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UNIVERSITY OF NORTHERN COLORADO

Greeley, Colorado

The Graduate School

THE EFFECT OF ALCOHOL ON ASSESSMENT OF THE AUDITORY
AND VESTIBULAR SYSTEM

A Doctoral Scholarly Project Submitted in Partial Fulfillment
of the Requirements for the Degree of
Doctor of Audiology

Jessica Kate Hamilton

College of Natural and Health Sciences
Department of Communication Sciences and Disorders
Program of Audiology

May 2023

This Doctoral Scholarly Project by: Jessica Kate Hamilton

Entitled: *The Effect of Alcohol on Assessment of the Auditory and Vestibular System*

has been approved as meeting the requirement for the Degree of Doctor of Audiology in the College of Natural and Health Sciences in the Department of Communication Sciences and Disorders, Program of Audiology.

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ABSTRACT

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Audiologists are healthcare professionals who are highly trained to evaluate the function of the auditory and vestibular systems with the intent of diagnosing hearing and or balance disorders. The focus of this clinical doctoral scholarly project was to determine if there was a relationship between alcohol consumption and diagnostic testing performed within the scope of practice of audiology. A review of the literature revealed that there were effects of alcohol on various audiological tests including pure tone audiometry, otoacoustic emissions, acoustic reflex thresholds, auditory evoked potentials, and vestibular testing. Based on the findings, some clinical considerations were suggested, including the recommendation to reschedule diagnostic testing of patients who have reported alcohol use in the last 48 hours.

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LIST OF ABBREVIATIONS

ABR	auditory brainstem response
ABV	alcohol by volume
AEP	auditory evoked potential
ALR	auditory late response
AMR	acoustic middle ear reflex
ANSI	American National Standards Institute
AP	action potential
ART	acoustic reflex threshold
ASHA	American Speech-Language-Hearing Association
BAC	blood alcohol concentration
BPPV	benign positional paroxysmal vertigo
CANS	central auditory nervous system
CERP	cognitive event related potential
CM	cochlear microphonic
cVEMP	cervical vestibular evoked myogenic potential
dB	decibel
DPOAE	distortion product otoacoustic emission
ECochG	electrocochleography
ENG	electronystagmography

ENT	ear, nose, and throat
Hz	hertz
ICD-10	International Statistical Classification of Diseases, 10th Revision
Kohms	kilohms
LARP	left anterior right posterior
LED	light-emitting diode
MLR	middle latency response
MMN	mismatched negativity
NHANES	National Health and Nutrition Examination Survey
OAE	otoacoustic emission
OPK	optokinetic
oVEMP	ocular vestibular evoked myogenic potential
PAM	preauricular muscle
PAN II	positional alcohol nystagmus II
P-OPK	pendular optokinetic
RALP	right anterior left posterior
SNR	signal-to-noise
SP	summating potential
SPL	sound pressure level
TEOAE	transient evoked otoacoustic emission
VEMP	vestibular evoked myogenic potential
vHIT	video head impulse test
VNG	videonystagmography

VOG

video-oculography

VOR

vestibulo-ocular reflex

CHAPTER I

INTRODUCTION AND REVIEW OF THE LITERATURE

Audiology is essentially the study of hearing. Audiologists are the health care professionals who help diagnose, manage, and rehabilitate hearing and balance disorders. In order to accomplish this task, audiologists undergo extensive training to understand how a person hears, how the human balance system works, and how to properly administer various diagnostic tests. It is also the responsibility of the audiologist to interpret the results to provide appropriate care to the patient. Additionally, there are many extrinsic factors that audiologists need to be aware of that may affect the results of the diagnostic tests such as noise exposure, ototoxic medication, and alcohol use. This chapter will focus on explaining the anatomy and physiology of the auditory and vestibular system, defining the audiological tests and the equipment used to complete the testing, as well as alcohol and its effect on the human body.

Anatomy and Physiology of the Peripheral Auditory System

The Outer Ear

The primary job of the outer ear is to transmit sound to the tympanic membrane (TM). The pinna, which is made of cartilage and covered by skin, gathers the acoustic sound and guides it into the external auditory meatus, or ear canal. The shape of the pinna is also thought to contribute to the localization of sound (Musiek & Baran, 2020). While the inner two thirds of the human ear canal are boney, the outer portion of the ear canal is cartilaginous and covered with skin that contain hair follicles and several sets of glands that produce cerumen, or ear wax. These

hairs and cerumen are in the outer ear canal to serve as a protective barrier from the outside environment (Alberti, 2001). The ear canal also acts as a resonator to allow for natural amplification of sound in the canal within the speech frequencies. The skin of the ear canal becomes thinner until it reaches the TM, which divides the outer ear from the middle ear space.

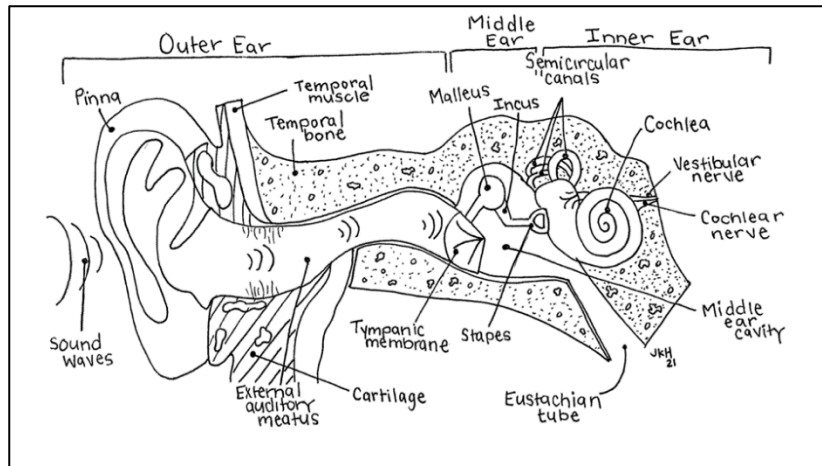
The Middle Ear

The middle ear is an air-filled space inside the temporal bone which houses the three smallest bones in the human body: the malleus, the incus, and the stapes. These three bones, or ossicles, conduct sound from the TM to the inner ear. When the sound hits the TM, it causes movement in response to the acoustic stimuli. This sends the ossicular chain into motion, where the primary purpose is to increase the energy that is passed on to the cochlea (Musiek & Baran, 2020). In order to send the sound along the auditory pathway, there needs to be an energy increase due to an impedance mismatch that exists between the air filled outer and middle ear (low impedance) and the fluid filled cochlea (high impedance). The middle ear is able to accomplish this task by the lever action of the ossicular chain and the difference in surface area between the TM and the stapes footplate (Musiek & Baran, 2020).

Other middle ear structures include the Eustachian tube, middle ear muscles, and a branch from the facial nerve (Musiek & Baran, 2020). The Eustachian tube allows for the pressure equalization of the middle ear space, as well as supplies fresh air to the cavity. This tube transforms in size, shape, and slope within the head as an individual ages (Musiek & Baran, 2020). The ossicles are supported by two muscle tendons, the tensor tympani and stapedius, that contract when exposed to loud sounds. Figure 1 illustrates some key anatomic structures of the outer, middle, and inner ear.

Figure 1

Schematic of the Human Outer, Middle, and Inner Ear



The Inner Ear

The cochlea is housed within the petrous portion of the temporal bone and inside the temporal bone is a cavity referred to as the boney labyrinth. This boney structure is filled with fluid called perilymph and coils around a central boney core of the cochlea called the modiolus. Encased in the boney labyrinth is the membranous labyrinth which contains endolymph fluid. The oval window, which is attached to the stapes footplate of the middle ear, is located in the wall of the vestibule at the base of the cochlea. This is where the inward movement of the stapes from the movement of the ossicular chain from the TM causes the movement of the fluids in the cochlea (Hayes et al., 2013).

The cochlea is divided into three fluid filled canals: the scala vestibuli, the scala tympani, and the scala media. These canals twist along the length of the cochlea and are separated from one another by two membranes. Reissner's membrane separates the scala vestibuli from the scala media and the basilar membrane separates the scala media from the scala tympani. At the apex of the cochlea there is a small opening called the helicotrema that allows the scala tympani and

scala vestibuli to communicate and share the same fluid, known as perilymph (Hayes et al., 2013). Perilymph is similar to cerebrospinal fluid due to the high concentration of sodium and low concentration of potassium. On the other hand, the scala media contains a fluid known as endolymph which is comprised of a high concentration of potassium and low concentration of sodium. The endolymph is comparable to intracellular fluid.

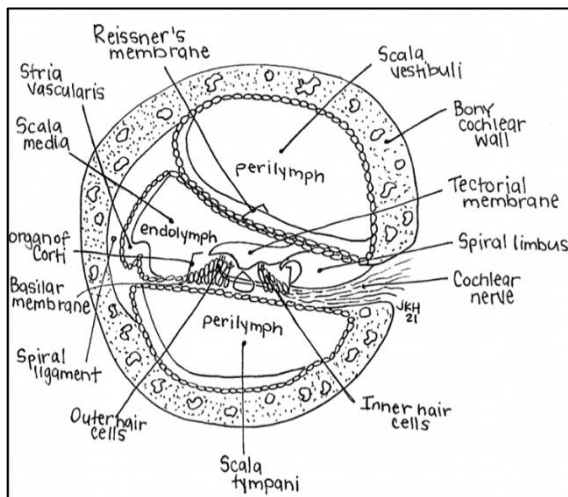
Another membrane covered opening called the round window is also located at the base of the cochlea. This is where the scala tympani terminates. The round window creates an outward movement into the middle ear space to relieve pressure in the cochlear fluid that occurs due to the inward movement of the oval window. This pressure fluctuation is generated in the perilymph by the action of the stapes footplate, creating a traveling wave on the basilar membrane that peaks more at the basal end for stimuli of higher frequencies and more apically for stimuli of lower frequencies (Pickles, 2015).

Within the scala media and resting on top of the basilar membrane is the organ of Corti. The tectorial membrane resides above the organ of Corti. The organ of Corti contains two types of sensory cells, the inner hair cells and the outer hair cells. These hair cells have a specialized structure that allows them to detect the movement of the cochlear fluids. Along the tops of each type of hair cells are stereocilia. The outer hair cells stereocilia are embedded into the tectorial membrane. See Figure 2 for a cross section of the cochlea. As the basilar membrane is set into motion, it causes the sheering of hair cells at the tectorial membrane. The inner hair cells are sheared due to the pressure of the cochlear fluid within the scala media. When stereocilia are sheared, ion gated channels open up. This allows the ions to move into and out of the cell, which in turn changes the polarity and eventually leads to the depolarization of the hair cell. This depolarization causes an action potential of the nerves that innervate that hair cell, leading to

electrical impulses from the cochlea being sent via the auditory nerve fibers. The auditory nerve fibers also code for frequency, intensity, and carry the signal up the central auditory nervous system for decoding and processing (Musiek & Baran, 2020).

Figure 2

Detailed Schematic of the Inner Ear



The Auditory Nerve

The auditory branch of the eighth cranial nerve connects the cochlea with the brainstem. Its primary function is to continue to relay the information about the frequency and timing of acoustic stimuli up towards the primary auditory centers of the brain (Musiek & Baran, 2020). The auditory nerve fibers maintain the tonotopic frequency organization present in the cochlea (Pickles, 2015). Action potentials that occur in response to acoustic stimulation continue to travel towards the cochlear nuclei in the brainstem (Musiek & Baran, 2020). The auditory nerve is the last anatomical structure in the peripheral auditory system.

