift, bilateral
H93.249	Temporary auditory threshold shift, unspecified ear
H93.25	Central auditory processing disorder
H93.291	Other abnormal auditory perceptions, right ear
H93.292	Other abnormal auditory perceptions, left ear
H93.293	Other abnormal auditory perceptions, bilateral
H93.299	Other abnormal auditory perceptions, unspecified ear

Diagnosis Code	Description
I67.2	Cerebral atherosclerosis
I67.81	Acute cerebrovascular insufficiency
I67.82	Cerebral ischemia
I67.850	Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy
I67.89	Other cerebrovascular disease
I68.0	Cerebral amyloid angiopathy
I68.8	Other cerebrovascular disorders in diseases classified elsewhere
I69.00	Unspecified sequelae of nontraumatic subarachnoid hemorrhage
I69.010	Attention and concentration deficit following nontraumatic subarachnoid hemorrhage
I69.011	Memory deficit following nontraumatic subarachnoid hemorrhage
I69.012	Visuospatial deficit and spatial neglect following nontraumatic subarachnoid hemorrhage
I69.013	Psychomotor deficit following nontraumatic subarachnoid hemorrhage
I69.014	Frontal lobe and executive function deficit following nontraumatic subarachnoid hemorrhage
I69.015	Cognitive social or emotional deficit following nontraumatic subarachnoid hemorrhage
I69.018	Other symptoms and signs involving cognitive functions following nontraumatic subarachnoid hemorrhage
I69.019	Unspecified symptoms and signs involving cognitive functions following nontraumatic subarachnoid hemorrhage
I69.020	Aphasia following nontraumatic subarachnoid hemorrhage
I69.021	Dysphasia following nontraumatic subarachnoid hemorrhage
I69.022	Dysarthria following nontraumatic subarachnoid hemorrhage
I69.023	Fluency disorder following nontraumatic subarachnoid hemorrhage
I69.028	Other speech and language deficits following nontraumatic subarachnoid hemorrhage
I69.090	Apraxia following nontraumatic subarachnoid hemorrhage
I69.091	Dysphagia following nontraumatic subarachnoid hemorrhage
I69.092	Facial weakness following nontraumatic subarachnoid hemorrhage
I69.093	Ataxia following nontraumatic subarachnoid hemorrhage
I69.098	Other sequelae following nontraumatic subarachnoid hemorrhage
I69.10	Unspecified sequelae of nontraumatic intracerebral hemorrhage
I69.110	Attention and concentration deficit following nontraumatic intracerebral hemorrhage
I69.111	Memory deficit following nontraumatic intracerebral hemorrhage
I69.112	Visuospatial deficit and spatial neglect following nontraumatic intracerebral hemorrhage
I69.113	Psychomotor deficit following nontraumatic intracerebral hemorrhage
I69.114	Frontal lobe and executive function deficit following nontraumatic intracerebral hemorrhage
I69.115	Cognitive social or emotional deficit following nontraumatic intracerebral hemorrhage
I69.118	Other symptoms and signs involving cognitive functions following nontraumatic intracerebral hemorrhage
I69.119	Unspecified symptoms and signs involving cognitive functions following nontraumatic intracerebral hemorrhage
I69.120	Aphasia following nontraumatic intracerebral hemorrhage
I69.121	Dysphasia following nontraumatic intracerebral hemorrhage
I69.122	Dysarthria following nontraumatic intracerebral hemorrhage
I69.123	Fluency disorder following nontraumatic intracerebral hemorrhage

Diagnosis Code	Description
I69.128	Other speech and language deficits following nontraumatic intracerebral hemorrhage
I69.190	Apraxia following nontraumatic intracerebral hemorrhage
I69.191	Dysphagia following nontraumatic intracerebral hemorrhage
I69.192	Facial weakness following nontraumatic intracerebral hemorrhage
I69.193	Ataxia following nontraumatic intracerebral hemorrhage
I69.198	Other sequelae of nontraumatic intracerebral hemorrhage
I69.20	Unspecified sequelae of other nontraumatic intracranial hemorrhage
I69.210	Attention and concentration deficit following other nontraumatic intracranial hemorrhage
I69.211	Memory deficit following other nontraumatic intracranial hemorrhage
I69.212	Visuospatial deficit and spatial neglect following other nontraumatic intracranial hemorrhage
I69.213	Psychomotor deficit following other nontraumatic intracranial hemorrhage
I69.214	Frontal lobe and executive function deficit following other nontraumatic intracranial hemorrhage
I69.215	Cognitive social or emotional deficit following other nontraumatic intracranial hemorrhage
I69.218	Other symptoms and signs involving cognitive functions following other nontraumatic intracranial hemorrhage
I69.219	Unspecified symptoms and signs involving cognitive functions following other nontraumatic intracranial hemorrhage
I69.220	Aphasia following other nontraumatic intracranial hemorrhage
I69.221	Dysphasia following other nontraumatic intracranial hemorrhage
I69.222	Dysarthria following other nontraumatic intracranial hemorrhage
I69.223	Fluency disorder following other nontraumatic intracranial hemorrhage
I69.228	Other speech and language deficits following other nontraumatic intracranial hemorrhage
I69.290	Apraxia following other nontraumatic intracranial hemorrhage
I69.291	Dysphagia following other nontraumatic intracranial hemorrhage
I69.292	Facial weakness following other nontraumatic intracranial hemorrhage
I69.293	Ataxia following other nontraumatic intracranial hemorrhage
I69.298	Other sequelae of other nontraumatic intracranial hemorrhage
I69.30	Unspecified sequelae of cerebral infarction
I69.310	Attention and concentration deficit following cerebral infarction
I69.311	Memory deficit following cerebral infarction
I69.312	Visuospatial deficit and spatial neglect following cerebral infarction
I69.313	Psychomotor deficit following cerebral infarction
I69.314	Frontal lobe and executive function deficit following cerebral infarction
I69.315	Cognitive social or emotional deficit following cerebral infarction
I69.318	Other symptoms and signs involving cognitive functions following cerebral infarction
I69.319	Unspecified symptoms and signs involving cognitive functions following cerebral infarction
I69.320	Aphasia following cerebral infarction
I69.321	Dysphasia following cerebral infarction
I69.322	Dysarthria following cerebral infarction
I69.323	Fluency disorder following cerebral infarction
I69.328	Other speech and language deficits following cerebral infarction
I69.390	Apraxia following cerebral infarction

Diagnosis Code	Description
I69.391	Dysphagia following cerebral infarction
I69.392	Facial weakness following cerebral infarction
I69.393	Ataxia following cerebral infarction
I69.398	Other sequelae of cerebral infarction
I69.80	Unspecified sequelae of other cerebrovascular disease
I69.810	Attention and concentration deficit following other cerebrovascular disease
I69.811	Memory deficit following other cerebrovascular disease
I69.812	Visuospatial deficit and spatial neglect following other cerebrovascular disease
I69.813	Psychomotor deficit following other cerebrovascular disease
I69.814	Frontal lobe and executive function deficit following other cerebrovascular disease
I69.815	Cognitive social or emotional deficit following other cerebrovascular disease
I69.818	Other symptoms and signs involving cognitive functions following other cerebrovascular disease
I69.819	Unspecified symptoms and signs involving cognitive functions following other cerebrovascular disease
I69.820	Aphasia following other cerebrovascular disease
I69.821	Dysphasia following other cerebrovascular disease
I69.822	Dysarthria following other cerebrovascular disease
I69.823	Fluency disorder following other cerebrovascular disease
I69.828	Other speech and language deficits following other cerebrovascular disease
I69.890	Apraxia following other cerebrovascular disease
I69.891	Dysphagia following other cerebrovascular disease
I69.892	Facial weakness following other cerebrovascular disease
I69.893	Ataxia following other cerebrovascular disease
I69.898	Other sequelae of other cerebrovascular disease
I69.90	Unspecified sequelae of unspecified cerebrovascular disease
I69.910	Attention and concentration deficit following unspecified cerebrovascular disease
I69.911	Memory deficit following unspecified cerebrovascular disease
I69.912	Visuospatial deficit and spatial neglect following unspecified cerebrovascular disease
I69.913	Psychomotor deficit following unspecified cerebrovascular disease
I69.914	Frontal lobe and executive function deficit following unspecified cerebrovascular disease
I69.915	Cognitive social or emotional deficit following unspecified cerebrovascular disease
I69.918	Other symptoms and signs involving cognitive functions following unspecified cerebrovascular disease
I69.919	Unspecified symptoms and signs involving cognitive functions following unspecified cerebrovascular disease
I69.920	Aphasia following unspecified cerebrovascular disease
I69.921	Dysphasia following unspecified cerebrovascular disease
I69.922	Dysarthria following unspecified cerebrovascular disease
I69.923	Fluency disorder following unspecified cerebrovascular disease
I69.928	Other speech and language deficits following unspecified cerebrovascular disease
I69.990	Apraxia following unspecified cerebrovascular disease
I69.991	Dysphagia following unspecified cerebrovascular disease
I69.992	Facial weakness following unspecified cerebrovascular disease
I69.993	Ataxia following unspecified cerebrovascular disease

