Recording and Analysis of Transient Otoacoustic Emissions During Outer Ear Canal Pressure Compensation

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RECORDING AND ANALYSIS OF TRANSIENT OTOACOUSTIC EMISSIONS DURING OUTER EAR CANAL PRESSURE COMPENSATION

By

Moises Perez

A DISSERTATION

Submitted to the Faculty of the University of Miami in partial fulfillment of the requirements for the degree of Doctor of Philosophy

Coral Gables, Florida

August 2012
A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy

RECORDING AND ANALYSIS OF TRANSIENT OTOACOUSTIC EMISSIONS DURING OUTER EAR CANAL PRESSURE COMPENSATION

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Otoacoustic Emissions (OAEs) are sounds generated by an active process in the auditory system’s cochlea. It has been widely accepted that the generation of OAEs is a precursor for healthy hearing. The measurement of evoked OAEs can be used to determine the general health of the cochlea and basilar membrane's response and sound transmission forward and backwards through the inner ear. OAEs are commonly used for newborn infant hearing screening where many middle ear pathologies are first detected. In most cases, secondary screening tests such as tympanometry are not conducted unless the patient has failed the OAE screening first. Increases in ear canal pressure have an almost identical effect on OAE recordings when compared to naturally occurring negative middle ear pressures (NMEPs) (Sun & Shaver, 2009). Thus arises the need for pressure compensated OAE screening. This study aims at reviewing the design of a self-compensating pressure system capable of generating steady meatal pressures during OAE subject screening. Facets of system design including patient safety, software interaction, and initial test results will be presented. We will also present the results of an IRB sanctioned volunteer study which collected the TEOAE and meatal responses of 20 individual ears during multiple pressure criteria. Testing and analysis of signals in both the time and frequency domains will be reviewed.
DEDICATION

This work is first and foremost dedicated to the love of my life, my beautiful wife of seven years Nicole Mixson Perez. You alone know the true level of commitment, the pain and personal sacrifice, the countless 4AM nights followed by 10 hour work days. In all of this you were my God given strength, my light at the end of the tunnel. I love you and thank you (and the Pooks) for everything you’ve done during this incredible process.

I would also like to dedicate this work to my family. My mother and father (Maby & Jesus Perez) who have encouraged and prayed for me since I started this journey in 2004. To my encouraging Abuelo (Felipe Alvarez) and my sweet departed Mima (Maria Alvarez). I promised I would see this through. Last but not least my great and supportive parents in law (Denise Mixson & Bill Mixson) who are never short on providing prayer, an ear (to test), a meal or just well wishes. Thank you all from the bottom of my heart.