Anatomy and Physiology of the Central Auditory Nervous System

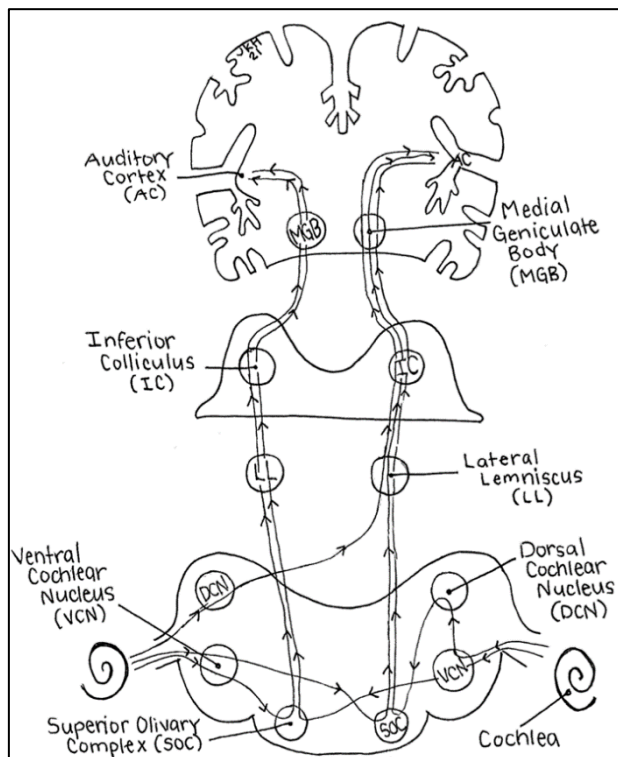
Auditory information is transmitted from the auditory nerve up to the cortex for perception through a series of nuclei. The nuclei that are represented bilaterally include the following structures: (a) cochlear nucleus, (b) superior olivary complex, (c) lateral lemniscus, (d) inferior colliculus, and (e) medial geniculate body of the thalamus (Peterson et al., 2020).

Ascending the auditory pathway, the auditory information begins with the auditory nerve that synapses with the cochlear nucleus. A large portion of this auditory signal is transmitted through crossing fibers into the superior olivary complex (Peterson et al., 2020). This information then ascends into the contralateral side of the brainstem and brain to the auditory cortex. Along the central auditory nervous system (CANS), many neurons have crossing fibers. This allows the CANS to process and collect auditory material from both the ipsilateral and contralateral sides. Figure 3 demonstrates the various pathways taken to reach the auditory cortex.

Along the CANS, different aspects of the sounds are analyzed in each of the auditory areas. For example, the superior olivary complex allows the brain to deduce the location of a signal by comparing very subtle timing and intensity differences between ears (Peterson et al., 2020). Intensity is also processed by neurons that fire at different rates based on the sound. Just like the auditory nerve, most nuclei are also tonotopically organized so that the frequency information ascending to the cortex can be maintained (Peterson et al., 2020).

Figure 3

Schematic of the Central Auditory Nervous System Pathways



The final destination for acoustic information is the primary auditory cortex, which is found on the superior surface of the temporal lobe. Most of the auditory information is transferred from the medial geniculate nucleus. Just like the cochlea, the cortex is arranged to form a tonotopical map so that different frequencies are processed in different areas. The central auditory pathway from the cochlear nuclei to the primary auditory cortex is also important to other aspects of sound processing, like environmental sound identification, and localization or where in space that sound may be coming from in the horizontal and/or vertical planes (*Know Your Brain: Auditory Cortex*, 2020). The auditory cortex is also believed to contribute to higher-level auditory processing, such as being able to distinguish specific aspects of sound that are related to speech (*Know Your Brain: Auditory Cortex*, 2020).

Assessing the Auditory System

Audiologists have many tools to help evaluate the status of an individual's auditory system. This includes otoscopy to visualize the outer portion of the ear, all the way to analyzing auditory evoked responses to help determine the integrity of the brainstem. By completing multiple tests that evaluate different portions of the auditory system, a comprehensive picture of an individual's auditory function is perceived.

Pure-Tone Threshold Audiometry

The American Speech-Language-Hearing Association (ASHA, 2005) defined pure tone threshold audiometry as "the measurement of an individual's hearing sensitivity" (para. 1). The objective was to find the softest level that an individual could hear various pure tones 50% of the time, also known as threshold. These thresholds could be obtained using manual audiometry, automatic audiometry, or computerized audiometry. Standardized practice and procedures have been developed to minimize differences between test providers and to provide consistency.

Equipment

According to the ASHA (2005), in order to obtain air and bone conduction thresholds, test providers need to use an audiometer and transducers that meet specifications of the American National Standards Institute (ANSI). A variety of transducers could be used for pure-tone audiometry including supra-aural earphones which sit on the outer ear, insert earphones that could be placed inside the ear canal, and circumaural earphones which could completely enclose the outer ear. These transducers would be paired with the audiometer and would not be interchanged unless they were recalibrated. Supra-aural and insert earphones would be appropriate when testing air conduction thresholds from 125 hertz (Hz) through 8000 Hz. Circumaural and insert earphones could be used additionally to test extended high frequencies

which range from 9,000 Hz through 20,000 Hz. A bone oscillator could be used to measure bone conduction thresholds and must also meet ANSI specifications. These transducers should be placed by the clinician on the individual and adjusted accordingly. In order to get optimal results, the pure-tone air and bone conduction testing should take place in a sound treated room.

American National Standards Institute S3.1-1999 defined the maximum permissible ambient noise levels for testing frequencies (125 Hz to 8,000 Hz) for supra-aural headphones as ranging from 21 decibel (dB) to 37 dB depending on the frequency being tested (Frank, 2000). When using insert earphones, the maximum permissible ambient noise levels established by ANSI S3.1-1999 ranged from 47 dB to 59 dB and varied based on the frequency tested (Frank, 2000). This ensured the reduction of outside environmental noises and distractions to the listener.

Anatomy and Physiology of Pure-Tone Audiometry

Pure-tone audiometry measures the function of the peripheral and central auditory systems. When performing pure-tone air conduction testing, the sound stimuli must travel from the outer ear to the middle ear, middle ear to the inner ear, and then onto the brain for auditory processing. When performing pure-tone bone conduction testing, the outer and middle ear are bypassed and only the inner ear (cochlea) and up the brain is evaluated.

By comparing the results from the bone conduction testing to those from the air conduction, a clinician could determine if there was a conductive component. If there was difference between the two tests, then the testing clinician could conclude that there was a problem with transmission of sound through the outer and middle ear. For example, if an individual had fluid in the middle ear space, then the sound stimuli presented would have trouble traveling through the fluid to continue on to the inner ear.

Otoacoustic Emissions

Otoacoustic emissions (OAEs) are low-level sounds that are produced by the outer hair cells within the cochlea in response to an auditory stimulus, or they can be spontaneously evoked. These emissions provide information regarding the function of the auditory pathway through the level of the outer hair cells. Testing OAEs is a commonly used audiological assessment tool because it is noninvasive, easy to obtain, relatively quick to run, and provides reliable information regarding an individual's cochlear status (Cunningham, 2011). Otoacoustic emissions could be used as a screening tool or as a diagnostic tool. They have often been used in newborn hearing screenings, the pediatric population to obtain frequency-specific information, difficult to test patients who cannot/will not produce reliable behavioral responses, and to monitor ototoxic medications (Cunningham, 2011).

Equipment

Equipment for testing OAEs consists of a probe that contains a microphone and speakers. The speakers in the probe generate the acoustic stimulus that are applied to the ear canal. The microphone in the probe records the OAE that is transferred back to the ear canal from the cochlea. The OAE equipment uses software for filtering, averaging techniques, and algorithms to extract the outer hair cell response. That response is then displayed on the device or computer screen. Clinicians have the ability to adjust testing protocols. For example, one clinic may recognize a present response as 6 dB over the noise floor while another may only use 5 dB. Testing should be conducted in a quiet room, away from excessive noise. It should also be imperative to have a tight seal from the probe tip in order to isolate the ear canal and the responses.

For clinical use, the most common type of OAE recorded are distortion product OAEs (DPOAEs) and transient evoked OAEs (TEOAEs). While DPOAEs are evoked by the presentation of two different frequency pure-tones at the same time, TEOAEs are evoked using a transient broadband stimulus, like a click (Meena et al., 2013).

Anatomy and Physiology of Otoacoustic Emissions

The waves generated by the basilar membrane and measured in the external auditory canal are OAEs. The stimulus is first delivered to the outer ear, travels through the middle ear, and then sent to the inner ear, which then causes movement on the basilar membrane. When the basilar membrane moves, it causes the outer hair cells to deflect, or move. When the outer hair cells deflect, their stereocilia will bend in one direction or the other, leading to a change in the hair cell membrane potential (Cunningham, 2011). When this happens, the voltage across the plasma membrane will lead the outer hair cells to change length (lengthening and shortening), which is also referred to as electromotility. The electromotility of the outer hair cells will then feedback on the basilar membrane, causing it to vibrate. This vibration is then echoed back through the middle ear to the outer ear where it is then measured by the equipment. Otoacoustic emissions have arose from the peripheral auditory system.

A present OAE reveals a few things about the auditory system. First, it shows that the conductive mechanism is functioning correctly for the tested ear. This means that there is no blockage in the external auditory canal, the tympanic membrane is moving normally, and there is a proper impedance matching system (Cunningham, 2011). Secondly, present OAEs are indicative of normal outer hair cell function. It is important to note that OAEs do not evaluate the inner hair cells, the vestibulocochlear nerve, ascending auditory pathway, or how the auditory

system processes the stimulus (Cunningham, 2011). Therefore, they are merely a test of function not a test of hearing sensitivity.

Acoustic Reflex Thresholds

Acoustic reflex threshold (ART) testing evaluates the involuntary stapedius muscle contraction that is caused by a high-level acoustic stimulation. Presenting the stimulus to one ear will elicit the response in both ears. In clinical measurements, a pure-tone stimulus of 500, 1,000, 2,000, and 4,000 Hz are often used, though one can also elicit a reflex with a broadband noise (Schairer et al., 2013). A reflex is considered to be present if the test ear decreases by a criterion amount, typically .02 mmho, while being under an activating stimulus. The lowest stimulus level that elicits the criterion change in admittance is defined as the ART (Schairer et al., 2013).

Equipment

Equipment to determine ART uses an immittance probe to measure responses in the ear. These measurements are made usually using a 226 Hz probe tone, along with a reflex-inducing stimulus that is presented to the ipsilateral or contralateral ear (Schairer et al., 2013). When testing ipsilateral responses, the probe sits in the test ear and produces an acoustic stimulus. This stimulus typically begins at an intensity of between 70- and 80-dB HL. The clinician will increase the stimulus intensity in 5 dB steps until a response is recorded. Reflex testing is typically stopped if no response is measured at a high intensity level (e.g., 110 dB HL). If measuring contralateral responses, a probe tip is placed in each ear, one tip will stimulate the ear while the opposite ear probe/insert earphone will measure the response. Some commercially available equipment allows for automated and screening ART testing (Schairer et al., 2013). Reproducibility of the reflex is confirmed when the response is present at the same intensity

twice during testing. Reflexes are indicated by the stimulus ear and should be conducted in a quiet environment.

Acoustic Reflex Threshold Pathway

The measurements of ART include both the peripheral auditory system and the CANS. When a sound enters the ear canal, it travels through the outer ear, middle and inner ear, along the vestibulocochlear nerve, and on to the brainstem. In the brainstem, the signal will first arrive at the cochlear nucleus. It then travels to superior olivary complex on both the left and right side and then down both facial nerve nuclei (Emanuel, 2009). The signal from the facial nuclei is passed along to both the left and right facial nerve, which causes the stapedius muscles in both ears to contract. This means that the stapes bones are moved away from the inner ear, in an outward and downward direction (Emanuel, 2009). This causes the middle ear system to stiffen, resulting in a minor decrease in admittance of acoustic energy through to the cochlea.

The acoustic reflex will be absent in individuals with a middle ear disorder due to the attenuation of the sounds entering the inner ear or due to the inability to measure a change in compliance due to the existing abnormality of the middle ear system. If there is a cochlear hearing loss, it is possible to elicit ART depending on the degree of loss. If the pure-tone conduction thresholds are no worse than about 50 dB HL, then ART responses could be obtained at the same level as an individual with normal hearing sensitivity (Emanuel, 2009). Normal ART responses range from 75 to 95 dB HL for 500, 1,000, and 2,000 Hz tone stimulus (Silman & Gelfand, 1981). As the pure-tone threshold increases, the likelihood of an elevated or absent response would also rise (Emanuel, 2009). Elevated ART responses range from 100 to 110 dB HL for 500, 1,000, and 2,000 Hz stimulation (Silman & Gelfand, 1981). Individuals with a retrocochlear pathology typically would have absent responses, regardless of degree of hearing

loss, depending on the site lesion. For example, if an individual has a large tumor growing on the right vestibulocochlear cranial nerve, then both their right ipsilateral and right contralateral pathway on that side would be affected and likely show an absent response. If the tumor happens to be on their left facial nerve, then the left ipsilateral pathway would be affected, as well as the right contralateral pathway.