Diagnosis Code	Description
I69.998	Other sequelae following unspecified cerebrovascular disease
I97.810	Intraoperative cerebrovascular infarction during cardiac surgery
I97.811	Intraoperative cerebrovascular infarction during other surgery
I97.820	Postprocedural cerebrovascular infarction following cardiac surgery
I97.821	Postprocedural cerebrovascular infarction following other surgery
P11.1	Other specified brain damage due to birth injury
P11.2	Unspecified brain damage due to birth injury
Q90.0	Trisomy 21, nonmosaicism (meiotic nondisjunction)
Q90.1	Trisomy 21, mosaicism (mitotic nondisjunction)
Q90.2	Trisomy 21, translocation
Q90.9	Down syndrome, unspecified
R41.89	Other symptoms and signs involving cognitive functions and awareness
R42	Dizziness and giddiness
R47.01	Aphasia
R47.02	Dysphasia
R47.1	Dysarthria and anarthria
R49.1	Aphonia
R62.0	Delayed milestone in childhood
R94.120	Abnormal auditory function study
R94.121	Abnormal vestibular function study
R94.128	Abnormal results of other function studies of ear and other special senses
S09.20XA	Traumatic rupture of unspecified ear drum, initial encounter
S09.21XA	Traumatic rupture of right ear drum, initial encounter
S09.22XA	Traumatic rupture of left ear drum, initial encounter
S09.311A	Primary blast injury of right ear, initial encounter
S09.312A	Primary blast injury of left ear, initial encounter
S09.313A	Primary blast injury of ear, bilateral, initial encounter
S09.319A	Primary blast injury of unspecified ear, initial encounter
S12.000A	Unspecified displaced fracture of first cervical vertebra, initial encounter for closed fracture
S12.000B	Unspecified displaced fracture of first cervical vertebra, initial encounter for open fracture
S12.001A	Unspecified nondisplaced fracture of first cervical vertebra, initial encounter for closed fracture
S12.001B	Unspecified nondisplaced fracture of first cervical vertebra, initial encounter for open fracture
S12.100A	Unspecified displaced fracture of second cervical vertebra, initial encounter for closed fracture
S12.100B	Unspecified displaced fracture of second cervical vertebra, initial encounter for open fracture
S12.101A	Unspecified nondisplaced fracture of second cervical vertebra, initial encounter for closed fracture
S12.101B	Unspecified nondisplaced fracture of second cervical vertebra, initial encounter for open fracture
S12.200A	Unspecified displaced fracture of third cervical vertebra, initial encounter for closed fracture
S12.200B	Unspecified displaced fracture of third cervical vertebra, initial encounter for open fracture
S12.201A	Unspecified nondisplaced fracture of third cervical vertebra, initial encounter for closed fracture
S12.201B	Unspecified nondisplaced fracture of third cervical vertebra, initial encounter for open fracture
S12.300A	Unspecified displaced fracture of fourth cervical vertebra, initial encounter for closed fracture
S12.300B	Unspecified displaced fracture of fourth cervical vertebra, initial encounter for open fracture

Diagnosis Code	Description
S12.301A	Unspecified nondisplaced fracture of fourth cervical vertebra, initial encounter for closed fracture
S12.301B	Unspecified nondisplaced fracture of fourth cervical vertebra, initial encounter for open fracture
S12.400A	Unspecified displaced fracture of fifth cervical vertebra, initial encounter for closed fracture
S12.400B	Unspecified displaced fracture of fifth cervical vertebra, initial encounter for open fracture
S12.401A	Unspecified nondisplaced fracture of fifth cervical vertebra, initial encounter for closed fracture
S12.401B	Unspecified nondisplaced fracture of fifth cervical vertebra, initial encounter for open fracture
S12.500A	Unspecified displaced fracture of sixth cervical vertebra, initial encounter for closed fracture
S12.500B	Unspecified displaced fracture of sixth cervical vertebra, initial encounter for open fracture
S12.501A	Unspecified nondisplaced fracture of sixth cervical vertebra, initial encounter for closed fracture
S12.501B	Unspecified nondisplaced fracture of sixth cervical vertebra, initial encounter for open fracture
S12.600A	Unspecified displaced fracture of seventh cervical vertebra, initial encounter for closed fracture
S12.600B	Unspecified displaced fracture of seventh cervical vertebra, initial encounter for open fracture
S12.601A	Unspecified nondisplaced fracture of seventh cervical vertebra, initial encounter for closed fracture
S12.601B	Unspecified nondisplaced fracture of seventh cervical vertebra, initial encounter for open fracture
S14.101A	Unspecified injury at C1 level of cervical spinal cord, initial encounter
S14.102A	Unspecified injury at C2 level of cervical spinal cord, initial encounter
S14.103A	Unspecified injury at C3 level of cervical spinal cord, initial encounter
S14.104A	Unspecified injury at C4 level of cervical spinal cord, initial encounter
S14.105A	Unspecified injury at C5 level of cervical spinal cord, initial encounter
S14.106A	Unspecified injury at C6 level of cervical spinal cord, initial encounter
S14.107A	Unspecified injury at C7 level of cervical spinal cord, initial encounter
S14.111A	Complete lesion at C1 level of cervical spinal cord, initial encounter
S14.112A	Complete lesion at C2 level of cervical spinal cord, initial encounter
S14.113A	Complete lesion at C3 level of cervical spinal cord, initial encounter
S14.114A	Complete lesion at C4 level of cervical spinal cord, initial encounter
S14.115A	Complete lesion at C5 level of cervical spinal cord, initial encounter
S14.116A	Complete lesion at C6 level of cervical spinal cord, initial encounter
S14.117A	Complete lesion at C7 level of cervical spinal cord, initial encounter
S14.121A	Central cord syndrome at C1 level of cervical spinal cord, initial encounter
S14.122A	Central cord syndrome at C2 level of cervical spinal cord, initial encounter
S14.123A	Central cord syndrome at C3 level of cervical spinal cord, initial encounter
S14.124A	Central cord syndrome at C4 level of cervical spinal cord, initial encounter
S14.125A	Central cord syndrome at C5 level of cervical spinal cord, initial encounter
S14.126A	Central cord syndrome at C6 level of cervical spinal cord, initial encounter
S14.127A	Central cord syndrome at C7 level of cervical spinal cord, initial encounter
S14.131A	Anterior cord syndrome at C1 level of cervical spinal cord, initial encounter
S14.132A	Anterior cord syndrome at C2 level of cervical spinal cord, initial encounter
S14.133A	Anterior cord syndrome at C3 level of cervical spinal cord, initial encounter
S14.134A	Anterior cord syndrome at C4 level of cervical spinal cord, initial encounter
S14.135A	Anterior cord syndrome at C5 level of cervical spinal cord, initial encounter
S14.136A	Anterior cord syndrome at C6 level of cervical spinal cord, initial encounter
S14.137A	Anterior cord syndrome at C7 level of cervical spinal cord, initial encounter