With great affection for every single one of you,

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LIST OF ABBREVIATIONS

$\eta_s$  Screw Efficiency
$\mu s$  Microsecond
$A$  Cross Sectional Area
AAA  American Academy of Audiology
ASHA  American Speech Language Hearing Association
BOM  Bill of Materials
$c$  Speed of Sound
CAD  Computer Aided Design
$cc$  Cubic Centimeter
COCH  Cochlea
D1  Length of Malleus Lever (Rigid Level Model)
D2  Length of Stapes (Rigid Level Model)
dAPA  Dekapascal
dB  Decibel
DC  Direct Current
DP  Distortion Product
DPOAE  Distortion Product OAE
EAC  External Auditory Canal
$F$  Force
$f$  Frequency
$F_1$  Force of Tympanic Membrane Vibration (Rigid Level Model)
$F_2$  Force of Stapes Motion (Rigid Level Model)
FDA  Food and Drug Administration
FFT  Fast Fourier Transform
FMN  Facial Motor Nucleus
HL  Hearing Level
Hz  Hertz
$I$  Motor, Loaded Current
$I_0$  Motor, No-Load Current
kHz  Kilohertz
$k_m$  Motor Torque Constant
$l$  Screw Lead
$L$  Syringe Length
$M$  Torque
ME  Middle Ear
MEATAL  External auditory meatus (EAC equivalent)
MEP  Middle Ear Pressure
mm  Millimeter
ms  Millisecond
$mV$  Millivolt
$n$  Rotational Speed
$nm$  Nanometer
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>NMEP</td>
<td>Negative Middle Ear Pressure</td>
</tr>
<tr>
<td>OAE</td>
<td>Otoacoustic Emission</td>
</tr>
<tr>
<td>P</td>
<td>Pressure</td>
</tr>
<tr>
<td>P</td>
<td>Static Pressure</td>
</tr>
<tr>
<td>PMEP</td>
<td>Positive Middle Ear Pressure</td>
</tr>
<tr>
<td>PWM</td>
<td>Pulse Width Modulating</td>
</tr>
<tr>
<td>r</td>
<td>Syringe Barrel Radius</td>
</tr>
<tr>
<td>RPM</td>
<td>Revolutions Per Minute</td>
</tr>
<tr>
<td>s</td>
<td>Linear Speed</td>
</tr>
<tr>
<td>S</td>
<td>Stapedius Muscle</td>
</tr>
<tr>
<td>SOAE</td>
<td>Spontaneous OAE</td>
</tr>
<tr>
<td>SOC</td>
<td>Superior Olivary Complex</td>
</tr>
<tr>
<td>SPDT</td>
<td>Single Pole, Double Throw</td>
</tr>
<tr>
<td>SPL</td>
<td>Sound Pressure Level</td>
</tr>
<tr>
<td>TEOAE</td>
<td>Transient Evoked OAE</td>
</tr>
<tr>
<td>TTL</td>
<td>Transistor-transistor Logic</td>
</tr>
<tr>
<td>TPP</td>
<td>Tympanometric Peak Pressure</td>
</tr>
<tr>
<td>TW</td>
<td>Tympanometric Width</td>
</tr>
<tr>
<td>U</td>
<td>Volume Velocity</td>
</tr>
<tr>
<td>V</td>
<td>Volume</td>
</tr>
<tr>
<td>VCN</td>
<td>Ventral Cochlear Nucleus</td>
</tr>
<tr>
<td>Vea</td>
<td>Equivalent Ear Canal Volume</td>
</tr>
<tr>
<td>VII</td>
<td>Seventh Cranial Nerve</td>
</tr>
<tr>
<td>VIII</td>
<td>Eighth Cranial Nerve</td>
</tr>
<tr>
<td>W</td>
<td>Watt (Measure of Power)</td>
</tr>
<tr>
<td>Ytm</td>
<td>Tympanometric Amplitude</td>
</tr>
<tr>
<td>Z₀</td>
<td>Characteristic Impedance</td>
</tr>
<tr>
<td>Zₜₘₑ</td>
<td>Tympanic Membrane Impedance</td>
</tr>
<tr>
<td>ρ_air</td>
<td>Density of Air</td>
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INTRODUCTION AND RESEARCH GOALS

It has been widely accepted that the generation of otoacoustic emissions (OAEs) is a precursor for normal healthy hearing. The generation of such emissions tells a great deal about the general health of the cochlea and basilar membrane's response to sound transmission within the inner ear. The OAE is also very telling of hearing health due to the physiological journey it must undergo before ever being recorded. Evoked OAEs validate the function of sound transmission from the ambient through the middle ear cavity (forward transmission) as well as from the inner ear back out to the auditory canal and to the measurement probe (backwards transmission). Thus, sound is transmitted in two directions (and twice passing the middle ear cavity) validating the function of the entire hearing mechanism, unlike most other clinical procedures which only require the forward transmission of sound. This is the reason that most middle ear pathologies are first flagged during OAE screening, as this study will discuss in greater detail within Chapter 3. For now it is sufficient to note that the health and status of the middle ear is crucial when recording OAE patterns in patients. It is also crucial to note that because OAE screening is typically one of the first forms of quick diagnostics, the audiologist or
clinician when recording OAEs is more than likely assuming normal middle ear condition, that is unless the failure of an evoked OAE test gives that particular clinician pause to consider additional testing, such as tympanometry or acoustic reflex to further diagnose potential pathologies. The old assumption regarding the correlation between a tympanometric peak pressure (TPP) at or around 0 daPa and normal healthy hearing has been removed from audiological testing criteria. While the 1979 ASHA guideline (ASHA, 1979) for acoustic-immittance screening recommended referrals for TPPs in excess of -200 daPa, the updated guideline in 1990 has advised that TPP shifts from 0 daPa are not stand-alone criteria for the detection of abnormal hearing health. In fact middle-ear pressure, which affects the TPP parameter during a tympanometric screening session, has been found to fluctuate greatly in normal healthy ears (Margolis & Heller, 1987). The normal range for TPP was expanded to -150 to +100 daPa (ASHA, 32, Suppl. 2, 1990). Thus, if no tympanometric measurements have been taken prior to OAE-based hearing screening (which is typically the case), then patients who have these naturally occurring moderate to large TPP shifts may experience a false referral result during OAE screening. In addition to this, secondary environmental considerations such as increases in ambient pressure have an almost identical effect on OAE recordings when compared to naturally occurring negative middle ear pressures (NMEPs) or significant NMEPs which occur from middle ear pathologies such as Eustachian tube dysfunction (Sun & Shaver, 2009). Thus, there is a larger probability that more false positives would be found in neonatal wards throughout Denver, CO (high altitude) instead of Miami, FL (low altitude). Thus arises the need for pressure compensated OAE screening.
The primary goal of this research is to validate the hypothesis that pressure compensation within the auditory canal, and more importantly on the wall of the tympanic membrane, can lead to better, more reliable otoacoustic (OAE) emission screenings. The effects of positive and negative middle ear pressure gradients on evoked OAE recordings have been documented in the past (e.g. Sun & Shaver, 2009; Trine et al., 1993, Zhang & Abbas, 1997), but as of the date of this research, pressure compensated OAE screening is still not an audiological standard among hearing screening practices. What is already known is that shifts in middle ear and auditory canal pressure cause parallel shifts in the physical compliance and ultimately the acoustic admittance of the tympanic membrane (the gateway to the amplified sound experience). These shifts in acoustic admittance are more commonly identified during tympanometric screening (which will be discussed in great detail in Chapter 3).