Auditory Evoked Potentials

Auditory evoked potentials (AEPs) are electrical signals that are produced from the CANS when auditory stimuli are presented (Paulraj et al., 2015). These potentials have typically been time locked to the stimuli such that waveforms from numerous stimuli could be averaged to improve the signal-to-noise ratio of the response for clinical interpretation. The AEPs are recorded by placing electrodes on the scalp at specific locations that represent opposite ends of source generator dipoles, which is important for capturing the maximum change related to the response from the acoustic stimulation for the differential amplifier (also helping to increase visibility of the small amplitude responses). These waveforms have well-defined morphology, such that there are specific peaks that clinicians look to find and label. These peaks are evaluated in terms of their amplitude and latency. There are generally two different purposes for AEP evaluations, neurodiagnostic assessment or threshold estimation. For neurodiagnostic assessments, normative latency data for high intensity stimulation is used to evaluate physiologic responses and interpret results. For threshold estimations, the clinician is looking for the lowest stimulus intensity to produce a repeatable AEP response.

Equipment

In order to run AEPs, the participant needs to be connected to the equipment by electrodes and a transducer. Prior to attaching electrodes, the skin of the test participant must be

prepped. This means that a slightly abrasive cleaner and alcohol pad is used to remove dead skin cells from where the electrodes would be placed. The electrodes used may be disposable, such as snap, or may be non-disposable, like a cup or disc. The electrode configuration (montage) would vary based on the type of testing that was being conducted. Electrodes are assigned to be either reference/inverting (-), active/non-inverting (+), or ground and data is recorded by using a differential amplifier that amplifies the difference between the active/non-inverting electrode and the reference/inverting electrode thereby eliminating what is the same between the electrodes. This process, also known as common mode rejection, improves the signal-to-noise ratio of the response and allows for evoked potential visualization. It is important to make sure that the cables from the transducer are separated from the leads for the electrodes to minimize artifact. Transducer type could vary, from bone anchored to supraaural headphones, to insert earphones. There are clear advantages to using insert earphones over supra-aural headphones, including reduction of stimulus related artifact (Atcherson & Stoody, 2012). The patient should be aware of the instructions for the test before the transducer is placed and cell phone use should not be permitted during the procedure.

Once the electrodes are attached, impedance values need to be checked. The objective is to have low readings of < 5 kilohms (Kohms) with no more than a < 2 Kohm difference between electrodes (Hill, 2018). Hill (2018) stated that the inter-electrode impedance was most crucial due to common mode rejection not being able to function as well if the impedance levels between each electrode differed greatly.

The classification of AEPs have been primarily on the time window in which they occurred: early latency response, middle latency response, auditory late response, and cognitive event related potentials. An early latency response is evaluated based on the earlier portion of

AEPs, from about 0 to 10 milliseconds (Paulraj et al., 2015). Early latency AEPs include the electrocochleography (ECochG) response and the auditory brainstem response (ABR).

Electrocochleography (ECochG)

The ECochG shows the electrical potentials that are derived from the cochlea (Gibson, 2017). There are three basic components that comprise the ECochG: the action potential (AP), the summing potential (SP), and the cochlear microphonic (CM). The AP stems from the afferent nerve fibers in the cochlea entering the habenula perforata (Gibson, 2017). The CM comes predominantly from the movement of the outer hair cells of the organ of Corti and can sometimes be confused with stimulus artifact if the recording electrodes are outside the cochlea. The SP is a direct current potential that mirrors the time-displacement pattern of the cochlea in response to the stimulus envelope (Ferraro, 2000). The SP has also been predominately generated by the outer hair cells of the organ of Corti. For clinical purposes, the action potential is examined for its latency and magnitude and is compared to the summing potential to form the SP/AP amplitude ratio (Ferraro, 2000). Evaluation of the ratio of the SP to the AP provides information about presynaptic and postsynaptic function within the auditory system which could help detect damage before it is even evident on an audiogram (Liberman et al., 2016).

Auditory Brainstem Response (ABR)

The ABR is made up of multiple peaks that occur after being exposed to an abrupt stimulus, like a click. Morphology of wave I through wave V of the ABR are consistently used for interpretation, with wave I and wave II originating from the vestibulocochlear nerve and wave III through wave V relying on the neural functioning of the cochlear nucleus through the lateral lemniscus (Norrix et al., 2012). In clinical audiology, the ABR is used to estimate the hearing sensitivity of an individual and to assess the functional integrity of the vestibulocochlear

nerve and the neurons of the auditory brainstem. The ABR is evaluated by examining the absolute latencies, interpeak latencies, amplitude, and morphology of the waveform.

Interpretation of ABR recordings have often been biased to the clinician's subjectivity in picking when/where peaks occur.

Middle Latency Response (MLR)

According to Interacoustics, the auditory middle latency response (MLR) was “used for the assessment of the functional integrity of the auditory pathway above the level of the brainstem in cases with suspected lesions and for the assessment of nonorganic hearing loss” (*Middle Latency Response*, 2020, para 2). It has also often been used to assess neurological outcomes for individuals who experience traumatic brain injuries, to determine the integrity of the auditory nerve of an individual prior to receiving a cochlear implant, and how auditory training programs may affect an individual with an auditory processing disorder. This test allows clinicians to see a more complete puzzle of an individual's auditory system as the generation of the MLR is thought to stem from structures along the thalamocortical pathway (Musiek & Nagle, 2018). The latency of the response ranges from 15 to 80 milliseconds. Clinicians measure the response of Na (~15 to 22 msec), Pa (~24 to 34 msec), Nb (~35 to 50 msec), and Pb (~50 to 60 msec; Musiek & Nagle, 2018). Electrode placement is similar to those used for an ABR; however, because of the latency of the middle latency response, the preauricular muscle (PAM) response occurs within the time window with a similar morphology and could be misinterpreted as a response. Therefore, earlobe placement is recommended rather than mastoid placement for recording. Additionally, the clinician should avoid high intensity stimuli (> 70 dB nHL) in order to minimize eliciting a somomotor response. Individuals being tested should also be instructed to

stay awake to get more reliable testing results. This test has not been recommended for children under 10 due to auditory system maturation.

Auditory Late Response (ALR)

The Auditory Late Response (ALR) is generated by higher regions of the CANS, such as the thalamocortical pathway, the primary auditory cortex, and other cortical regions (Ventura et al., 2009). The ALR time window ranges from 50 milliseconds to 300 milliseconds. This type of testing could be done to estimate hearing sensitivity in adult populations. It has often been used in situations where an individual may be unwilling or unable to provide accurate behavioral responses when a sound is presented. The key waveforms measured and observed are P1, N1, and P2 (Ventura et al., 2009). Patient attention state does affect the ALR. If the individual being tested becomes drowsy or falls asleep, the N1 amplitude becomes smaller while the P2 amplitude becomes larger. Therefore, during this test, the patient should be advised to quietly sit while either reading a book or watching a video with closed-captioning and/or the sound muted.

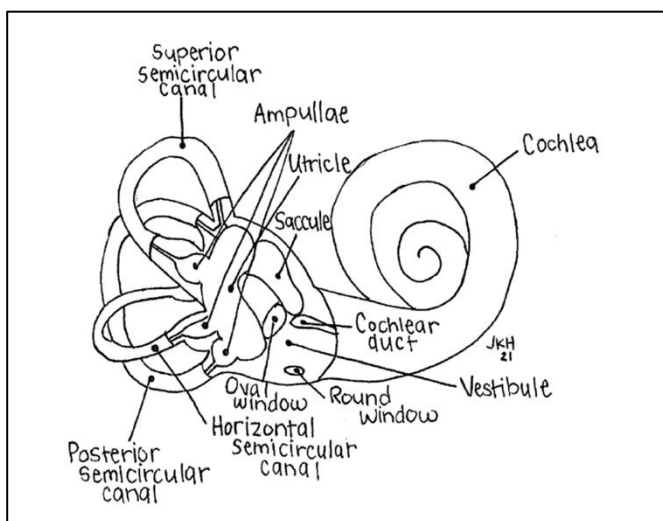
Cognitive Event Related Potentials (CERP)

The CERPs are AEPs that represent the discrimination between two different stimuli and/or identification of a change in stimulus parameters. In order to obtain CERPs, typically an oddball paradigm is used. This is done by presenting two stimuli to the patient that differ by only one characteristic, such as frequency, intensity, or duration. One of the characteristics is kept consistent by frequently occurring, while the other stimulus only occurs infrequently in a pseudorandom pattern. Mismatched negativity (MMN) is measured by taking the averaged response to the frequently occurring stimuli and subtracting it from the rarer, random stimuli (Bishop & Hardiman, 2010). Then the amplitude of this difference waveform in the time window is examined. The P300 response is observable in this infrequent averaged waveform. Both the

MMN and P300 are an automatic neuronal response that occurs with a change in events (e.g., auditory discrimination). The MMN and P300 are useful when examining auditory discrimination, how attention plays a role in auditory perception, as well as working memory (Bishop & Hardiman, 2010; Papadanill et al., 2016). The MMN occurs within 150 to 250 msec after an oddball paradigm occurs and is a negative peak (Papadanill et al., 2016). The P300 is a positive peak that occurs between 250 and 500 msec after the presentation of the stimulus (Papadanill et al., 2016).

Anatomy and Physiology of the Vestibular System

The vestibular system works in conjunction with the visual and proprioceptive systems to help maintain balance as well as visual acuity, particularly during movement. The vestibular system is comprised of five end organs within the membranous labyrinth of the inner ear on each side of the head. These organs include: the utricle, the saccule, the anterior semicircular canal, the posterior semicircular canal, and the lateral semicircular canal (see Figure 4). Vestibular organs sense linear and rotational movements and convert these forces into electrochemical signals that could be used by the central nervous system (Fife, 2010).

Figure 4*Schematic of the Vestibular System***Semicircular Canals**

There are three semicircular canals positioned in the membranous labyrinth on each side: the lateral (horizontal), the anterior (superior), and the posterior (inferior). Each of these canals has a different geographical orientation and contains endolymph. Each of the canals has an ampulla at its base. The ampulla contains the sensory epithelium called crista where the hair cells are found. These hair cells extend into a gelatinous mass, the cupula, which covers the distance across the ampulla. This forms a barrier that endolymph is unable to travel through, which results in the cupula becoming distorted when the endolymph is displaced. Therefore, when the head rotates on one of the planes of the semicircular canals, the inertia of the endolymph creates a force across the cupula which then causes a dislodgment of the hair cell bundles within the crista (Purves et al., 2001). This allows for the addition from each of the semicircular canals so that all rotating movements of the head could be perceived (Fife, 2010).

Otolith Organs

The utricle and saccule are two otolith organs in the membranous labyrinth. Both of these organs contain a sensory epithelium known as the macula. These structures also contain hair cells. The stereocilia of these hair cells expand into an elastic, gelatinous mass. On top of this gelatinous mass are otoconia that are connected by very fine collagen connective fibers (Kingma & van de Berg, 2016).

Kingma and van de Berg (2016) used the example of the physics of the otolith system to a car with an antenna. Imagine that placed on the top of the car antenna is a golf ball. When the car begins to accelerate, the golf ball will start to bend backward. Because the antenna is flexible, the antenna will revert to its original position when the car establishes a steady speed. If the car begins to decelerate, the golf ball will shift forward with the deceleration. Once the car has stopped, the antenna will again return to its initial, upright position. If the car were to spin, the antenna would bow away from the car because of the centrifugal force. This is exactly what happens with the human otolith organs. If the head were to experience a linear acceleration, the otoconia on top of the membrane would lag behind, causing a bending of the stereocilia (Kingma & van de Berg, 2016).