Diagnosis Code	Description
S14.151A	Other incomplete lesion at C1 level of cervical spinal cord, initial encounter
S14.152A	Other incomplete lesion at C2 level of cervical spinal cord, initial encounter
S14.153A	Other incomplete lesion at C3 level of cervical spinal cord, initial encounter
S14.154A	Other incomplete lesion at C4 level of cervical spinal cord, initial encounter
S14.155A	Other incomplete lesion at C5 level of cervical spinal cord, initial encounter
S14.156A	Other incomplete lesion at C6 level of cervical spinal cord, initial encounter
S14.157A	Other incomplete lesion at C7 level of cervical spinal cord, initial encounter
T20.011S	Burn of unspecified degree of right ear [any part, except ear drum], sequela
T20.012S	Burn of unspecified degree of left ear [any part, except ear drum], sequela
T20.019S	Burn of unspecified degree of unspecified ear [any part, except ear drum], sequela
T20.111S	Burn of first degree of right ear [any part, except ear drum], sequela
T20.112S	Burn of first degree of left ear [any part, except ear drum], sequela
T20.119S	Burn of first degree of unspecified ear [any part, except ear drum], sequela
T20.211S	Burn of second degree of right ear [any part, except ear drum], sequela
T20.212S	Burn of second degree of left ear [any part, except ear drum], sequela
T20.219S	Burn of second degree of unspecified ear [any part, except ear drum], sequela
T20.311S	Burn of third degree of right ear [any part, except ear drum], sequela
T20.312S	Burn of third degree of left ear [any part, except ear drum], sequela
T20.319S	Burn of third degree of unspecified ear [any part, except ear drum], sequela
T20.411S	Corrosion of unspecified degree of right ear [any part, except ear drum], sequela
T20.412S	Corrosion of unspecified degree of left ear [any part, except ear drum], sequela
T20.419S	Corrosion of unspecified degree of unspecified ear [any part, except ear drum], sequela
T20.511S	Corrosion of first degree of right ear [any part, except ear drum], sequela
T20.512S	Corrosion of first degree of left ear [any part, except ear drum], sequela
T20.519S	Corrosion of first degree of unspecified ear [any part, except ear drum], sequela
T20.611S	Corrosion of second degree of right ear [any part, except ear drum], sequela
T20.612S	Corrosion of second degree of left ear [any part, except ear drum], sequela
T20.619S	Corrosion of second degree of unspecified ear [any part, except ear drum], sequela
T20.711S	Corrosion of third degree of right ear [any part, except ear drum], sequela
T20.712S	Corrosion of third degree of left ear [any part, except ear drum], sequela
T20.719S	Corrosion of third degree of unspecified ear [any part, except ear drum], sequela
T28.411S	Burn of right ear drum, sequela
T28.412S	Burn of left ear drum, sequela
T28.419S	Burn of unspecified ear drum, sequela
T28.911S	Corrosions of right ear drum, sequela
T28.912S	Corrosions of left ear drum, sequela
T28.919S	Corrosions of unspecified ear drum, sequela
T36.5X1A	Poisoning by aminoglycosides, accidental (unintentional), initial encounter
T36.5X1D	Poisoning by aminoglycosides, accidental (unintentional), subsequent encounter
T36.5X1S	Poisoning by aminoglycosides, accidental (unintentional), sequela
T36.5X2A	Poisoning by aminoglycosides, intentional self-harm, initial encounter
T36.5X2D	Poisoning by aminoglycosides, intentional self-harm, subsequent encounter

Diagnosis Code	Description
T36.5X2S	Poisoning by aminoglycosides, intentional self-harm, sequela
T36.5X3A	Poisoning by aminoglycosides, assault, initial encounter
T36.5X3D	Poisoning by aminoglycosides, assault, subsequent encounter
T36.5X3S	Poisoning by aminoglycosides, assault, sequela
T36.5X4A	Poisoning by aminoglycosides, undetermined, initial encounter
T36.5X4D	Poisoning by aminoglycosides, undetermined, subsequent encounter
T36.5X4S	Poisoning by aminoglycosides, undetermined, sequela
T36.5X5A	Adverse effect of aminoglycosides, initial encounter
T36.5X5D	Adverse effect of aminoglycosides, subsequent encounter
T36.5X5S	Adverse effect of aminoglycosides, sequela
T36.6X1A	Poisoning by rifampicins, accidental (unintentional), initial encounter
T36.6X2A	Poisoning by rifampicins, intentional self-harm, initial encounter
T36.6X3A	Poisoning by rifampicins, assault, initial encounter
T36.6X4A	Poisoning by rifampicins, undetermined, initial encounter
T36.6X5A	Adverse effect of rifampicins, initial encounter
T36.8X1A	Poisoning by other systemic antibiotics, accidental (unintentional), initial encounter
T36.8X2A	Poisoning by other systemic antibiotics, intentional self-harm, initial encounter
T36.8X3A	Poisoning by other systemic antibiotics, assault, initial encounter
T36.8X4A	Poisoning by other systemic antibiotics, undetermined, initial encounter
T36.8X5A	Adverse effect of other systemic antibiotics, initial encounter
T45.1X1A	Poisoning by antineoplastic and immunosuppressive drugs, accidental (unintentional), initial encounter
T45.1X2A	Poisoning by antineoplastic and immunosuppressive drugs, intentional self-harm, initial encounter
T45.1X3A	Poisoning by antineoplastic and immunosuppressive drugs, assault, initial encounter
T45.1X4A	Poisoning by antineoplastic and immunosuppressive drugs, undetermined, initial encounter
T45.1X5A	Adverse effect of antineoplastic and immunosuppressive drugs, initial encounter
T45.1X5S	Adverse effect of antineoplastic and immunosuppressive drugs, sequela
T79.8XXA	Other early complications of trauma, initial encounter
Z01.110	Encounter for hearing examination following failed hearing screening
Z01.118	Encounter for examination of ears and hearing with other abnormal findings
Z13.40	Encounter for screening for unspecified developmental delays
Z13.41	Encounter for autism screening
Z13.42	Encounter for screening for global developmental delays (milestones)
Z13.49	Encounter for screening for other developmental delays
Z13.5	Encounter for screening for eye and ear disorders
Z57.0	Occupational exposure to noise
Z76.5	Malingerer [conscious simulation]
Z77.122	Contact with and (suspected) exposure to noise
Z87.820	Personal history of traumatic brain injury
Z92.21	Personal history of antineoplastic chemotherapy

Description of Services

Otoacoustic emissions (OAEs) are physiologic measurements of the response of the cochlear outer hair cells to acoustic stimuli and are used to assess cochlear integrity and preneural function. The test only detects hearing disorders that affect the cochlea and the pathway to the inner ear. OAEs do not diagnosis hearing loss; they reflect inner ear mechanics and provide information that further defines the auditory system's integrity and sensitivity. OAEs that are recorded in response to auditory signals are known as evoked OAEs. OAEs are measured by acoustic stimuli such as a series of very brief clicks to the ear through a probe that is inserted in the outer third of the ear canal. The probe contains loudspeakers that generate the clicks and a microphone for measuring the resulting OAEs. The sound moves along the pathway from the outer ear, through the middle ear and into the cochlea. When the cochlea is functioning properly, an otoacoustic emission is produced that travels back out through the middle and the outer ear. This emission is calculated by the probe and analyzed by a computer. When an emission is adequate, "pass" is displayed on the monitor. In instances of dysfunction or blockage along the pathway to the cochlea, the equipment will be unable to measure the emission, and the monitor will display "fail" or "refer." (AAA, 2011; ASHA, 2004). OAE testing requires no behavioral or interactive feedback by the individual being tested.

OAEs are used as a screening test for hearing in newborns. Other potential applications of OAE testing include screening children or at-risk populations for hearing loss and characterizing sensitivity and functional hearing loss and differentiating sensory from neural components in people with known hearing loss.

OAE devices use either transient evoked OAE (TEOAE) or distortion product EOE (DPOAE) technology. TEOAE devices emit a single brief click that covers a broad frequency range. DPOAE devices emit two brief tones set at two separate frequencies. TEOAEs are used to screen infants, validate other tests, and assess cochlear function, and DPOAEs are used to assess cochlear damage, ototoxicity, and noise-induced damage. Spontaneous otoacoustic emissions (SOAEs) are sounds emitted without an acoustic stimulus (i.e., spontaneously). Stimulus-frequency otoacoustic emissions (SFOAEs) are sounds emitted in response to a continuous tone. At present, SOAEs and SFOAEs are not used clinically.

The OAE measures are effective for screening middle-ear abnormalities and moderate or severe degrees of hearing loss, because normal OAE responses are not obtained if hearing thresholds are approximately 30- to 40-dB hearing levels or higher. A "failed" OAE test only implies that a hearing loss of more than 30 to 40 dB may exist or that the middle-ear status is abnormal. The OAE test does not further quantify hearing loss or hearing threshold level.

The OAE test also does not assess the integrity of the neural transmission of sound from the eighth nerve to the brainstem and, therefore, will miss Auditory Neuropathy (AN) and other neuronal abnormalities. Therefore, used in combination with auditory brainstem response (ABR) testing, OAE will assist in diagnosing AN. The hallmark of AN is an absent or very abnormal ABR reading together with a normal OAE reading. A normal OAE reading is a sign that the outer hair cells are working normally. (Harlor, 2009; National Institutes for Health, 2018)

Clinical Evidence

Otoacoustic Emissions (OAEs) for Neonatal Hearing Screening

Evidence from the peer-reviewed published scientific literature, textbook, some professional societies, and the U.S. Preventive Services Task Force support the use of Otoacoustic Emissions (OAEs) testing for use in newborns as a preventive service in infants who are 90 days or younger.