Through the use of animal models, it has already been demonstrated that pressure buildups within the middle ear and/or inner ear have a direct effect on the physical characteristics of the tympanic membrane which allow for sound transmission into and out of the middle ear cavity. Lee and Rosowski (2001) reported the biomechanical effects of increased ear canal pressure on the middle ear structure of gerbils. Specifically the vibration velocity of both the pars tensa and pars flaccida, the two primary components of the tympanic membrane, were analyzed for varying amounts of artificially induced tympanometric pressure. Using laser Doppler velocimetry it was determined that non zero middle ear pressures (i.e. pressures not equal to the ambient surrounding pressure of the external ear canal) had the specific effect of reducing the velocity of both tympanic membrane components for stimulating frequencies less than or equal to 2000Hz (Lee &
Rosowski, 2001). This reduction of vibration velocity directly impacts the mechanical transduction of the ossicular chain and its impact on the oval window and thus the end stimulation of the basilar membrane and its hair cell structure (additional anatomical and physiological detail will be reviewed in the proceeding chapter).

In a different study inner ear pressure was monitored in a total of 10 guinea pigs who were analyzed while sedated. Increases in middle ear pressure were accomplished by artificially increasing the perilymphatic and endolymphatic pressure within the cochlea (via tube innervation through the subarachnoid space). Increases in positive pressures resulted in tympanic membrane stiffness which resulted in low frequency losses in sound transduction (Jang et al., 2009). This agrees with the definition of acoustic admittance and its inverse property acoustic impedance which this study will review in detail during Chapter 3. For now it is sufficient to note that changes in middle ear pressure create changes or modifications in the physical compliance of the tympanic membrane. And much like the head of a drum, increases in stiffness bring about changes in acoustic admittance and reflectivity, thus it should not be surprising to come to the conclusion that changes in middle ear pressure directly affect the result of middle ear dependent testing, such as the OAE.

1.1 REVIEW OF PAST RESEARCH EFFORTS

The effects of static pressure on otoacoustic emissions have been recorded in several studies in the past. Naeve et al. (1992) recorded the effects of static pressure within the -200 to +200 daPa range. The transient OAE (TEOAE) responses of several volunteers were recorded under normal and induced pressures. Both positive and negative ear canal pressures induced the same outcome which was a reduction of low frequency
components within the TEOAE signal. The response of the OAE signal to static pressure resembled the inherent characteristics of a high pass filter with cut off frequency of 2600Hz and a slope of 4dB per octave. The higher ear canal pressures brought the TEOAE signals to below the noise levels of the recording instrumentation, thus suggesting that severe negative middle ear pressures, occurring naturally in patients, could result in false positives during an OAE screening session (Naeve et al., 1992).