Vestibular Function Assessment

The vestibular system works in tandem with the visual and proprioceptive systems to provide the brain with information about head/body position, spatial orientation, and motion. It is also involved with motor function like balance, continuing posture, and stabilization. This system is crucial for normal movement and steadiness. Disruption of the vestibular system could result in loss of balance, vertigo, and nausea. To obtain information about vestibular function, clinicians could examine how an individual's eye move when given different stimuli and/or

responses to muscle contractions. The eyes are examined due to the vestibulo-ocular reflex (VOR) that helps to stabilize gaze when the head is moved. The responses to a variety of tests could distinguish impairments involving the peripheral vestibular system or the central nervous system.

Nystagmus

Nystagmus refers to a condition where there is involuntary eye movement that is quick and uncontrollable (Boyd, 2020). It could be both psychological (normal) or pathological (abnormal; McCaslin, 2020). A nystagmus could be induced in a myriad of ways. One example of a psychological nystagmus would be when it occurs during caloric irrigation. Pathological nystagmus are nystagmus that occur when there is no condition inducing a VOR response. These include nystagmus in different body positions, gaze evoked nystagmus, or spontaneous nystagmus (McCaslin, 2020). The nystagmus normally happens in both eyes and could vary between fast and slow and upward or downward beating. Quantifying the nystagmus relies on a few variables, such as latency, amplitude, velocity, and duration (McCaslin, 2020).

Equipment

Posturography

Posturography is used to help evaluate a person's reliance on different body systems to maintain balance across different testing conditions. Static posturography assesses the postural control of an individual while on a fixed surface in a comparatively calm state (Visser et al., 2008). This does not inhibit the individual from self-correction movements or the effects of gravity. Dynamic posturography involves using a moveable support that the individual is standing on, whether that movement be in one direction or multiple directions depending on the equipment. Balance could then be assessed when the platform makes a sudden jolt or movement.

It is common to use fast and short movements to evaluate the postural reaction of an individual, but sometimes slow movements are also used to evaluate how an individual anticipates and adapts to the platform (Visser et al., 2008). When undergoing posturography, individuals are harnessed into the device so that injury does not occur during testing. Individuals would also have varying conditions, such as eyes open or eyes closed and a moving visual field or a stable visual surrounding, during which exercises are stationary or occur with abrupt platform movements.

Vestibular Evoked Myogenic Potential (VEMP)

The VEMP evaluates vestibular function by stimulating one ear with loud clicks or tone bursts and then measuring the response from selected muscles. There are two types of VEMP testing: cervical (c) and ocular (o). The cVEMP could be measured when a sound stimulus is applied and causes a response with the ipsilateral sternocleidomastoid muscle that is then recorded with surface electrodes and averaged (Fife et al., 2018). This response evaluates the pathway from the saccule, to the inferior vestibular nerve, to the lateral vestibular nucleus, on to the accessory nerve nucleus, and then the sternocleidomastoid muscle (Hain, 2021c). Clinicians examine two components of the biphasic waveform, P1 (~13 msec) and N1 (~23 msec). Research has suggested that most dependable gage of the response was to examine the absolute amplitude of the wave (Hain, 2021c). It is important for the individual being tested to activate the neck muscle during testing. This could be done by having the individual lift their head up and actively turning their head to one side.

Comparably, oVEMP are recorded when a sound stimulus to the ear causes the contralateral inferior oblique muscle to respond, which are also recorded from a surface electrode and then averaged (Fife et al., 2018). To activate the inferior oblique muscle, individuals are

asked to look upward while collecting data (Hain, 2021a). Performing an oVEMP provides insight into the status of the utricle and superior vestibular nerve by stimulating the pathway. When examining oVEMP results, clinicians focus on two waveform components, the N1 (~11 msec) and P1 (~18 msec) of the waveform.

Video Head Impulse Test (vHIT)

The vHIT was developed as an objective measure of the vestibulo-ocular reflex (VOR) and how it responded to angular head accelerations (McGarvie et al., 2015). This test evaluates all six of the semicircular canals. Individuals wear specialized goggles that record eye movements on computerized equipment. During testing, the patient is seated and oriented into head rotations by the clinician: lateral, right anterior left posterior (RALP), and left anterior right posterior (LARP). During these specific rotations, each semicircular canal is evaluated based on a waveform that is produced. Individuals with a history of head and neck problems should not be evaluated using vHIT.

Electronystagmography (ENG)/ Videonystagmography (VNG)

The ENG is commonly used to record eye movements by applying the corneal-retinal potential variation principle (Ganança et al., 2010). Electrodes are placed depending on the number of available channels. Horizontal eye recordings are made by placing the electrodes next to the right or left external periobitrary corners, where vertical eye recording electrodes are above and below the eye (Ganança et al., 2010).

The VNG uses a computerized system to record eye movements with infrared sensors in masks or glasses (Ganança et al., 2010). A video camera is installed in light-proof lenses so that an examiner could directly observe and record the movement of the individual's eyes in both

darkness and with the eyes open. This is done by measuring the movement from the center of the pupils.

Gaze Testing

Gaze testing evaluates an individual's ability to focus their eyes on a motionless target (McCaslin, 2020). Using a lightbar, this target could be set at midline or a deviated position. When the target stimulus is set away from the midline, it causes the excitation of paired agonist extraocular muscles, as well as the inhibition of the paired antagonist muscles, as to not allow the eye to be pulled back to the midline which is considered its primary position (McCaslin, 2020). The neural integrator is responsible for this signal generation. Studies have shown that "the midbrain and medulla oblongata within the brainstem contain nuclei involved in the integration of information from conjugate eye movement systems for stable gaze holding" (Sanchez & Rowe, 2016, p. 111). When patients have impairments with their neural integrator, it generally causes a slow drift of eye movement from the target that is then corrected with a quick movement back to the target stimulus.

Saccade Testing

Saccadic testing evaluates what is known as "saccadic control" or the ability for quick and precise conjugate eye movements that move the eyes into different positions as the object of focus moves quickly (McCaslin, 2020). The speed of these eye movements depends on the distance that the eye has to travel in order to find the target stimulus. The movement is faster for targets that are further away and slower for targets that are closer. In order for the eye to accomplish this movement, the cortex, brainstem and neural integrator are used to calculate the amplitude of the target stimuli and where the eyes should go to fixate on the target, leaving the "pulse-step" like recording (McCaslin, 2020).

Smooth Pursuit Testing

The pursuit allows an individual to voluntarily keep their eyes on a slow-moving target (less than 70° per second) when the head is stationary (McCaslin, 2020). The way this system is able to do this is by persistently sampling where the target is and adjusting how much the target is slipping off the retina of the eye (McCaslin, 2020). While it is still not completely known what neural pathway is fully responsible for the smooth pursuit movement, the cortical, cerebellar, and brainstem centers are involved in the smooth tracking eye movements (McCaslin, 2020).

Optokinetic Testing

The optokinetic (OPK) is an involuntary function of eye movement that works in conjunction with the vestibular system to steady a moving visual environment when the head is immobile (McCaslin, 2020). This optokinetic reflex is a behavioral response that allows the retina to stabilize a moving image (Cahill & Nathans, 2008). When the reflex is generated, the individual typically has the sensation that they are moving in the opposite direction than the stimuli. This response is complimentary to the vestibular labyrinths because the semicircular canal system in the peripheral vestibular system is responsible for transducing angular head movements, where the OPK provides the ability to keep the target stimuli steady, allowing both systems to generate a slow compensatory eye movement that is proportional to the velocity of the head movement (McCaslin, 2020).

Positioning Testing

Positioning testing aims to identify benign positional paroxysmal vertigo (BPPV). This vertigo is caused by free-floating otoconia in the semi-circular canals. Ninety percent of the time the otoconia displacement occurs in the posterior canal. The Dix Hallpike maneuver is the gold standard for diagnosing BPPV in this position (Talmud et al., 2022). The maneuver is done by

having the patient sit in an upright position with their head turned as far to the right or as far to the left as they could. They are then asked to lie back rather quickly with their head hanging back and turned toward the affected ear (Talmud et al., 2022). This causes the otoconia to pass superiorly along the canal (Talmud et al., 2022). If the Hallpike is positive, then the patient's eyes would exhibit a rotary or up-beating nystagmus. Various maneuvers help to treat the three different canals. Other maneuvers include Epley, Brandt-Daroff, and Semont.

Positional Testing

This testing is done for two reasons: to evaluate an individual's complaint of position-induced dizziness or vertigo and to report how gravity and static body positions are impacting the afferent neural outputs that come from the peripheral vestibular system (McCaslin, 2020). In order to do this testing, the eye movements from the individual are recorded when the head is placed into a sequence of varying positions. These positions consist of supine, head right, body right, head left, and body left.

Caloric Testing

Caloric testing involves stimulating the inner ear by changing the temperature of the bone that surrounds the horizontal semicircular canal. This test employs the vestibulo-ocular reflex to look for a unilateral peripheral deficit (Murphy & Anikumar, 2021). This is typically done with water or with air that is below or above body temperature. When warm air or water is used, the endolymph is also warmed and causes a synthetic current that moves hair cells in the horizontal semicircular canal (Murphy & Anikumar, 2021). This creates an imbalance between the left and right vestibulo-ocular reflex and causes the individual being tested to become dizzy. The patient would have an increase in the firing rate of the hair cells in the horizontal semicircular canal and a nystagmus with a fast phase in the direction of the ear that is being stimulated (Parker, 1993).

Using a cool stimulation results in a reduction of the firing rate of neurons from the hair cells of the horizontal semicircular canal and a nystagmus with a fast phase in the opposite direction of the stimulated side for a normal patient (Parker, 1993). The nystagmus is typically measured with vision denied (eyes closed or goggles covered). At some point, vision is allowed and the patient is asked to fixate on a point projected in the goggles or by looking at the clinician's finger placed above their head. Fixation is done as close to the maximal response as possible. A normal response is deemed as nystagmus suppression with visual fixation (Parker, 1993).

Rotary Chair

Rotary chair testing is typically used in conjunction with electronystagmography or video head impulse testing (Hain, 2021b). This test includes three parts: a motorized chair that turns slowly to produce a nystagmus which evaluates dizziness, an optokinetic test where the individual looks at moving stripes to evaluate dizziness, and a fixation test where the individual focuses on a dot of light that rotates with them to measure the nystagmus (Hain, 2021b). During optokinetic testing, the individual is stationary in the chair and the stripes are rotated around the person. Rotary chair testing is considered to be the “gold standard” when it comes to diagnosing bilateral vestibular loss. The chair could change velocity, as well as move in a sinusoidal fashion. Clinicians need to calibrate chair velocity and distance to eyes daily prior to testing (Hain, 2021b).

Research has allowed audiologists to determine normative data for each of the previously mentioned assessments. However, there are extrinsic factors that could alter the outcomes of the various evaluations of the auditory and vestibular system, such as alcohol consumption.

Alcohol

When consumed, alcohol is distributed via the bloodstream throughout the body. Most tissues, such as the muscles, brain, and heart are subjected to the same concentration of alcohol as the blood (Paton, 2005). The liver is the exception, where the concentration is higher because the blood supply is obtained straight from the stomach and small bowel by way of the portal vein (Paton, 2005). A very small amount is able to penetrate fat due to its poor solubility. This means that tissue and blood concentrations are greater in women because they have more subcutaneous fat and a smaller blood volume (Paton, 2005). In addition, women may have a decreased amount of alcohol dehydrogenases in the stomach than men, so that less alcohol is processed before it is absorbed (Paton, 2005).