A retrospective study conducted by Güven (2019) evaluated the screening results of 2,653 newborns born between January 2013 and May 2017 according to the type of delivery (i.e., vaginal versus caesarean section). The study intentionally excluded any newborns that had any risk factors as defined by the 1994 position statement by the Joint Committee on Infant Hearing. Based on the results of the study, the author concluded that the mode of delivery was not identified to have a significant effect on the results of neonatal hearing screening tests. However, the authors found that infants, regardless of the mode of delivery, were observed to be more successful in the screening test when given beyond 48 hours after birth and concluded that performing the OAE test 15 days to one month after birth would aid in eliminating the possibility of false positives in hearing loss; thus, allaying unnecessary parental anxiety and reduce costs.

Escobar-Ipuz, et al. (2019) also conducted a retrospective study collecting data on OAE testing evaluation on 9,698 newborns from 2007 to 2017. The screening protocol for included three phases. In the first phase, 9,390 newborns received OAE testing prior to discharge with 8245 (87.8%) passing the screening test ad 114 (12.1%) presenting an abnormal OAE and were included in the second screening phase. A repeat OAE examination was performed on 177 newborns (94) in the second phase with 87.3% passing the test and 136 newborns (12.6%) failing the retest and being referred to continue on to phase three. Furthermore, 181 newborns (1.8%) presented high-risk factors at birth and were also included in this third phase. However, in the registries of children referred to this phase, only 255 (80%) ABR evaluations were confirmed. In total, 227 newborns (2.3%) were missed from the first to third phases of the screening process. According to the database of the clinical neurophysiology service, ABRs evaluations were performed in 352 newborns referred between December 2007 and December 2017. Of this sample, 38.9% were boys and 61.1% were girls. From among cases underwent ABR, 34% of newborns did not pass the OAEs. The most common risk factor was prematurity (with admission to the neonatal intensive care unit for more than five days), affecting 28%. Abnormal ABRs waveforms were found in 43.9%, with 12.3% having a sensorineural hearing loss, 26.5% showing mixed hearing loss and, conductive hearing loss being present in 61.9%. Considering sensorineural hearing loss and other types of severe hearing loss, affected patients constituted only 1.7% of the total number of individuals studied. Finally, regarding quality control of the program participation in the first phase of care included 97.2% of all newborns, yielding a third phase referral rate of 2.9%, confirmation of a diagnosis before the fourth month of life in more than 90% of cases with an average of 3.4 months of age, and a hearing impairment detection rate as an outcome indicator of 4.5%. The authors concluded that their data was similar to those of previous studies on screening for hearing loss in newborns and demonstrated the advantages of carrying out this protocol in three phases using the otoacoustic emissions together with auditory brainstem response, diagnostic tools that remain as a Gold Standard to ensure timely referrals in the early stages of development, avoiding future disabilities.

Akinpelu et al. (2014) reviewed ten articles on eligible studies published from January 1990 until August 2012 involving a total of 119,714 newborn participants. The main objective of this review was to determine the effects of different screening protocols on the referral rates and positive predictive values (PPV) of the OQE newborn screening test. Data extracted included the number of newborns screened, age at screening, OAE pass criteria, frequencies screened, number of retests, referral rates, and the number of newborns identified with permanent congenital hearing loss. The results found that the pooled referral rate was 5.5%. Individual referral rates ranged from 1.3% to 39%; with positive predictive values (PPS) from 2 to 40%. Increasing the age at initial screening and performing retests reduced the referral rate. The authors concluded that delaying newborn hearing screening improves test results but may not be practical in all contexts. The use of higher frequencies and more sophisticated OAE devices may be useful approaches to ensure better performance of the OAE test in newborn hearing screening.

Another group of investigators compared clinical outcomes, including speech and language development, in 25 infants who were screened as part of the Colorado Universal Newborn Screening program with outcomes in 25 matched infants who were born in a hospital without a universal newborn hearing screening program (Yoshinaga-Itano et al., 2000). This study found that children who were identified as hearing impaired through the hospital-based newborn hearing screening program had significantly better scores on tests of speech and language development than did children who were identified later.

A controlled trial which involved 53,781 newborns provided a direct comparison of hearing impairment detection rates during periods of newborn hearing screening and no screening in the same hospitals (Wessex Universal Hearing Screening Trial, 1998). During the trial, 25,609 infants were born during a period of screening and underwent a two-stage screening test, with transient evoked otoacoustic emissions (TEOAE) at birth, followed by automated auditory brainstem response (AABR) before discharge if the first screen was failed. If the second screen was also failed, the babies were referred to an audiologist at 6 to 12 weeks of age. In this study, 4% of infants with hearing loss were missed during the screening period, while 27% were missed during the period of no screening. Neonatal screening is effective in identification of congenital permanent childhood hearing impairment (PCHI) early and may be particularly useful for babies with moderate and severe PCHI for whom early management may have the most benefit.

Clinical Practice Guidelines

Centers for Disease Control and Prevention (CDC), National Center on Birth Defects and Developmental Disabilities

In 2016, the CDC's National Center on Birth Defects and Developmental Disabilities stated that hearing loss that gets worse over time is known as acquired or progressive hearing loss. Hearing loss that develops after the baby is born is called delayed-

onset hearing loss. Therefore, it is important to find out if a child may be at risk for hearing loss. As a result, the organization published the following guidelines for screening and diagnosis of hearing loss in children:

- All babies should be screened for hearing loss no later than 1 month of age. It is best if they are screened before leaving the hospital after birth.
- If a baby does not pass a hearing screening, it's very important to get a full hearing test as soon as possible, but no later than 3 months of age.
- Children who are at risk for acquired, progressive, or delayed-onset hearing loss should have at least one hearing test by 2 to 2 1/2 years of age.
- If a child does not pass a hearing screening, it's very important to get a full hearing test as soon as possible.

U.S. Preventive Services Task Force (USPSTF)

The USPSTF recommends that newborn hearing screening programs include: (USPSTF, 2014)

- A one-step or two-step validated protocol which frequently involves otoacoustic emissions (OAEs) followed by auditory brainstem response (ABR) in those who failed the first test;
- Protocols to ensure that infants with positive screening-test results receive appropriate audiologic evaluation and follow-up after discharge;
- Screening and follow-up should be in place for newborns delivered at home, birthing centers, or hospitals without hearing screening facilities; and
- All infants should have hearing screening before one month of age. Those infants who do not pass the newborn screening should undergo audiologic and medical evaluation before 3 months of age.

Joint Committee on Infant Hearing (JCIH)

The JCIH, which includes organizations such as the American Academy of Pediatrics (AAP), the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS), the American Academy of Audiology (AAA), and American Speech-Language-Hearing Association (ASHA) published a position statement on principles and guidelines for early hearing detection and intervention programs.

In 1994, the JCIH listed 10 factors that identify infants at greatest risk for hearing impairment:

- Family history of hearing loss
- Congenital infections
- Craniofacial anomalies
- Low birth weight (<1,500 g)
- Hyperbilirubinemia at serum level requiring exchange transfusion
- Ototoxic medications (aminoglycosides in multiple courses and/or in combination with loop diuretics)
- Bacterial meningitis
- Low Apgar scores (0-4 at 1 minute or 0-6 at 5 minutes)
- Mechanical ventilation for at least 5 days
- Syndromes associated with congenital hearing loss – Usher syndrome, Pndred syndrome, Jervell and Lange-Nilsen syndrome, biotiidase deficiency, Refsum sndrome, Waardeburg syndrome, CHARGE syndrome, neurofibromatosis, and osteopetriosis. (Wroblewska-Seniuk, 2017)

The JCIH endorses early detection of and intervention for infants with hearing loss. To maximize the outcome for infants who are deaf or hard of hearing, the JCIH recommended hearing of all infants should be screened at no later than 1 month of age. Those who do not pass screening should have a comprehensive audiological evaluation at no later than 3 months of age. Infants with confirmed hearing loss should receive appropriate intervention at no later than 6 months of age from health care and education professionals with expertise in hearing loss and deafness in infants and young children. Separate protocols are recommended for NICU and well-infant nurseries. NICU infants admitted for more than five days are to have auditory brainstem response (ABR) included as part of their screening so that neural hearing loss will not be missed. For infants who do not pass automated ABR testing in the NICU, the JCIH recommends screening this group with the technology capable of detecting auditory neuropathy/dyssynchrony: automated ABR measurement. (JCIH, 2007)

American Academy of Pediatrics (AAP)

In February 1999, the American Academy of Pediatrics endorsed the goal of universal detection of hearing loss in infants before 3 months of age, with appropriate intervention no later than 6 months of age. (AAP, 1999)

National Institutes of Health (NIH)

In 1993, the National Institutes of Health (NIH) held a consensus conference, sponsored by the National Institute on Deafness and Other Communication Disorders (NIDCD), on identifying hearing loss in infants and young children. In its consensus statement, the NIH recommended that all newborns be tested for hearing loss at birth or within the first three months of life. Two common screening techniques were advocated: auditory brain response (ABR) testing, which examines the brain's electrical response to sound to determine whether the ear is functioning properly, and evoked otoacoustic emissions (EOAE) testing, which monitor sounds produced by the inner ear in response to stimulation. The NIH confirmed as recently as February 2017 that ABR and EOAE are standard tests in diagnosing auditory neuropathy.