Trine et al. (1993) performed a follow up study analyzing 14 patients who were specifically exhibiting tympanometric peak pressures (TPP) from -100 to -310 daPa. Unequalized ear results showed a dramatic attenuation effect at low frequencies (i.e. ≤ 1kHz). Pressure equalization, via static negative ear canal pressure produced by the measurement probe, brought upon increased TEOAE amplitudes as well as a higher rate of reproducibility in almost all patients. Perhaps the most dramatic result of the study was the TEOAE effect in two specific patients during equalization. With both of these patients, their initial, unequalized OAE screening yielded a FAIL result (i.e. reproducibility less than 50%) which indicated that they would be ideal candidates for middle ear pathology, thus additional testing would be needed. However, after pressure equalization, reproducibility rates improved from 42% to 83% in one patient and 49% to 78% in the other. This result clearly indicates that negative middle ear pressures have a direct correlation with false positive screenings in patients with mild to progressive NMEPs. This study also demonstrated that the best improvements are not proportional to the largest NMEP present. Therefore pressure equalization can have a very dramatic end effect on patients who may not even be aware of naturally occurring NMEPs. Pressure equalization also brought on an improvement of stimulus presentation within the ear
canal with respect to stimulus ringing as measured by the instrumentation probe. The stimulus spectrum was also smoother, which could have contributed to the increase in TEOAE amplitudes, outside of the boost effect gained during admittance changes.

Similar low frequency attenuation effects were seen by Plinkert et al. in 1994, who conducted research on the effect of middle ear pressure on both the TEOAE and DPOAE. Again static pressure within the ear canal was varied between -200 and +200 daPa. Both TEOAE and DPOAE signals were greatly reduced at frequencies less than or equal to 2kHz. A slight dependence on the polarity of the ear canal pressure was noted with positive ear canal pressures (i.e. negative middle ear pressures) producing attenuations 0.6dB higher than those at normal atmospheric conditions. However these results, when showed with respect to the drop off in acoustic admittance at normal and compensated pressures, tend to become more similar to each other than the results when the ear canal has no extraneous pressure gradient and the middle ear exhibits a normal 0daPa TPP spike.

Marshall et al., in their 1997 study, had the main purpose of illustrating the effects of negative middle ear pressure on both the stimulus and the evoked TEOAE. Their study was composed of three parts: (1) measuring NMEPs and TEOAEs over the course of several months, (2) measuring the effect of varying ear canal pressures on the stimulus, and (3) measuring TEOAEs while compensated and compare the results to when the patient had shown normal middle ear pressure. The significant factor of this study was that it was performed on one single volunteer who during the course of several months recorded a variable middle ear pressure due to extraneous conditions. Thus OAE conditions were tested on the patient when he was exhibiting normal middle ear TPP,
when significant negative TPP was present, and when the patient exhibited normal TPP but a negative middle ear pressure was artificially created by induced positive ear canal pressure. The study showed that NMEPs at -105, -135, and -165 daPa significantly affected both the stimulus and TEOAE spectra for the frequency range up to 2 kHz. Compensation for these NMEPs via pressurization of the ear canal resulted in a restoration of the TEOAE spectral signal amplitudes for frequencies above 2 kHz. Lower frequency components were elevated slightly relative to the amplitudes seen during a normal 0 daPa TPP. Results such as these illustrate the effect of MEP on test reliability.

Typically for all OAE measurements, the stimulus sound emitted from the diagnostic probe is calibrated within the patient's ear. The calibration ensures that the stimulus is as close to having a flat frequency response as possible. Significant changes to the stimulus spectrum within the ear canal could be seen as early as -16 to -30 daPa from normal ambient calibration. However, stimulus variation was not practically significant until around -50 daPa, where the additional deviations could cause artificial OAEs to be recorded in the system. Both positive and negative pressures had very similar effects to the stimulus amplitude in the frequency domain thus hinting that stimulus changes are not sensitive to pressure polarity. Stimulus changes were as large as 10 dB in some frequencies which are large enough to alter emission amplitudes. This result hints that the flat spectral response the clinician assumes is being presented to the tympanic membrane may not be flat at all when the testing is pressure compensated to elicit the most repeatable OAE responses. Thus, in ear calibration of the stimulus is needed once the compensation pressure has been reached. The study also showed that simulation of NMEPs by positive ear canal pressures does indeed correlate well with naturally
occurring NMEPs and can be used to generate data with regard to negative pressure within the middle ear.