In the United States, it is illegal to drive with a blood alcohol concentration (BAC) at or above .08. To reach this prescribed level is the equivalent of one drink per hour. However, the rate of alcohol absorption depends on a multitude of factors: body build and size of the individual, gender, if there has been previous exposure to alcohol, the type of drink, and even phase of a female's menstrual cycle (Paton, 2005). When consumed on an empty stomach, it is absorbed much faster and would peak around one hour after consumption (Paton, 2005). This would vary based on the amount that was ingested by the individual. Conversely, consuming carbohydrates prior and during consumption of alcohol delays the absorption rate. If an alcoholic beverage is aerated with carbon dioxide, such as champagne or a mixed drink with soda, the alcohol will enter the system quicker (Paton, 2005). Alcohol is metabolized in a linear fashion, meaning that the concentration would decline at the same rate over several hours following consumption. Alcohol is thought to be removed from the blood at a rate of 3.3 millimoles/hour

(15 mg/100 ml/hour; Paton, 2005), but again, this would vary based on the amount that was ingested and the composition of the individual who consumed it.

Research has been conducted in order for health care professionals to better understand the epidemiology of alcohol and its role in health and disease. Alcohol consumption has been linked to more than 60 varying medical conditions such as cirrhosis of the liver, epilepsy, esophageal cancer, hemorrhagic stroke, self-inflicted injury, and poisonings (Room et al., 2005). With scientific attention on alcohol consumption and the human body, it was not surprising that researchers explored how alcohol specifically could affect hearing and balance by examining various parts of the diagnostic test battery.

CHAPTER II

APPLICATION TO THE FIELD OF AUDIOLOGY

The field of clinical audiology has developed an extensive diagnostic test battery to help determine the cause of problems that could occur in the auditory and vestibular systems. These tests were summarized in Chapter I. Clinicians are well versed in these diagnostic tests and the expected test results associated with different pathologies. Because alcohol has been a substance that has been commonly used by many individuals, it would not be uncommon for a clinician to routinely see a patient that struggles with alcohol abuse or consumes alcohol socially. When alcohol has been involved, many clinicians may not be completely aware of the effects of alcohol on various diagnostic tests. This chapter will explore evidence within the literature regarding the effect of alcohol on various diagnostic tests within the scope of practice of audiology.

Alcohol and Hearing Thresholds

Upile et al. (2007) investigated the effects of alcohol on auditory thresholds in 26 healthy volunteers. Inclusion criteria included no knowledge of an abnormality in the vestibulocochlear system, over 18 years of age, and able to reach a minimum breath alcohol threshold level of 30 units per liter (which has been the legal driving limit in the United Kingdom; Upile et al., 2007). Pure tone air conduction thresholds were measured at 250, 500, 1,000, 2,000, 4,000, and 8,000 Hz before and after alcohol consumption. In addition, a timed psychometric and visuo-spatial skill test was administered prior to the hearing evaluation to determine the effect of alcohol on their psychomotor ability and decision making. Any volunteer with a change in their hearing threshold was invited back for further audiometric testing.

Upile et al. (2007) observed increases in hearing thresholds related to alcohol, as every participant demonstrated some increase in hearing thresholds post-alcohol consumption. However, some frequencies were affected more than others. Ninety percent of the participants had a mean change of 7 dB with three or more frequencies being affected (Upile et al., 2007). Additionally, there were greater changes in threshold noted in lower frequencies (250-1000 Hz) for females.

Verma et al. (2006) examined the audiovestibular functions in participants with a long-term dependence on alcohol and compared them to abstainers and milder alcoholics. Twenty participants, ranging from 30 to 60 years of age, who fulfilled the International Statistical Classification of Diseases, 10th Revision (ICD-10) for long-term alcohol dependence of more than 2 years were used as the subject group. These participants were under-going a de-addiction program and selected at random to be included. Two control groups were age matched to the subject group, the first being social drinkers that did not fulfill ICD-10 criteria for alcohol dependence, and the second being lifetime abstainers. No participants had any outside confounding factors such as syphilis, ototoxic medications, noise exposure, or head injury. Any long-term dependent participant refrained from alcohol consumption for a minimum of 1 week prior to undergoing audiovestibular testing which included pure-tone audiometry (Verma et al., 2006).

When examining pure-tone audiometry, Verma et al. (2006) discovered that the alcohol dependent group had hearing thresholds that were 5 to 10 dB higher at all frequencies compared to the social drinking group, and thresholds that were 15 to 20 dB higher compared to the abstinent group (Verma et al., 2006). The authors also noted statistically significant threshold elevations ($p < .001$) at 4,000 Hz and 8,000 Hz in the long-term dependent group. Fifty percent

of the participants exhibited elevated pure-tone audiometry results, with 25% of the individuals exhibiting flat sensorineural hearing loss and 25% having a high-frequency hearing loss (Verma et al., 2006).

Pearson and Timney (1999) measured the effect of alcohol on monaural and binaural auditory sensitivity in comparison to a placebo condition. This research consisted of six participants, 3 males and 3 females, ranging from 21 years of age to 29 years of age. Each participant was in good health, had no family history of alcoholism, and had no prior drinking problems. Pearson and Timney (1999) measured hearing thresholds at 100, 200, 400, 800, 1,600, and 3,200 Hz. Each participant responded to the randomized stimuli by clicking a mouse over a virtual button on a display monitor. When the computer received a “yes” result, the intensity was decreased by 1 dB sound pressure level (SPL) and when the answer was “no,” the intensity was increased by 1 dB SPL. When participants were in the alcohol condition, their BAC was raised to 80 milligrams per deciliter, or .08. This was monitored every 15 minutes using a breath measuring device, which also occurred during the placebo condition. Two trials were done under each testing condition, one for monaural and one for binaural. The alcohol and placebo testing conditions occurred on different days for each participant and were randomly assigned. Forty-eight total threshold estimates were obtained for each participant.

Pearson and Timney (1999) found that the effect of alcohol decreased as the frequency of the stimuli increased and that the effects of alcohol were higher under monaural listening conditions than binaural. Under monaural listening conditions, detection levels were elevated in the alcohol condition for 200 Hz and 400 Hz. Under the binaural listening condition, 200 Hz was the only frequency significantly affected by alcohol consumption (Pearson & Timney, 1999). Pearson and Timney (1999) stated that binaural listening had an advantage over monaural

listening due to binaural summation, which was the neural combination of the signal from each ear at the superior olivary complex.

In contrast, Murata et al. (2001) explored how different amounts of alcohol consumption may affect thresholds over time. Fifteen participants (eight females and seven males) who were considered social or occasional drinkers were used for this experiment. Every participant had thresholds of 15 dB HL or less at octave frequencies ranging from 250 to 8,000 Hz. Alcohol was given in the form of beer at a 5% alcohol by volume (ABV). The control used was an alcohol-free beer. These were distributed in doses of 125, 250, 500, and 1,500 ml. All participants were asked to consume the drink within 10 minutes, except for 1,500 ml which was required to be consumed within 30 minutes. Pulsed pure-tone stimuli were presented monaurally in 5 dB steps to obtain auditory thresholds. Thresholds were measured 5, 30, 60, 120, 240, and 480 minutes after ingesting either the beer or the alcohol-free beer. Blood alcohol concentrations were measured by breath samples taken at the time of threshold measurements.

Murata et al. (2001) observed that ingestion of 500 ml of beer significantly reduced thresholds across all frequencies tested and 250 ml significantly reduced thresholds at all frequencies except for 8,000 Hz. These results were in contrast with aforementioned studies. However, after ingestion of 1,500 ml of beer, thresholds increased at both 250 and 500 Hz but was only considered significant at 250 Hz. When participants consumed 125 ml of beer, there was no significant effect on auditory threshold. Murata et al. (2001) revealed that the effect on threshold, either reduction or elevation, may have depended on the amount of alcohol that was ingested. No sex difference was observed in this experiment.

Lin et al. (2017) looked at the interaction of moderate alcohol consumption, gender, and hearing thresholds using the dataset from the National Health and Nutrition Examination Survey

(NHANES). This study included NHANES data from 1999-2004 and contained 4,075 participants. When comparing the moderate alcohol consumption group thresholds to lifetime abstainers, a significant negative association in high-frequency and low-frequency thresholds was found, along with female moderate alcohol consumption drinkers tending to have an overall reduced hearing threshold (Lin et al., 2017). This supported the idea that moderate alcohol consumption may result in a decrease in threshold in both high and low frequencies, compared to people who never drank, particularly in females.

Lin et al. (2017) suggested that consuming a moderate amount of alcohol may have a protective effect on hearing threshold in women through a few mechanisms. First, Lin et al. (2017) suggested that alcohol may increase plasma high-density lipoprotein cholesterol concentrations which could contribute to optimal cochlear circulation and result in a decrease of hearing impairment. Second, Lin et al. (2017) also suggested that cardiovascular disease was correlated with hearing loss and, since research has shown that consuming alcohol may decrease cardiovascular disease risk, it may also lead to a decreased risk of a hearing loss (Lin et al., 2017). Lastly, Lin et al. (2017) stated that alcohol had an anti-inflammatory property, which could help the cellular survival inside the cochlea. Because women have been more susceptible to the effects of alcohol, innate biological differences, such as height and weight, were likely the reason that a significant difference was seen in women and not noted in men (Lin et al., 2017). A summary of all the findings related to the effects of alcohol on hearing thresholds can be found in Table 1.

Table 1*Research Results Regarding the Effect of Alcohol on Pure Tone Hearing Thresholds*

Study	Findings
Lin et al. (2017)	<ul style="list-style-type: none"> • Thresholds were reduced in the low and high frequencies when compared to those that were lifetime abstainers. • Reduced thresholds more prominent in females
Murata et al. (2001)	<ul style="list-style-type: none"> • Reduction in thresholds for small amounts of alcohol • Increased thresholds for larger amounts of alcohol • Most changes were in lower frequencies (250 and 500 Hz) • No gender differences
Pearson & Timney (1999)	<ul style="list-style-type: none"> • Increased thresholds for low frequencies (most predominant at 200 Hz) • Monaural thresholds affected more than binaural thresholds
Upile et al. (2007)	<ul style="list-style-type: none"> • Increased thresholds for all frequencies tested (250, 500, 1000, 2000, 4000, and 8000 Hz) after alcohol consumption • Increase in low frequencies thresholds more pronounced in females
Verma et al. (2006)	<ul style="list-style-type: none"> • Thresholds increased from abstainer to social drinkers to alcoholics • Statistically significant threshold elevations were found at 4000 and 8000 Hz

Alcohol and Otoacoustic Emissions (OAEs)

Torre and Reed (2019) examined if alcohol was associated with changes in DPOAEs in young adults with normal hearing sensitivity. Torre and Reed (2019) recruited 161 females and 55 males and information about alcohol consumption was collected using self-reported measures. Each participant was asked about their alcohol consumption within the last 30 days, how many of those days they drank, and to state the number of drinks they typically consumed in a given sitting. A drink was defined as a shot of liquor, a 12-ounce beer, a mixed drink that contained liquor, or 5 ounces of wine. Participants were divided based on reported drinking habits into either light drinking ($n = 78$), heavy drinking ($n = 77$), or no drinking ($n = 61$) groups--with

equal representation of gender in all three groups. The average number of drinks reported was 14, which served as the defining factor of which drinking group participants were placed into.

All participants underwent bilateral pure-tone air conduction testing with normal hearing was reported as 20 dB HL or less for all frequencies tested (250, 500, 1,000, 2,000, 3,000, 4,000, 6,000, and 8,000 Hz). Each participant was confirmed to have normal ear function bilaterally. A test ear for DPOAEs was randomly selected for each participant. The DPOAEs were obtained for the 1,000-6,000 Hz frequency range using a frequency ratio of 1.22. Lower-level stimuli were used, 55- and 40-dB SPL, due to being sensitive to a smaller noise-induced change in otoacoustic emissions (Torre & Reed, 2019). A +6 dB signal-to-noise ratio (SNR) was used to measure positive responses over the background noise.

For men who reported drinking, Torre and Reed (2019) found a trend that indicated poorer DPOAEs at 1,500, 2,000, and 3,000 Hz when compared to men who did not report drinking in the past 30 days; however, this trend was not statistically significant. For females, the DPOAEs were similar between the group that did consume alcohol during the 30 days compared to those that did not drink during that time.