OAE Evaluation for Hearing Loss in Children

The current medical literature regarding OAE testing for the evaluation of hearing loss in children has demonstrated that it is a useful tool in screening children or at-risk populations for hearing loss, and characterizing sensitivity and functional hearing loss and differentiating sensory from neural components in people with known hearing loss.

Foust, et al. (2013) evaluated using otoacoustic emissions to screen young children for hearing loss in primary care settings. Three federally funded clinics serving low-income and uninsured people in a metropolitan area participated in the 10-month study. Subjects included 846 children (842 in the target population < 5 years of age and 4 older siblings) who were screened during routine visits to their primary care providers using a distortion product OAE instrument. A multistep screening and diagnostic protocol, incorporating middle ear evaluation and treatment, was followed when children did not pass the initial screening. Audiological evaluation was sought for children not passing a subsequent OAE screening. Of the 846 children screened, 814 (96%) ultimately passed the screening or audiological assessment and 29 (3%) exited the study. Three children (one was younger than 5 years of age and two were older than 5) were identified with permanent hearing loss. OAE screening holds the potential for being an effective method for helping to identify young children with permanent hearing loss in primary care settings.

Eiserman et al. (2008) screened underserved children 3 years or younger for hearing loss using otoacoustic emissions (OAE) technology and systematically document multi-step screening and diagnostic outcomes. A total of 4,519 children in four states were screened by trained lay screeners using portable OAE equipment set to deliver stimuli and measurement levels sensitive to mild hearing loss as low as 25 decibels (dB) hearing level. The screening and follow-up protocol specified that children not passing the multi-step OAE screening be evaluated by local physicians and hearing specialists. Of the 4,519 children screened as a part of the study, 257 (6%) ultimately required medical or audiological follow-up. One hundred and seven children were identified as having a hearing loss or disorder of the outer, middle or inner ear requiring treatment or monitoring. The investigators concluded that OAE screening, using a multi-step protocol, is a feasible and accurate practice for identifying a wide range of hearing-health conditions warranting monitoring and treatment among children 3 years or younger in early childhood care programs.

Chiong et al. (2007) evaluated evoked otoacoustic emission (OAE) and auditory brainstem response (ABR) results for hearing screening in infants. The objective of the study was to correlate hearing screening outcomes of a cohort of infants with developmental outcomes at 6 and 12 months. A total of 565 infants had both OAE testing and ABR. Overall in 1,130 ears, OAE and ABR testing showed an observed agreement of 99%, agreement due to chance of 96%, and kappa agreement of 79% in diagnosing bilateral hearing losses. OAEs had a sensitivity of 86.4% and a specificity of 99.4%.

Dille et al. (2007) compared transient evoked otoacoustic emissions (TEOAE) with distortion product otoacoustic emissions (DPOAE) to determine if they resulted in equivalent signal-to-noise ratios (SNRs) when used for hearing screening in a preschool population in a community setting. Thirty-three preschool children ages 4 months to 4 years, 4 months were tested using DPOAE and TEOAE. The frequencies 800-4000Hz were compared. The tympanometric gradient was obtained from a tympanogram done on each ear. A multivariate statistic was used to compare the emission SNR from both methods. The agreement between the pass/refer rates from the OAE screens and from the tympanometric gradient were compared. TEOAE and DPOAE SNRs were significantly different in the low frequency however, there were no significant differences found in the high frequencies. There were no significant pass/refer differences found between the methods at any frequency. When comparing the agreement between the OAE methods with the tympanometry, both methods produced nearly equivalent agreement with tympanometric gradient. However, the overall correspondence between OAE findings and tympanometry was not perfect. The investigators concluded that both methods are effective and especially equivalent in the high frequencies and can be recommended for use in a preschool population in the field. Tympanometric gradient disagreed with both OAE

screening results about 25% of the time. The study also concluded that higher refer rates can be expected when young (younger than 3 years old) preschool children are included in the screen.

Lyons et al. (2004) examined the test performance of distortion product otoacoustic emissions (DPOAEs) when used as a screening tool in the school setting. A total of 1,003 children (mean age 6.2 years) were tested with pure-tone screening, tympanometry, and DPOAE assessment. Optimal DPOAE test performance was determined in comparison with pure-tone screening results using clinical decision analysis. The results showed hit rates of 0.86, 0.89, and 0.90, and false alarm rates of 0.52, 0.19, and 0.22 for criterion signal-to-noise ratio (SNR) values of 4, 5, and 11 dB at 1.1, 1.9, and 3.8 kHz respectively. DPOAE test performance was compromised at 1.1 kHz. In view of the different test performance characteristics across the frequencies, the use of a fixed SNR as a pass criterion for all frequencies in DPOAE assessments is not recommended. When compared to pure tone plus tympanometry results, the DPOAEs showed deterioration in test performance, suggesting that the use of DPOAEs alone might miss children with subtle middle ear dysfunction. However, when the results of a test protocol, which incorporates both DPOAEs and tympanometry, were used in comparison with the gold standard of pure-tone screening plus tympanometry, test performance was enhanced. The investigators concluded that in view of its high performance, the use of a protocol that includes both DPOAEs and tympanometry holds promise as a useful tool in the hearing screening of schoolchildren, including difficult-to-test children.

Clinical Practice Guidelines

American Academy of Audiology (AAA)

The American Academy of Audiology (AAO, 2011) endorses the detection of hearing disorders in early childhood and school-aged populations using evidence-based hearing screening methods. OAEs are recommended for preschool and school age children for whom pure tone screening is not developmentally appropriate (ability levels less than 3 years).

American Academy of Pediatrics (AAP)

In a clinical report for hearing assessment in infants and children, the AAP states that ABR and OAEs are tests of auditory pathway structural integrity but are not true tests of hearing. Even if ABR or OAE test results are normal, hearing cannot be definitively considered normal until a child is mature enough for a reliable behavioral audiogram to be obtained. Behavioral pure-tone audiometry remains the standard for hearing evaluation. According to the AAP, a failed infant hearing screening or a failed screening in an older child should always be confirmed by further testing. Audiologists may repeat the audiometric tests in a sound booth and using a variety of other tests. ABR can also be used for definitive testing of the auditory system. Diagnostic ABR is often the definitive test used by audiologists in children and infants who are unable to cooperate with other methods of hearing testing. A diagnostic ABR is usually performed under sedation or general anesthesia in children aged approximately 3 to 6 months and older. Diagnostic ABR provides information that is accurate enough to allow for therapeutic intervention. According to the AAP, the OAE test also does not assess the integrity of the neural transmission of sound from the eighth nerve to the brainstem and, therefore, will miss auditory neuropathy and other neuronal abnormalities. Infants with such abnormalities will have normal OAE test results but abnormal auditory brainstem response (ABR) test results. A failed OAE test only implies that a hearing loss of more than 30 to 40 dB may exist or that the middle-ear status is abnormal (Harlor, 2009).
In a policy statement for the pediatrician's role in the diagnosis and management of autistic spectrum disorder in children, the AAP states that any child who has language delays should be referred for an audiologic and a comprehensive speech and language evaluation. If the child is uncooperative, diagnostic otoacoustic emissions or sedated brainstem auditory evoked responses should be obtained. (AAP, 2001)

According to the American Academy of Pediatrics (AAP) guideline titled "Hearing Assessment in Infants and Children: Recommendations Beyond Neonatal Screening", the technology used for hearing screening should be age appropriate. Evoked OAE testing is appropriate for children of any developmental age and automate ABR testing is appropriate for infants with a developmental age between birth to 9 months. Behavioral audiological testing for infants and children between the developmental ages of 9 months to 2½ years is generally performed using visual reinforcement audiometry and play audiometry is generally used for children with a developmental age between 2½ to 4 years. (Harlor, 2009)

American Speech-Language-Hearing Association (ASHA)

The ASHA Practice Portal lists the following recommendation for newborn infant and childhood screening:

- Newborn Infant Hearing Screening indicates OAE - either transient-evoked OAEs (TEOAEs) or distortion product OAEs (DPOAEs)—are recommended for use in newborns. Because OAEs are sensitive to outer ear debris and middle ear fluid

that may be present at birth, most OAE screening protocols involve an outpatient rescreening of those newborns who fail the screening at hospital discharge. Newborns who have initially passed a hearing screening are rescreened if readmitted to the hospital or if risk factors for hearing loss develop over the infant's hospital stay following the initial screening.