1.2 RESEARCH PURPOSE AND GOALS

As previously discussed naturally occurring moderate to large TPP shifts occur in a large percentage of the population and are not correlated with hearing loss when evaluated as a standalone parameter. However, these shifts do have a significant effect on recorded OAE signal amplitudes which is the primary characteristic taken into account when determining the pass/refer test result. In addition to this, secondary non-hearing related testing conditions such as increased altitude or barometric pressure have been shown to impart a similar effect on the recorded OAE amplitude (Hauser et al., 1993).

Commercially available OAE diagnostic screeners do not compensate for naturally occurring middle ear pressures or ambient ear canal pressures which can impart similar false positive OAE screening results. The development of a self-compensating pressure system, which can also simultaneously administer OAE screening, will correct for this and provide compensated OAE screening results which accurately reflect the health of the cochlear system independent of these non-hearing health related test conditions.

This study is divided into two main parts. The first section is a design study which focuses on the proposal and implementation of a self-compensating syringe pump aimed at evaluating and compensating for naturally occurring TPP shifts in subjects. This section delves into the engineering aspects necessary in producing an early stage prototype capable of administering a static pressure within the canal based on a TPP threshold established during a pre-test screen and maintaining that pressure during
TEOAE screening. The second part of the study concentrates on IRB sanctioned volunteer testing aimed at establishing correlations between the effects of static pressure and the frequency/time domain responses of the meatal portion of the collected signal as well as the TEOAE portion. The research aspect will conduct a subject evaluation of 20 ears testing several pressure and stimulus conditions. The resulting test matrix will allow us to statistically compare the correlative effects of external ear canal pressure during OAE screening. There are a number of questions this research will explore. How does pressure affect TEOAE morphology changes in the time domain? How does pressure affect meatal morphology changes in the time domain? How does pressure affect the TEOAE frequency bands? The purpose will be to perform both frequency and time domain analysis on the collected signals so as to quantify the effects a potentially market viable pressure compensation OAE screener would help mitigate.

During our review of past research efforts, an overwhelming percentage of the results have been limited to the simple evaluation of OAE signal amplitude changes in the frequency domain. In order to better understand the effects of pressure compensation on the otoacoustic emission, this study will focus on the individual analysis of both the subject’s meatal response as well as TEOAE results in both the time and frequency domains. We will explore the correlation of signals based on pressure polarity and will analyze trends in signal phase shifts experienced during a range of static pressures. Lastly, we will review statistical significance of TEOAE and meatal responses collected at the various static pressures within the test battery.

The following chapters will provide a brief summary of hearing physiology and a review of some important diagnostic testing basics. Subsequent chapters will present a
detailed discussion of design and testing methods used within the study, including a
detailed review of the prototype electro-mechanical pump design which includes patient
safety considerations. The proposed system requirements will be discussed as well as the
testing protocol established for the IRB volunteers. Conclusions and further discussion of
the results will be addressed in the final chapter.
As complex as the human hearing mechanism is, very often it is beneficial to simplify its intricate biological functionality into subcomponent mechanical analogues. This allows us to view and define acoustic signal transmission through the auditory canal, tympanic membrane, middle ear space and ultimately into the cochlear mechanism, thus further understanding the variations and transformations those signals undergo on their way to their final expressions.

This chapter will focus on the review of the anatomical and physiological mechanisms involved in sound transmission, amplification, attenuation and frequency resolution within the human ear. This chapter will also give a short review of the neurological signal transmission of the hair cell system due to its vital role in sound conditioning. Finally, the chapter will examine the effects of sound pressure levels within the auditory canal and their dynamic effect on tympanic membrane compliance and acoustic impedance.
2.1 THE HUMAN HEARING SYSTEM

The human ear is divided into three main anatomical areas: (1) outer ear (2) middle ear and (3) inner ear. The outer ear is comprised of the cartilaginous pinna and the auditory canal or external acoustic meatus. The role of the outer ear (external ear) is seemingly simple in comparison to the functions of both the middle and inner ear sections. The primary function of the external ear is to funnel in sound from an individual’s environment and then transmit those acoustic signals down the conduit of the external acoustic meatus towards the tympanic membrane which is the opening of the middle ear. Secondary functions include sound localization and directional analysis based on the convolutions which are characteristic of the outer ear. With regards to this research, much emphasis will be given on the external auditory canal and its properties as most of the testing explained in further sections specifically deal with pressure and sound level characteristics within the ear canal.