Hwang et al. (2003) investigated the effect alcohol consumption may have on the function of outer hair cells using DPOAEs. Eight participants volunteered for this study (five females and three males) and underwent pure-tone audiometry and DPOAE testing. After receiving initial results, each participant was then required to consume different amounts of whiskey based on their weight (3ml/kg) over a 30-minute period while eating breakfast. All participants reached a level of intoxication, distinguished by slurred speech, slight ataxia, and dizziness. Pure-tone thresholds and DPOAEs were rerecorded at 30 minutes, 1 hour, 2 hours, and 3 hours after alcohol ingestion. The DPOAEs were recorded with a frequency ratio of 1.22. The

f_2 tested frequency range was 750-7187 Hz and stimuli levels of f_1 and f_2 were both set to 70 dB SPL. Hwang et al. (2003) discovered that at frequencies greater than 5,500 Hz, a significant decrease in DPOAE amplitude occurred when measuring 30 minutes and 1 hour after alcohol consumption. This amplitude did return to baseline when measured again at the 2 hour and 3-hour mark post alcohol consumption. At test frequencies below 4,375 Hz, there were no noted amplitude changes in any of the subjects during any data collection period. Hwang et al. (2003) reported no significant difference in the noise level during the entirety of the DPOAE testing.

It was unclear what the underlying mechanism was for the decreased high frequency DPOAE amplitudes. The authors suggested several possibilities including impairment of the outer hair cell function in the cochlea due to ototoxicity of the alcohol, suppression of excitatory transmissions within the inner ear due to alcohol, or possibly an impaired middle ear transfer function due to alcohol's effect on the middle ear muscles (Hwang et al., 2003).

Alcohol and Acoustic Reflex Threshold (ARTs)

Uhles et al. (2000) examined the effect of alcohol on the ART of the chinchilla. Six chinchillas were randomly selected, four males and two females, and put into recording pairs. These pairs were measured during four intervals that alternated every other hour during the day. Each recording pair was tested on successive days for each condition with only one pair being observed each day. Each recording pair received a 2-week rest period between conditions to make sure that there was no contamination between experimental testing conditions. For the baseline, each chinchilla was given 7.5 ml/kg of water through a syringe. After an hour, the chinchilla was placed into restraints for a blood sample to be collected and to test the acoustic middle ear reflex (AMR). The blood sample was taken from a vein in each subject's pinna. The AMR was recorded every other hour for four total data collections, starting 1 hour after

ingestion. Each session had two descending trials and two ascending trials, which were flip-flopped between each of the data collections. The condition for alcohol ingestion was recorded in the same matter as the baseline, but each chinchilla was given 7.5 ml/kg of Jägermeister instead of water.

To measure the AMR for each chinchilla, an 800 Hz pure-tone stimulus was used. This tone was presented to the chinchilla's right ear and impedance was recorded contralaterally. For the descending trial, the pure-tone was presented at 110 dB SPL and was brought down in 2.5 dB increments until the impedance plateau disappeared. For the ascending trial, the pure-tone stimulus was presented at 60 dB SPL and increased by 2.5 dB increments until the researchers observed a noticeable change in the impedance plateau. An impedance plateau for ascending trials was defined as the "qualitative increase in the output of the impedance of the middle ear, which remained stable as the stimulus level continued to increase" (Uhles et al., 2000, p. 524). The descending trials impedance plateau was defined as when the impedance levels would no longer decrease as the stimulus level continued to go down (Uhles et al., 2000). The probes were inserted directly in the external auditory meatus and held in place by Velcro on the pinna. If the researchers believed there to be artifacts in any trial, the trial was ended and remeasured.

After analyzing data, Uhles et al. (2000) found that the blood alcohol volume ranged from .087% to .121%, with the average being .099% after 1 hour of ingestion. All chinchilla's displayed intoxicated behaviors including being unable to right themselves, swaying, and being lethargic. Although this study was based on an animal model, these numbers were a very close representation to the legal driving limit in humans being .08% and common signs of intoxication. Uhles et al. (2000) observed statistically significant differences in the chinchilla pairs from baseline to 1 hour after ingestion when comparing the control condition to the alcohol condition.

After 3 hours, the differences between the two conditions were not shown to have statistical significance; however, they were close to meeting significance.

One possible explanation given for the change between the control and alcohol condition was that ethanol affected the stapedius muscle and reduced the muscles ability to attenuate the amplitude of the noise passing through the middle ear space (Uhles et al., 2000). Uhles et al. (2000) also suggested that ethanol may also indirectly affect the stapedius muscle through the neural pathway that innervates it, particularly the neurotransmitters glutamate and aspartate on the afferent neural pathway. This may be due to inhibiting the activity of these neurotransmitters, which then inhibited the transmission of the auditory signal which was needed to elicit the reflex.

Cohill and Greenberg (1979) studied the effects of ethyl alcohol ingestion on ipsilateral and/or contralateral acoustic reflex threshold. There were 16 participants total used in this study, 8 females and 8 males, who were considered to have normal hearing. Normal hearing was defined as pure-tone air conduction thresholds that were not worse than 15 dB HL from 500 Hz to 4,000 Hz, as well as normal middle ear pressure and compliance. Both impedance and audiological measurements were recorded prior to the consumption of alcohol. Presentation to both the ipsilateral and contralateral response were given in 1 dB steps. The participants were then given a 50% solution of 100 proof vodka and were required to drink the entirety in under 15 minutes. Blood-alcohol measurements using an electronic breath alcohol tester and all participants reached a maximum BAC of .10%. Cohill and Greenberg (1979) suggested this level would induce slurred speech, slight vertigo, and loss of fine motor control. Baseline ARTs were measured prior to alcohol ingestion and then again when a participants' blood alcohol concentration changed from .03 to .10% in .01 increments. The lowest level to produce a .04 cc

change in compliance were defined as ARTs. The ART threshold obtained post-alcohol was subtracted from the pre-alcohol ART to determine if there was a shift in the threshold.

Cohill and Greenberg (1979) reported that the largest shift in ART occurred when participants reached a BAC of .10%. The maximum shifts were around 7 dB for ipsilateral ART presentation and around 11 dB for contralateral ART presentation. Cohill and Greenberg (1979) noted a significant difference between the contralateral and ipsilateral response when examining the level of BAC. The ipsilateral response did not show a significant threshold shift at any frequency measured until the participants reached a BAC of greater than .04, whereas the contralateral response began a significant threshold shift at the start of the BAC measurement. Change in frequency had no significant effect on threshold shifts for either pathway measured (Cohill & Greenberg, 1979).

Bauch and Robinette (1978) examined the acoustic reflex to see if differing stimuli and sex of participants had any effect on threshold. All 18 participants (9 male and 9 female) had normal hearing thresholds, normal tympanograms, and normal ART (not exceeding 100 dB HL at 500, 1,000, or 2,000 Hz) for both ears. Four varying stimuli were used: narrow band noise, white noise, a recording of rock music, and a recording of factory noise. A mixer of ethanol and orange juice were consumed where the amount of ethanol was determined by the body weight of the participant. This mixture was distributed into three glasses. Data were collected at 4 BACs: 0.0%, .10%, .15%, and descending back to .10%. Each participant was tested under all conditions in one session that lasted approximately 8 hours. The stimuli were randomized between participants.

Post alcohol consumption, all stimuli had a significant threshold elevation ($p = 0.05$). The greatest threshold shift of 7 dB occurred at 0.15% BAC when using a stimulus of white noise,

where the smallest shift of 2.2 to 2.6 dB occurred using a narrow band stimulus at all post-alcohol measured levels (Bauch & Robinette, 1978). Bauch and Robinette (1978) did note that several participants had ARTs that had little to no change during each testing condition. Only 8 of the 18 participants had drastic changes in ART post-consumption of alcohol. Results indicated no significant difference between sex for all stimuli at each BAC level tested (Bauch & Robinette, 1978).

Alcohol and Auditory Evoked Potentials (AEPs)

Verma et al. (2006) evaluated how long-term alcohol dependence affected the ABR by comparing responses across three groups: those who abstained from alcohol consumption, individuals who socially drank, and individuals who were considered to be alcohol dependent. Twenty participants who were defined as alcohol dependent underwent testing. Each of the 20 participants were then age-matched with controls that were considered social drinkers, as well as those that had abstained from alcohol consumption throughout life. The ABR was recorded using 22,000 clicks at 85 dB nHL and 100 dB nHL while masking noise was presented in the contralateral ear. The stimulus was delivered at a rate of 11 clicks per second. A bandwidth filter of 150 Hz to 3,000 Hz was also used and a minimum of 2 trials were run for each ear to show reproducibility. Responses were considered to be prolonged if the latencies or interpeak latencies of waves I through V were ± 2 standard deviations of the mean latency for the control group (Verma et al., 2006).

When comparing the latency data across groups, the alcohol dependent group had a mean prolonged absolute latency of wave III and V, as well as a prolonged mean I-V interpeak latency; however, this was not considered statistically significant (Verma et al., 2006). In the study group, Verma et al. (2006) also observed that the absolute latency of wave I was prolonged in two

participants, along with wave III in two participants, and wave V in four participants. This meant that 8 of the 20 participants, or 40%, had abnormal ABR recordings. Additionally, I-III interpeak latencies were prolonged in four participants, III-V interpeak latencies were prolonged in three participants, and I-V interpeak latencies were prolonged in three participants.

Begleiter et al. (1981) examined ABR results from chronic alcoholics (those who had been heavily consuming alcohol for a minimum of 6 years to an average of 16 years) who had been abstinent from drugs and alcohol for 2 to 3 weeks. These results were compared to a control group who was known to occasionally consume an alcoholic beverage socially. Seventeen males were recruited for each group. The control group was then age and education matched to the test group. Using earphones, the click evoked ABR was measured using a stimulus rate of 10 clicks per second, an intensity level of 70 dB nHL and 2,000 stimuli repetitions. The response was a bandpass filtered from 100 Hz to 2,000 Hz. The researchers measured the ABR response by the absolute latency of waves I-V, as well as the interpeak latencies from waves I to each successive peak.

The research by Begleiter et al. (1981) revealed a statistically significant difference between the absolute latencies for waves II, III, IV, and V in the alcoholic group compared to the control group. Wave I did not show a significant difference in absolute latency. The interpeak latencies (I-II, I-III, I-IV, and I-V) also showed a statistical significance from the chronic alcoholics to the control group. While wave I generated from the auditory nerve was not affected, Begleiter et al. (1981) results showed a significant delay in each of the peaks that followed. Begleiter et al. (1981) suggested this may be due to a demyelination of the neuronal pathway caused by excessive alcohol consumption.

Squires et al. (1978) also examined the effect of alcohol on ABR recordings. There were six participants with normal hearing thresholds and no prior history of alcohol abuse, however, each self-reported social drinking. Alcohol doses were distributed based on the amount that was normally consumed by each participant in social settings, making the range of dose from .55ml/kg to 1.65ml/kg (Squires et al., 1978). Each participant was required to drink vodka mixed with orange juice within a 30-minute time window. Auditory stimuli were presented monaurally with .5 msec clicks at a rate of 10 per second through supraaural headphones at 55 dB SL and 75 dB SL relative to each participant's hearing threshold. Waveforms were collected prior to the consumption of alcohol and within 2 hours after consumption. After alcohol consumption, participants exhibited a prolongation in waves II to wave VII, but it was still considered to be within the range of normal variation. These results were similar for both stimulus intensities; however, Squires et al. (1978) noted that only wave III and wave V at 55 dB could be reliably distinguished. These results were similar to those reported by Begleiter et al. (1981). The researchers also noticed that amplitude of the waveform was not affected for any participant in the alcohol condition. Squires et al. (1978) suggested that a higher dose of alcohol may be required to significantly impact ABR wave latencies.