- Childhood Hearing Screening indicates the use of OAE technology may be appropriate for screening children who are difficult to test using pure-tone audiometry (those who cannot respond to traditional pure tone or conditioned play techniques; Stephenson, 2007). Multiple OAE screenings may be needed/used to limit false positive findings and medical referrals for children who fail the initial OAE screen, but who do not actually need treatment. (Eiserman et al., 2008)

OAE Testing in Individuals Who Cannot Cooperate with Other Methods of Hearing Testing

Tas et al. (2007) evaluated hearing in autistic children by using transient evoked otoacoustic emission (TEOAE) and auditory brainstem response (ABR). Tests were performed on 30 children with autism and 15 typically developing children, following otomicroscopy and tympanometry. The children with autism were sedated before the tests. Positive emissions and normal hearing level at ABR were obtained in both ears of all children in the control group and of 25 children with autism. TEOAE and ABR results varied in the remaining five children with autism. The mean III-V interpeak latencies (IPLs) in both ears of children with autism were longer than those in the control group. According to the investigators, hearing loss may be more common in children with autism than in typically developing children.

Tharpe et al. (2006) described the auditory characteristics of children with autism relative to those of typically developing children and described the test-retest reliability of behavioral auditory test measures with this population of children with autism. Audiometric data were obtained from 22 children diagnosed with autism and 22 of their typically developing peers. The audiologic test battery consisted of behavioral measures (i.e., visual reinforcement audiometry, tangible reinforcement operant conditioning audiometry, and conditioned play audiometry) and physiological measures (auditory brain stem response audiometry, distortion product otoacoustic emissions, and acoustic reflexes). The investigators concluded that children with autism demonstrated essentially equivalent results on a battery of physiological auditory tests as those obtained from typically developing children. However, on average, behavioral responses of children with autism were elevated and less reliable relative to those of typically developing children. Furthermore, approximately half of the children with autism demonstrated behavioral pure-tone averages outside of the normal hearing range (i.e., >20 dB HL) despite having normal to near-normal hearing sensitivity as determined by other audiometric measures.

During the German Special Olympics Summer Games 2006, 552 athletes with intellectual disabilities (ID) had their hearing screened according to the international protocol of Healthy Hearing, Special Olympics. This screening protocol includes otoscopy, measurement of distortion product otoacoustic emissions, and, if necessary, tympanometry and pure tone audiometry (PTA) screening at 2 and 4 kHz. Additionally, 195 athletes underwent a full diagnostic PTA. The results of the screening and diagnostic PTA were compared. Of the 524 athletes who completed the screening protocol, 76% passed and 24% failed it. Ear wax was removed in 48% of all athletes. 42% of the athletes were recommended to consult an otolaryngologist or an acoustician. Of the 99 athletes whose screening-based suspicion of a hearing loss was confirmed with diagnostic PTA, 74 had an undetected hearing loss. The correlation (Cramer's V) between screening and diagnostic PTA was .98. The sensitivity of the screening was 100% and the specificity 98%. The investigators concluded that the screening reliably detects hearing disorders among persons with ID. The prevalence of hearing impairment in this population is considerably higher than in the general population, and the proportion of undetected hearing impairments is large, even among people with only mild and moderate ID, as examined in this study. Therefore, a screening is highly recommended for persons with ID. (Hild, 2008)

In a prospective, clinical, observational study, Hamill et al. (2003) assessed hearing impairment in adults admitted to a university surgical intensive care unit in order to identify patients at risk for impaired receptive communication. Patients included in the study were 442 adult patients admitted to the surgical intensive care unit for trauma, a critical illness, or postoperative monitoring. As part of a continuing quality improvement protocol, adults admitted to the surgical intensive care unit were screened for hearing loss. Screening included otoscopy, tympanometry, and distortion product otoacoustic emissions. Almost two thirds of patients studied failed the screening protocol. The investigators concluded that screening with otoscopy, tympanometry, and DPOAE is an efficient and sensitive way to identify patients at risk for impaired auditory acuity.

American Academy of Neurology (AAN)

In a practice parameter for the evaluation of the child with global developmental delay, the AAN recommends that audiometric assessment for children with global developmental delay can include behavioral audiometry or brainstem auditory evoked response testing when feasible (Level C; class III evidence). The AAN also states that early evidence from screening studies

suggests that transient evoked otoacoustic emissions should offer an alternative when audiometry is not feasible (Level A; class I & II evidence). Level A rating requires at least one convincing class I study or at least two consistent, convincing class II studies. According to the AAN, global developmental delay is a subset of developmental disabilities defined as significant delay in two or more of the following developmental domains: gross/fine motor, speech/language, cognition, social/personal, and activities of daily living. The term global developmental delay is usually reserved for younger children (i.e., typically less than 5 years of age). (Shevell, 2003)

OAE Testing for Ototoxicity

A nonbehavioral method for monitoring ototoxicity in patients treated with cisplatin is needed because patients enduring chemotherapy may not be well or cooperative enough to undergo repeated hearing tests. Distortion-product otoacoustic emissions (DPOAEs) provide a nonbehavioral measure of auditory function that is sensitive to cisplatin exposure. Among patients receiving cisplatin for the treatment of cancer, Reavis et al. (2011) sought to (1) identify the combination of DPOAE metrics and ototoxicity risk factors that best classified ears with and without ototoxic-induced hearing changes; and (2) evaluate the test performance achieved by the composite measure as well as by DPOAEs alone. The odds of experiencing hearing changes at a given patient visit were determined using data collected prospectively from 24 veterans receiving cisplatin. The investigators concluded that DPOAEs alone and especially in combination with pre-exposure hearing and cisplatin dose provide an indication of whether or not hearing has changed as a result of cisplatin administration.

Al-Noury (2011) measured otoacoustic emissions in patients treated with a first dose of cisplatin in a prospective study of 26 patients (mean age at treatment, 11.3 years). Audiograms and transient-evoked otoacoustic emissions (TEOAEs) and distortion-product otoacoustic emissions (DPOAEs) were measured before and after the first dose of cisplatin. Baseline readings were compared with those recorded after the administration of the first dose of cisplatin. Two patients showed a loss of TEOAEs at high frequencies above 4 kHz, and this was consistent with the 25-dB hearing loss of the high frequencies detected in their audiograms; there was a significant threshold shift for DPOAEs at a frequency >3 to 4 kHz. The authors concluded that DPOAE testing appears to be a more sensitive method to detect cochlear damage than conventional pure-tone audiometry. The authors stated that the measurement of DPOAE thresholds is a useful approach to detect the early auditory changes induced by cisplatin therapy.

Yilmaz et al. (2009) investigated cisplatin ototoxicity by using the transient evoked otoacoustic emission (TEOAE) test and the pure tone audiometer. Twenty adult lung cancer patients and 20 control group patients were included in the study. The investigators compared the hearing of the patients who received 100 mg/m (2) 4-cycle cisplatin for lung cancer, with pure tone audiometer and transient evoked otoacoustic emission test in 1,000, 2,000, and 4,000 Hz. A 55% hearing decrease with pure tone audiometer was found in patients that are receiving 100 mg/m (2) 4-cycle cisplatin for lung cancer. An established emission amplitude decrease with TEOAE test was found in 85% of the patients. When the patients' pure tone audiometer in 1,000, 2,000, and 4,000 Hz and TEOAE amplitude changes were compared, there were no statistically significant results, but when the patients' TEOAE amplitude changes in 1,000, 2,000, and 4,000 Hz was compared with the control group, statistically significant results were found. The investigators concluded that the study results demonstrate that cisplatin ototoxicity could be found with TEOAE test before it is seen with pure tone audiometer.

Delehaye et al. (2008) compared the efficacy of otoacoustic emissions (distortion-product otoacoustic emissions) with that of pure-tone audiometry as method of audiological monitoring in 60 patients undergoing Deferoxamine therapy. Distortion-product otoacoustic emissions were obtained as DP-grams. Threshold changes from baseline were found to be statistically significant from 4 to 8kHz in 68.4% of the subjects. Distortion-product otoacoustic emissions demonstrated a significant threshold shift and a decreased amplitude in the frequencies >3kHz. Furthermore, DP-gram amplitude also reduced significantly at 3kHz without any similar change in pure-tone audiometry. According to the investigators, ototoxicity screening tool DP-gram was extremely sensitive and superior to pure-tone audiometry. Their use is recommended for regular monitoring of cochlear function, aiming in prevention of permanent damage.

Clinical Practice Guidelines

American Academy of Audiology (AAA)

In a position statement and clinical practice guideline on ototoxicity monitoring, the American Academy of Audiology states that over the past decade, three main approaches have emerged for monitoring the effects of ototoxic medications on hearing loss: basic audiologic assessment, high frequency audiometry (HFA; 10-18 kHz), and OAEs.

Using OAEs to monitor ototoxic medications requires a baseline evaluation so that later results have the clearest basis for interpretation. Ototoxic drugs exert their effect on outer hair cells (OHC) function (although not solely on OHCs), and OAEs are OHC dependent. With ototoxicity, OAEs have been shown to decrease simultaneously with changes in HFA thresholds and before changes appear in the conventional audiometric frequencies. Although both TEOAEs and DPOAEs can be used to monitor the effects of ototoxic medications, DPOAEs have some distinct advantages over TEOAEs. First, DPOAEs test higher frequencies than TEOAEs, making them more sensitive to the frequency area affected first. Second, DPOAEs can be recorded in the presence of more hearing loss than TEOAEs. Therefore, if a hearing loss already exists, that patient is still able to be monitored (so long as their hearing loss is not too great), which means DPOAEs can monitor more people. Third, using DPOAEs can provide some indication of degree and configuration of the hearing loss. (AAA Position Statement, 2009)

American Speech-Language-Hearing Association (ASHA)

In the Audiologic screening section of the Preferred Practice Patterns for the Profession of Audiology, ASHA indicates that otoacoustic emissions (OAE) may be used to monitor for toxicity before, during, and after administration of or exposure to agents known to be toxic (e.g., aminoglycosides, chemotherapy agents, and heavy metals). (ASHA, 2006)
Ototoxicity is considered an otologic urgency because there is less recovery of functional damage when a treatment plan is not implemented promptly. Once the ototoxic medication is administered, regular monitoring should be a proactive step. A comprehensive assessment of ototoxicity should include sensitive audiological tests such as audiometry and DPOAEs that assess ultra-high frequencies and appropriate ototoxic grading criteria with high sensitivity and specificity.

The ASHA's "Guidelines for the audiologic management of individuals receiving cochleotoxic drug therapy" states that baseline audiometric test should be done within 24 hours of administering chemotherapeutic agents and within 72 hours of administering aminoglycoside antibiotics. Audiological reassessment done within 24 hours of the baseline test can determine patient reliability for behavioral threshold testing. It was also highlighted that testing should be initiated with a comprehensive case history including possible otologic disorders, co-morbid conditions, exposure to noise, family history of ear disorders/genetic susceptibility to ototoxic drugs, and prior usage of ototoxic medication. Ototoxicity typically begins in the frequencies above 8,000 Hz and progresses to lower speech frequencies.

Therefore, ASHA and the American Academy of Audiology (AAA) recommend that baseline assessment should include behavioral measures such as pure-tone audiometry (PTA) from 250 Hz to 8,000 Hz and high-frequency audiometry (HFA) from 9,000 Hz to 20,000 Hz, plus objective measures such as distortion product of otoacoustic emissions (DPOAEs) and tympanometry, along with self-evaluating questionnaires. Each measure provides valuable information in an ototoxicity monitoring program (peripheral and/or central auditory function, apical versus basal cochlear turn, and subjective versus objective measure), where test protocol selection is driven by both clinical purpose and patient epidemiological characteristics.

OAE Testing for Early Identification of Noise-Induced Hearing Loss

Hearing loss is common in school age individuals with Williams Syndrome (WS) and extensive in adults. Prior studies with relatively small sample sizes suggest that hearing loss in WS has an early onset and may be progressive, yet the auditory phenotype and the scope of the hearing loss have not been adequately characterized. Marler et al., (2010) used standard audiometric tools: Otoscopy, tympanometry, air conduction (bone conduction when available) behavioral testing, and distortion product otoacoustic emissions (DPOAEs) to measure hearing sensitivity and outer hair cell function. Eighty-one individuals were tested with WS aged 5.33–59.50 years. Sixty-three percent of the school age and 92% of the adult participants had mild to moderately severe hearing loss. The hearing loss in at least 50% was sensorineural. DPOAE testing corroborated behavioral results. Strikingly, 12 of 14 participants with hearing within normal limits bilaterally had 4,000 Hz DPOAE input/output (DPOAE IO) functions indicative of outer hair cell damage and impaired cochlear compression. The study results indicated that hearing loss is very common in WS. Furthermore, individuals with WS who have "normal" hearing as defined by behavioral thresholds may actually have subclinical impairments or undetected cochlear pathology. According to the researchers, the findings suggest outer hair cell dysfunction in otherwise normal hearing individuals. The DPOAE IO in this same group revealed growth functions typically seen in groups with noise-induced damage. Given this pattern of findings, individuals with WS may be at increased risk of noise-induced hearing loss.

Fetoni et al. (2009) evaluated whether distortion product otoacoustic emissions (DPOAEs) can discriminate normal subjects with a risk of damage induced by sound exposure, the effectiveness of OAEs in monitoring the protective effects of Coenzyme Q10 terclatrate (QTer), and the role of blood parameters in monitoring preventive therapies. Twenty volunteers were randomized to two groups: the first (n=10) was treated with Q-Ter (200 mg orally once daily) for seven days before noise

exposure and the second group was treated with placebo using the same schedule. All participants were exposed to white noise of 90 dB HL for 15 minutes. DPOAEs and pure-tone audiometry (PTA) were measured before and 1 h, 16 h, and 7 and 21 days after exposure. Inflammatory and oxidative stress parameters were measured before and 2 and 24 h after exposure. In the placebo group, DPOAE amplitudes were reduced 1 and 16 h after exposure compared with the baseline values. In the Q-Ter group, DPOAEs did not show any significant difference between baseline and post-exposure. PTA threshold values in the Q-Ter and placebo groups did not differ before and after exposure. No significantly different levels of the inflammatory markers were observed in the Q-Ter and placebo groups at the different time points. The investigators concluded that this pilot study confirms that DPOAEs represent a sensitive test for monitoring the effects of noise in preclinical conditions and pharmacological treatment.

Korres et al. (2009) evaluated noise-induced hearing loss in a group of industrial workers, using distortion product otoacoustic emissions (DPOAEs) in conjunction with standard pure tone audiometry (PTA). A total of 105 subjects were included in the study. PTA, tympanometry, and DPOAEs were performed. Statistically significant lower DPOAE levels were found in the noise-exposed group as compared to the control group. Based on the results of the study, the investigators concluded that DPOAEs and PTA are both sensitive methods in detecting noise-induced hearing loss, with DPOAEs tending to be more sensitive at lower frequencies.

Marshall et al. (2009) measured audiometric thresholds and otoacoustic emissions (OAEs) in 285 U.S. Marine Corps recruits before and three weeks after exposure to impulse-noise sources from weapons' fire and simulated artillery, and in 32 non-noise-exposed controls. At pre-test, audiometric thresholds for all ears were <or=25 dB HL from 0.5 to 3 kHz and <or=30 dB HL at 4 kHz. Ears with low-level or absent OAEs at pre-test were more likely to be classified with significant threshold shifts (STSs) at post-test. A subgroup of 60 noise-exposed volunteers with complete data sets for both ears showed significant decreases in OAE amplitude but no change in audiometric thresholds. STSs and significant emission shifts (SESs) between 2 and 4 kHz in individual ears were identified using criteria based on the standard error of measurement from the control group. There was essentially no association between the occurrence of STS and SES. There were more SESs than STSs, and the group of SES ears had more STS ears than the group of no-SES ears. The authors concluded that the increased sensitivity of OAEs in comparison to audiometric thresholds was shown in all analyses, and low-level OAEs indicate an increased risk of future hearing loss by as much as ninefold.