He et al. (2013) explored how alcohol affected the MMN by looking at intensity, frequency, location, and duration of multiple oddball paradigms to see if the pre-attentive processing was different between different sound features. Twelve participants with normal hearing were used in the study and considered to be healthy, normal social drinkers. All of the participants completed two experimental sessions, one placebo and one alcohol condition, that were separated by 2 weeks. The alcohol condition was comprised of a dose of .65 g/kg of white wine which required each participant to consume the beverage within 10 minutes. The breath

alcohol concentration was then tested every 5 minutes following alcohol consumption until the level was considered steady. Participants in the placebo condition were given a dose of .02 g/kg of white wine that was mixed with distilled water. The breath alcohol concentration was measured in the same fashion as the alcohol condition and testing began once a steady level was achieved. Stimuli were presented binaurally through headphones at an intensity of 70 dB. The standard tone was made up of three sinusoidal partials (523 Hz, 1,046 Hz and 1,569 Hz) with the last 2 frequencies being 3 and 6 dB lower in intensity and presented at 75 milliseconds. The deviant frequency changed from the standard tone by $3/8$, $10/8$, and $21/8$ semitone. The intensity changed by 5 dB step decreases (65, 60, and 55 dB) and the location was changed to be perceived at 10° , 40° , or 90° to the left or right of the participants. The durations were shorter than the standards by 16 ms (59, 43, and 27 ms). There was a total of 156 presentations for each of the 12 deviants listed (He et al., 2013). The waveform was a bandpass filtered from 0 to 40 Hz at a 500 Hz sampling rate. Once testing was finished, the breath alcohol concentration was tested for each condition.

He et al. (2013) revealed that there was no significant difference for frequency, intensity, or location when looking at the placebo and alcohol conditions regardless of varying the deviations between the variables in the MMN. The peak latency of the MMN was delayed in the alcohol condition and the researchers did notice that the amplitude of the MMN was larger in the placebo condition than the alcohol condition.

Kähkönen et al. (2005) explored if alcohol affected the neural correlates of involuntary attention. Eleven participants were used in this study who were all considered to be moderate, social drinkers. This was defined as up to 10 drinks per week. Prior to the experimental procedure, the participants were required to abstain from drinking for 48 hours and to not

consume food 3 hours beforehand. On the test day, each participant was given 30 minutes to consume .8 g/kg of ethanol mixed with orange juice or just orange juice to serve as the placebo in this double-blind design. Testing began 1 hour after the ingestion of the placebo or alcohol and BAC was measured prior to the consumption of the liquid and again 30 minutes following. Each participant was tested around 1 week prior to obtaining a control session. During the testing, each participant was asked to watch a silent video and told to not pay any attention to the tones that were presented monaurally to the right and left ear in a randomized order. The tones consisted of a 700 Hz standard tone, and 2 deviant tones of 665 Hz and 560 Hz. These tones were presented at 60 dB above the individuals' threshold which were determined prior to the testing session. An electrode cap was used to record all AEPs with the nose electrode used as reference. A 750 msec time window was used for the recordings, along with a sampling rate of 397 Hz. The following AEPs were evaluated: N1 of the auditory late response, MMN, and P300. Band pass filtering was done 1-30 Hz for N1 and at 2-20 Hz for the MMN and P300. Responses were considered to be present if the amplitude was two standard deviations above the pre-stimulus baseline.

Kähkönen et al. (2005) found that alcohol had a main effect on the N1 and P300 amplitude and a near-significant effect on the MMN amplitudes. More specifically, Kähkönen et al. (2005) found that the N1 amplitudes were considerably smaller in the alcohol condition at the central electrodes and frontocentral electrodes when the right ear was stimulated. The P300 amplitude was also smaller after the participants consumed alcohol after left ear stimulation at the place of the frontocentral electrodes. The alcohol showed to have no effect on the N1, MMN, or P300 latencies. These results suggested that alcohol may negatively impair the brain's ability to discriminate frequency changes at different phases in the auditory processing pathway.

A summary of all the findings related to alcohol's effect on objective measures of auditory function including OAEs, ARTs, and AEPs can be found in Table 2.

Table 2

Research Findings on Effect of Alcohol on Objective Measures of Auditory Function

Study	Findings
Otoacoustic Emissions (OAEs)	
Hwang et al. (2003)	<ul style="list-style-type: none"> • Decrease in DPOAEs amplitude > 5,500 Hz
Torre & Reed (2019)	<ul style="list-style-type: none"> • Decrease in DPOAEs amplitudes at 1,500, 2,000, and 3,000 Hz
Acoustic Reflex Threshold (ART)	
Bauch & Robinette (1978)	<ul style="list-style-type: none"> • Increase in threshold at all stimuli post alcohol consumption • No sex difference
Cohill & Greenberg (1979)	<ul style="list-style-type: none"> • Shift in both ipsilateral (~7 dB) and contralateral (~11 dB) ART
Uhles et al. (2000)	<ul style="list-style-type: none"> • Statistically significant change in the AMR in chinchillas
Auditory Evoked Potentials (AEPs)	
Begleiter et al. (1981)	<ul style="list-style-type: none"> • Increased waves II and V absolute latency • Increased interpeak latencies in all waves for the alcohol group
He et al. (2013)	<ul style="list-style-type: none"> • Reduced amplitude and delayed latency of MMN
Kähkönen et al. (2005)	<ul style="list-style-type: none"> • Reduced amplitude for N1 and P300 • Reduced amplitude of the MMN
Squires et al. (1978)	<ul style="list-style-type: none"> • Longer latencies for wave II through wave V
Verma et al. (2006)	<ul style="list-style-type: none"> • Longer latencies for waves III and V • Prolonged interpeak I-V latency

Alcohol and Vestibular Function

Romano et al. (2017) focused their research on how a controlled amount of alcohol could influence gaze by looking at eye-drift velocity. Fourteen healthy adults with no reported history

of vertigo or gait imbalance participated. At the time of the experiment, none of the study participants were using any drugs that may have affected gaze-holding. For the experiment, the participants were standing in an upright position on a turntable that was connected to a three servo-controlled motor-driven axes (Romano et al., 2017). Molded thermoplastic masks were used to stabilize and limit head movement during the testing. Romano et al. (2017) also used safety belts to reduce any artifacts that may be caused by trunk movement. At 1.5 meters, a light-emitting diode (LED) visual stimulus was presented on a mounted lightbar at 0 degrees azimuth, at the patient's eye-level. This was attached to a hemispherical full-field screen that was on a platform that could be rotated vertically. Video-oculography (VOG) was used to record horizontal eye movements. Both eyes were recorded simultaneously; however, one eye received an optic filter so that tracking of eye movement was still recorded, but vision was denied. Romano et al. (2017) believed this would help eliminate double vision. The LED reached an eccentricity of 40 toward the side of the eye that had vision and 20 toward the eye that was covered (Romano et al., 2017). The covered eye was randomized between participants, as well as the direction of the LED. Each participant underwent 2 testing conditions, a baseline prior to alcohol consumption and 30 minutes after they ingested the designated amount of alcohol. The amount of alcohol, in the form of red wine, given to each participant was determined based on sex, height, and weight.

Romano et al. (2017) measured a faster centripetal eye drift after ingestion of alcohol, which increased with increasing eccentricity when being compared to baseline measures. When comparing eye movements between the baseline and alcohol condition, Romano et al. (2017) noted that alcohol reduced the gaze angle where the nystagmus was clearly recognizable, which they believed was due to a higher eye drift velocity when examining the same gaze eccentricity.

When comparing drift velocity from both eyes of each participant, Romano et al. (2017) found that it was nearly identical and that symmetry was not affected by alcohol consumption. These findings implied that, under the influence of alcohol, the neural commands generating eye movements that were processed by the brainstem neural network lost efficiency.

As discussed previously, Verma et al. (2006) compared long-term alcohol dependence to participants that abstain and participants that socially drink. Verma et al. (2006) performed cold-caloric testing by irrigating each participant's ear with 250 milliliters of saline for 40 seconds at 30° C and 44° C. Verma et al. (2006) noted that cold caloric testing revealed an abnormal response in 6 of the 20 participants in the long-term dependence group. More specifically, 3 of the 20 participants showed a unilateral weakness, 2 with a right-sided weakness and 1 with a left-sided weakness. In the long-term alcohol group, the latent period was prolonged, total number of beats was reduced, the total duration of the nystagmus was reduced, and there was a slowing trend seen for the maximum speed of the slow component; however, these differences were not significant. None of the responses in the two control groups showed any abnormalities (Verma et al., 2006).

Chiang and Young (2007) looked at alcohol and its effect on vestibular function when the participant was close to the legal limit. Twenty healthy, males with no history of ear disorders participated. Each participant underwent VEMP testing and caloric testing with visual suppression prior to consuming alcohol to establish a baseline. To achieve a breath alcohol concentration close to the limit (.25 milligrams per liter), the participants were given plum wine. Once the limit was met, testing in the alcohol condition was performed. When testing VEMP, the participant was in the supine position with active electrodes on both sides of the upper half of the sternocleidomastoid muscle and reference electrode at the suprasternal notch. When recording,

participants were instructed to keep their head elevated. Stimuli were 500 Hz tone bursts at an intensity of 95 dB HL were delivered through insert earphones and at a stimulation rate of 5 Hz per second. A total of 200 responses were averaged. Consecutive runs were made to ensure reproducibility. For caloric testing, the participant was placed in a supine position at 30°. The researchers used cold water at 20° C to irrigate each ear canal for 20 seconds. Visual suppression was used during the testing when the nystagmus reached a continuous response by turning on a light for 10 seconds and having the participant focus on the index finger of the researcher. After the 10 seconds, the light was switched off with the participants eyes open but covered until the nystagmus dissipated (Chiang & Young., 2007).

Chiang and Young (2007) found that all participants (40 ears) had VEMP responses prior to the ingestion of alcohol. After consuming the plum wine, only 36 ears had VEMP responses. In addition, the latency of the peak P1 significantly increased under the alcohol condition. The amplitude of P1 to N1 between both conditions did not reveal a statistically significant difference. For the caloric testing, there was a significant difference noticed in the alcohol condition for slow phase velocity of the nystagmus, which was reduced when compared to the baseline. There was also a decrease in the visual suppression in the alcohol condition that was statistically significant. Chiang and Young (2007) hypothesized that alcohol caused cerebellar atrophy resulting in a lack of visual suppression.

Tianwu et al. (1995) investigated effects on vestibular function from acute alcohol intoxication by examining the vestibulo-ocular reflex (VOR), calorics, EquiTest and pendular optokinetic nystagmus test (P-OPK). Ten male participants were used and received an examination from an ear, nose, and throat doctor (ENT), as well as a brief physical prior to the experiment to rule out abnormality and determine overall health. For the P-OPK test, the

participants were placed on a rotating chair in an optokinetic drum. The drum was used to provide the pendular sinusoidal stimulation. Optokinetic stimuli at an amplitude of 120° were rotated. After undergoing P-OPK testing, the participants underwent VOR testing under the same conditions as the P-OPK testing, just rotated sinusoidally. For caloric testing, cold air was used at 24°C to each ear for 60 seconds. The EquiTest consisted of a support surface and visual surround that was controlled by a computer. Six sensory conditions were used. The first through third condition had a fixed support surface where the participant stood with their eyes open (condition 1) and then closed (condition 2), where in condition 3 the visual surround moved in relation to the participants anterior-posterior sway. In the fourth through sixth condition, the support surface moved in relation to the participants anterior-posterior sway. The visual conditions were the same as they were for the first through the third. Conditions 3 to 6 were repeated 3 times in 20 second trials to establish a stable value. Each participant received 15 milliliters of whiskey and was asked to consume its entirety within 5 minutes. Tianwu et al. (1995) took blood samples before the ingestion of alcohol, as well as at 30, 90, and 150 minutes after alcohol consumption.

Tianwu et al. (1995) discovered that there was a significant difference in P-OPK, VOR, and caloric testing when comparing results to the participants baseline. The gain for P-OPK testing before drinking was .85 ($SD = 0.08$) and after drinking was .70 ($SD = 0.11$). For VOR, the gain prior to consuming alcohol was 0.91 ($SD = 0.09$) and afterwards was decreased to 0.67 ($SD = 0.11$). Before drinking for the caloric testing, the maximum velocity for the right ear was 38.1° per second ($SD = 11.4$), where after alcohol consumption was reduced to 23.1° per second ($SD = 6.1$). Tianwu et al. (1995) also reported that the left ear maximum slow-phase velocity before consumption was 31.3° per second ($SD = 12.4$) and then again declined to 21.2° per

second ($SD = 5.1$) after consumption. For the EquiTest, significant decreases were noticed in condition 4 and 5 when comparing baseline to the alcohol condition which indicated postural instability (Tianwu et al., 1995). A summary of all the effects of alcohol on vestibular function/testing is presented in Table 3.