OAE Testing for Sudden Hearing Loss

Mori et al. (2011) investigated whether distortion product otoacoustic emissions (DPOAEs) can be a prognostic indicator of hearing outcomes in 78 patients with idiopathic sudden sensorineural hearing loss (ISSNHL). Based on the results of the study, the authors concluded that there was significant correlation between hearing recovery and DPOAEs measured before treatment. The authors stated that DPOAEs are a potentially useful means of predicting hearing prognosis in ISSNHL.

Amiridavan et al. (2006) conducted a prospective study with performing some audiologic tests, including pure tone audiometry (PTA), auditory brainstem responses (ABR), and OAE (TEOAE) before beginning treatment of 53 patients with SSNHL. The purpose was to assess whether OAEs have prognostic value. Patients were randomly assigned to two treatment groups: oral steroids + acyclovir vs. intravenous urographin. Twenty-eight patients underwent Magnetic Resonance Imaging (MRI) of the Brain. Based on the results of the study, the authors concluded that ABR has limitations for use in SSNHL and seems not to obviate the need for brain MRI, but may help in determining the site of lesions such as ischemia or neuropathy. Overall correlation (and S/N ratio) in TEOAE is a valuable prognostic factor in SSNHL; hence TEOAE in every patient with SSNHL was recommended.

OAE Testing for Tinnitus

National Institutes of Health (NIH)

In 2020, the National Institutes of Health (NIH) published guideline NG155 covering the assessment, investigation and management of tinnitus in primary, community and secondary care. The guideline offers advice to healthcare professionals on supporting people presenting with tinnitus and on when to refer for specialist assessment and management. The guideline indicates not to offer otoacoustic emissions tests as part of an investigation of tinnitus unless the tinnitus is accompanied by other symptoms and signs such as mild hearing loss or hearing being monitored for people on ototoxic medication. The committee recognized that although otoacoustic emissions tests are not unpleasant or harmful, the result are unlikely to affect a person's management plan for the treatment of tinnitus.

Park et al. (2013) evaluated whether abnormalities in outer hair cell (OHC) function were related to tinnitus through interaural comparison of distortion product otoacoustic emissions (DPOAEs) in a cross-sectional study. The study included 27 patients with unilateral tinnitus and pure-tone average of both ears ≤25 dB hearing loss. Pure-tone thresholds observed at 500 to 16,000 Hz and DPOAE amplitudes at f2 frequencies of 1001 to 6348 Hz were compared between the tinnitus ears and non-tinnitus ears in patients with unilateral tinnitus. The pure-tone averages in the non-tinnitus ears were similar to those in the tinnitus ears. There were no differences in pure-tone averages at all frequencies tested. While the DPOAE amplitudes measured at f2 frequencies of 1001 to 3174 Hz in tinnitus ears were not different from those in the non-tinnitus ears, the tinnitus ears showed significantly reduced DPOAE amplitudes when compared with the non-tinnitus ears at frequencies of 4004 to 6348 Hz. The authors concluded that OHC dysfunction was correlated with tinnitus at high frequencies, and DPOAE amplitudes can provide additional information about cochlear dysfunction, which is complementary to pure-tone audiometry.

Zhou et al. (2011) assessed cochlear function, perceptual thresholds and distortion product otoacoustic emissions (DPOAEs) that were measured with high frequency resolution for patients with tinnitus and non-tinnitus control subjects (n = 29 and n = 18) with and without hearing loss. For 19 of 29 of subjects, perceptual thresholds were correlated with the tinnitus likeness ratings across frequencies and this correlation was significantly improved when low input-level DPOAE were included as an additional variable. According to the authors, cochlear function is strongly associated with the tinnitus percept and measures of cochlear function using DPOAEs provide additional diagnostic information over perceptual thresholds alone.

OAE Testing for Other Indications in Adults

Engdahl et al. (2013) evaluated the association between otoacoustic emissions (OAEs), pure-tone thresholds, and self-reported hearing disability in a population-based cohort study of 4,202 adults. Participants were examined with air conduction pure-tone audimetry, transient OAE (TEOAE), and distortion product OAE (DPOAE). Based on the results of the study, OAEs were shown to be a valid measure of self-reported hearing disability of the general population with the correlation being stronger in men than in women and became more manifest with age. but added no additional information to what pure-tone hearing thresholds had already captured.

Otoacoustic emissions (OAEs) testing has also been used for other indications such as evaluating pseudohypacusis (Balatsouras, 2003), facioscapulohumeral muscular dystrophy (Balatsouras, 2007), diagnosing endolymphatic hydrops (Rotter, 2008), and evaluating vestibular schwannoma (Ferri, 2009). The evidence is insufficient to determine the usefulness of OAE testing to diagnose or manage these conditions.

The clinical evidence was reviewed on February 2, 2021 with no additional information identified that would change the conclusion.

National Institutes of Health (NIH)

In 2018, the National Institutes of Health (NIH) published guideline NG98 covering the assessment and management of hearing loss for adults with hearing loss. The guideline covered aged over 18, including adults whose age of onset of hearing loss was under 18 but who present for the first time in adulthood. The guideline cites the following should be included as part of the audiological assessment for adults:

- A full history including relevant symptoms, comorbidities, cognitive ability, physical mobility and dexterity
- The person's hearing and communication needs at home, at work or in education, and in social situations
- Any psychosocial difficulties related to hearing
- The person's expectations and motivations with respect to their hearing loss and the listening and communication strategies available to them
- Any restrictions on activity, assessed using a self-report instrument such as the Glasgow Hearing Aid Benefit Profile or the Client-Orientated Scale of Improvement
- Otoscopy
- Pure tone audiometry
- Tympanometry

No mention of OAE testing was mentioned as part of the audiologic assessment for adults.

Clinical Practice Guidelines

American Speech-Language-Hearing Association (ASHA)

The ASHA Practice Portal lists the following recommendation for adults:

- Adult Hearing Screening cites a three-pronged approach for audiologic screening for hearing disorders, impairments, or disabilities including:
  - A brief case history with a visual or otoscopic inspection to identify any significant otologic history or obvious anatomic abnormalities of the ear;
  - Pure tone screening; and
  - Use of self-report questionnaires to identify perceived difficulties related to hearing.

U.S. Preventive Services Task Force (USPSTF)

The USPSTF has determined there is inadequate evidence to determine the balance of benefits and harms of screening for hearing loss in asymptomatic adults aged 50 years or older and therefore, has no recommendation.

U.S. Food and Drug Administration (FDA)

This section is to be used for informational purposes only. FDA approval alone is not a basis for coverage.

There are a number of diagnostic auditory brainstem response (ABR), automated ABR, transient evoked otoacoustic emissions (EOAE), and distortion EOAE devices currently approved for marketing by the FDA. These devices are designated by the FDA as Class II medical devices suitable for infant and adult hearing assessment.

Refer to the following website for more information: http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfPMN/pmn.cfm. [Use product codes GWJ (evoked response auditory stimulator) or EWO [(audiometer); otoacoustic emission test.] Note that not all of these clearances are for otoacoustic emission testing. (Accessed February 2, 2021)

Note that devices in product category EWO (audiometer) are 510(k) exempt devices. Although manufacturers may voluntarily submit product information via the 510(k) process, it is not a requirement. All manufacturers are, however, required to register their establishment and submit a “Device Listing” form.

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Policy History/Revision Information

Date	Summary of Changes

10/01/2021	**Applicable Codes**
	• Updated list of applicable ICD-10 diagnosis codes to reflect annual edits:
	  ○ Added F78.A1 and F78.A9
	  ○ Removed F78
	Supporting Information
	• Archived previous policy version CS323IN.02

Instructions for Use

This Medical Policy provides assistance in interpreting UnitedHealthcare standard benefit plans. When deciding coverage, the federal, state or contractual requirements for benefit plan coverage must be referenced as the terms of the federal, state or contractual requirements for benefit plan coverage may differ from the standard benefit plan. In the event of a conflict, the federal, state or contractual requirements for benefit plan coverage govern. Before using this policy, check the federal, state or contractual requirements for benefit plan coverage. UnitedHealthcare reserves the right to modify its Policies and Guidelines as necessary. This Medical Policy is provided for informational purposes. It does not constitute medical advice.

UnitedHealthcare may also use tools developed by third parties, such as the InterQual® criteria, to assist us in administering health benefits. The UnitedHealthcare Medical Policies are intended to be used in connection with the independent professional medical judgment of a qualified health care provider and do not constitute the practice of medicine or medical advice.