Table 3

Research Results Regarding the Effect of Alcohol on Vestibular Function

Study	Findings
Chiang & Young (2007)	<ul style="list-style-type: none"> • VEMP responses were absent in 10% of ears after alcohol consumption • The latency of the VEMP increased significantly • Reduced slow phase velocity during caloric testing • Decrease in visual suppression
Romano et al. (2017)	<ul style="list-style-type: none"> • Faster centripetal eye drift • Reduced gaze angle
Tianwu et al. (1995)	<ul style="list-style-type: none"> • When compared to baseline, a significant reduction in eye speed velocity and gain was reported in P-OPK (gain), VOR (gain) and caloric testing (velocity) • Postural instability was noted in two conditions when comparing consuming alcohol to baseline
Verma et al. (2006)	<ul style="list-style-type: none"> • ~ 30% abnormal responses during cold caloric testing • Prolonged latent period, reduced number of beats, reduced number of nystagmus, and slowing

Summary of Research Results

A review of the literature helped to evaluate alcohol and its overall effect on specific parts of the auditory and vestibular system. Each research study had varying inclusion criteria, number of participants, and methods. Some areas of audiological assessment had more research conducted than others. While it was clear from the research that there were effects of alcohol on

auditory and vestibular testing, much of the research provided inconsistencies, and so there was no true consensus in regard to the results that could/should be expected for different auditory and/or vestibular tests.

CHAPTER III
CRITICAL APPRAISAL OF THE RESEARCH AND
FUTURE DIRECTIONS

Assessment and Gaps of Existing Literature

Research on hearing thresholds and alcohol all revealed a relationship between alcohol consumption and pure-tone thresholds, regardless of the sample size or experimental procedure. Lin et al. (2017) and Murata et al. (2001) found that alcohol could reduce auditory thresholds, while Pearson and Timney (1999), Verma et al. (2006), and Upile et al. (2007) noted that auditory thresholds were increased when a person was under the influence of alcohol. It is still unclear in the research found whether the majority of the changes occur in the low frequencies, high frequencies, or both.

The research reviewed regarding alcohol's effects on OAEs had differing results. Hwang et al. (2003) reported a change in DPOAE amplitude in the higher frequencies (> 5,500 Hz) where Torre and Reed (2019) only noted a trend in men at 1,500, 2,000, and 3,000 Hz. This could be due to Hwang et al. (2003) research requiring the consumption of alcohol during data collection and Torre and Reed (2019) only looking at self-reported measures. Torre and Reed also had a rather large sample size when compared to the eight volunteers that were used in the Hwang et al. (2019) experiment.

Reviewing the research on ART, the results from all studies (Bauch & Robinette, 1978; Cohill & Greenberg, 1979; Uhles et al., 2000) suggested that alcohol ingestion could affect the ART in both the human and animal model. Statistically significant differences in ARTs were

observed when alcohol was present in the bloodstream compared to the baseline measures. It should be noted that research in this area was fairly limited and older.

Multiple studies examined revealed that alcohol did have an effect on AEPs. Verma et al. (2006) and Begleiter et al. (1981) revealed prolonged absolute latencies in recorded ABRs. He et al. (2013) and Kähkönen et al. (2005) showed that the amplitude of the MMN was decreased when tested under an alcohol condition.

There was a consistent finding across all of the research reviewed that alcohol had a significant effect on vestibular function. This effect could be measured across multiple clinical tests that were typically used in vestibular evaluations. Chiang and Young (2007), Tianwu et al. (1995), and Verma et al. (2006) all showed that alcohol consumption reduced caloric results for both the left and right ear. Romano et al. (2017) and Tianwu et al. (1995) results revealed that gaze and optokinetic tests had a decrease, implicated the neural responses of the brainstem. Chiang and Young (2007) also noted that VEMP responses may become absent after alcohol ingestion.

Research was limited on alcohol and its effect on ART. Out of the studies found, one used animal subjects in the research. Although chinchillas have a similar sensitivity to intensities and frequency range (Trevino & Lobarinas, 2019), there were still differences between the two. For example, the average length of a human cochlea was 31.5 mm with two and a half cochlear turns, where a chinchilla averaged 18 mm and had three cochlear turns (Trevino & Lobarinas, 2019). Additionally, chinchillas used for research were commonly acquired from ranches that farm them for the use of their fur. Typically, the ones taken to use in studies were the ones whose fur had not met the criteria for fur quality (Trevino & Lobarinas, 2019), which could potentially indicate health issues with the animal.

Likewise, finding research on OAEs and how alcohol could affect results was also limited. The research that was found only looked at DPOAEs without delving into transient evoked otoacoustic emissions (TEOAEs). Transient evoked OAEs were better at determining cochlear function in the mid-frequencies where DPOAEs were better when evaluating 4000 Hz and higher (Tzanakakis et al., 2016). Though DPOAEs were generated from more specific places of the cochlea, perhaps looking at TEOAEs, which got contributions from a larger region of the cochlea (Tzanakakis et al., 2016), would provide more insight into how alcohol alters the cochlea.

Research Challenges

One of the biggest challenges observed in analyzing previous research was whether or not participants of the study were self-reporting alcohol use or if they were given alcohol to consume during the research study. An advantage of having the research participants consume alcohol during the testing was being able to control for the type of alcohol ingested, as well as the amount. Dosage of alcohol could also have been adjusted based on gender and body size to make sure that each participant ingested the same amount to be metabolized by their body. This protocol would allow researchers real time results to determine how long the alcohol effected the testing results.

For those that chose to do self-reports, the questionnaires asked how many drinks were consumed within a period of time. The definitions of drinks may have been different for each participant. For example, two people could have consumed a 12 oz. beer each evening, one of those may have been a light beer with a low ABV of 4% and the other could have been a Belgian style beer with a high abv of 12%. While both of the participants did only consume one beer an evening, they were drastically different in strength and effect on the body. There was never a

clear definition of what constituted a drink in the questionnaire nor was there a universal standard between research studies.

Clinical Recommendations

Integration of determining alcohol consumption was conducted through the patient case history. This was accomplished by asking questions such as:

1. Do you consume alcoholic beverages?

If the patient answered yes:

- a. What type of alcoholic beverage do you consume?
 - b. How many alcoholic beverages do you consume in a given week?
 - c. Have you consumed alcohol in the last 48 hours?
2. Are you taking any medications to combat long term alcohol use?
 - a. If answered yes, please list medication:

No significant amount of time was added to filling out a case history with these additional questions. Asking these questions also allowed case histories to define the word beverage. This meant 6 oz. of wine, 12 oz. of beer, or 1.5 oz of liquor were equal to one drink.

Clinics could make sure that case histories were filled out prior to the appointment to have ample opportunity to read all the information provided by the patient. One way to accomplish this was to send case history forms through email or mail with the request to receive the responses back the day before the scheduled appointment. If sending forms through the mail, return address envelopes should be included for the patient to send back to the practice. Emails were sent to an encrypted site to protect the patient's information. Establishing a website portal for patients to upload information to the practice was also one way to receive the case history in advance. If patients cannot review and fill out the case history before the appointment, clinics

could schedule extra time to discuss all aspects of the case history with the individual before beginning the appointment testing.

If a patient had reported alcohol use on their case history, the clinician would then look at the following:

1. Was alcohol consumed within a 48-hour time window?

If the patient answers yes:

- a. What type of alcohol was consumed?
 - b. How much was consumed?
2. What is the reason for the patient visit?

Any patient that answered “yes” to consuming alcohol within a 48-hour time window and reported enough consumption to elevate their BAC to the legal limit should be rescheduled, especially if the patient was undergoing diagnostic testing. Based on previously collected data, clinicians could not get accurate testing results and, therefore, may not be providing appropriate care for the patient. This should be left to the discretion of the clinician.

Clinical Examples

Example One

A patient comes in for a comprehensive hearing test after having consumed alcohol in the past 24 hours. Because of alcohol consumption, their pure-tone thresholds are increased. This particular patient is a good candidate to be fit with amplification based on their hearing loss and speech discrimination. The amplification devices are ordered and the patient is scheduled to return in a few weeks to get fit with hearing aids. When the patient returns, they have not had any alcohol since the last time they came in. The hearing aids are programmed to the thresholds that were recorded a few weeks prior. The patient then reports everything is too loud. After

turning down the gain to adjust for the volume issue, the clinician runs verification and reveals that the current settings in the hearing aids are not appropriate for the patient's hearing loss. The patient is then taken back into the sound booth to verify thresholds. This time when being tested, the threshold response to the frequencies has decreased.

Example Two

A patient was scheduled to have a VNG due to experiencing episodic vertigo. On their case history, the report having consumed alcohol in the past 12 hours due to anxiety of the upcoming test. Once beginning the evaluation, direction-changing nystagmus was recorded in positional tests. When the patient turned their head to the right in supine position, the nystagmus recorded was left beating. When the patient rotated their head to the left in supine position, the nystagmus switched to right beating. The clinician then did not know if the nystagmus was true responses to the VNG test or if the patient was experiencing positional alcohol nystagmus II (PAN II) which occurred several hours after alcohol consumption. The clinician included suspected PAN II in the testing impressions and the patient was scheduled for an additional VNG to verify results.

In clinical example one, the pure-tone threshold testing needed to be completed again because the first results obtained were invalid. In clinical example two, the patient needed to undergo another VNG test to determine if alcohol was contributing to the recorded nystagmus. In both of these examples, the clinician and the patient's time were wasted. The patient had to take time out of their schedule in order to make the appointment, only for the testing to have to be completed again. Having to retest patients took away appointment times for the clinician to see other people, as well as time in the appointment to get everything accomplished for that patient. Repeat testing may also accrue cost to the patient. Insurance may or may not cover the cost of

additional testing. If the patient did not have insurance and was self-pay, the patient may not have the finances to cover repeat diagnostic tests.

Expectations must be established from the beginning when scheduling the appointment in order to avoid clinical scenarios like the ones mentioned above. Office staff should verbally include the importance of refraining from alcohol consumption 48 hours prior to undergoing audiological evaluation. This should be reiterated when confirming the appointment with the patient. Another way to avoid rescheduling patients due to alcohol consumption would be to include written material. This could be done simultaneously when collecting the case history by including an attachment in emails, a pamphlet sent in the mail, or a link in a patient portal.

Future Directions

In order to progress the field of audiology on the effects of alcohol on assessment of the auditory and vestibular system, ongoing research studies need to be continued. For hearing thresholds, further research could be conducted to determine if alcohol affected the entirety of the basilar membrane, or if it occurred only at the apical or basal end. Researchers should also further explore the protective properties of moderate alcohol consumption. If there were protective factors, researchers could further investigate to determine if those protective factors only occurred when consuming a specific type of alcohol or if they occurred regardless of what type of alcohol was ingested.

Another area of research could be strictly focusing on TEOAEs and alcohol use. These results could then be compared to those who researched DPOAEs. This may allow researchers to determine if alcohol had a blanket influence on the cochlea, or if alcohol also targeted specific regions of the cochlea.

Furthermore, future researchers could delve into gender differences and how alcohol effected different aspects of the test battery. Since males and females absorb alcohol at different rates, it would be interesting to compare the differences among the different tests. One fascinating research avenue would be to examine transgender individuals who have been taking hormone replacements and how alcohol did/could affect their auditory and vestibular system.

Summary

From analyzing previous research, different audiological tests have shown that alcohol consumption affected results. It should be audiologists' responsibility to keep conducting research. More complete and accurate documentation of the various tests would only increase the audiological field and their knowledge on how hearing could be affected by alcohol. Once researchers have a better understanding of the relationship between alcohol and the auditory and vestibular systems, clinical audiologists could then take the necessary steps to improve patient care and establish specific clinical protocol to obtain the most reliable testing results.

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