Typical meatal geometry varies quite substantially between males and females and even within the same sex. Although usually depicted as a straight tube connecting the pinna to the tympanic membrane surface, most ear canals offer a 30-45 degree incline angle from the meatal opening to the back of the canal. In addition there are usually at least two primary turns within the canal which sound must navigate before reaching the tympanic membrane. These anatomical features greatly aid in the physical separation and protection of the sensitive and delicate tympanic membrane and middle/inner ear components from the outside environment. Typically the meatal opening is not circular in cross section but exhibits a more elliptical area with its vertical length approximating 1.5–2 times the dimensional width in the horizontal plane. As the ear canal progresses
closer to the tympanic membrane it becomes somewhat more circular. When considering this elliptical cross section, typical findings as reported by Salvinelli et.al, place the vertical dimension at 9.3mm and the horizontal at 4.8mm or a height to width ratio of 9.3/4.8 which typically happens around the second bend (Salvinelli et al., 1991). These findings were based on dimensional analysis of castings produced by a sample size of 280 individual ears. The average male canal (after the initial opening) showed an average ratio of 9.7/5.1 and the female average was found to be 8.5/4.4. This coupled with the overall canal length, which averages to about 23.5mm across the total population (i.e. 25.2mm in males, and 22.5mm in females) creates the mechanical analogue of a pipe resonator, effectively boosting hearing sensitivity in the range between 2000 and 5000 Hz.

Towards the end of auditory canal, sound comes into contact with the tympanic membrane or eardrum. This elastic membrane is the primary component in the middle ear region which further consists of the ossicles, tensor tympani muscle, and stapedius muscle. The two core functions of the middle ear system are force amplification and sound transduction. Within the middle ear region there is also a pressure shunting branch via the Eustachian tube. This branch acts as pressure relief valve within the middle ear due to the air tight sealing around the tympanic membrane. The activation of this shunting branch is activated during swallowing and can quickly equalize pressure differences between the middle and outer ear. Chapter 3 will later describe several testing methods which take advantage of this shunting branch to introduce pressure buildups within the middle ear during hearing diagnostics, as well as natural pressure offsets occurring in a wide population of children and adults.
The final region of the human hearing system is the inner ear. The interface between the middle and inner ears is the stapes and its physical connection with the oval window of the cochlea. The primary function of the inner ear is frequency resolution and neurological transduction of hydrodynamic motion into frequency coded impulses which ultimately act on the auditory cortex of the brain. Further sections will look at specific mechanisms which allow middle and inner ear functions as described above, however for the meantime the human ear can be globally depicted as shown in figure 2.1

Figure 2.1 Summary diagram of the human hearing system showing the three partitions of the ear (outer, middle, and inner ears). Sound pressure from the ambient surround is funneled through the outer ear, acting on the tympanic membrane which transduces vibrations into mechanical energy within the middle ear. This transduced energy arrives at the oval entry to the inner ear where sound is tonally mapped and neurologically transmitted to the auditory centers of the brain.

The above diagram summarizes the functionality of the hearing system as a whole. Sound enters the ear through the auditory canal where it is amplified mechanically in the middle ear, and ultimately separated by frequency via the inner ear mechanism. Detail surrounding middle and inner ear mechanisms will be covered in the proceeding sections. These specialized physiological apparati impart certain acoustic properties upon sound transduction through the entire system which will ultimately affect the core of the
research proposed in this dissertation. For the sake of comparison, an illustration of the human ear is given in figure 2.2. The anatomical illustration can be directly compared to the block diagram shown in figure 2.1. Notice the representation of the cochlea exhibited in figure 2.1 is much different than the anatomical illustration. However, the frequency or tonal mapping still applies. High frequency sounds are mapped closest to the oval window and low frequencies are mapped as sound waves propagate towards the helicotrema at the distal end of the cochlea.

![Anatomical illustration of major components of the Outer, Middle and Inner Ear](http://commons.wikimedia.org/wiki/File:Anatomy_of_the_Human_Ear_blank.svg, April 4, 2011)

**Figure 2.2 Anatomical illustration of major components of the Outer, Middle and Inner Ear (Adapted from Wikimedia Creative Commons; Chittka L, Brockmann)**

2.2 MIDDLE EAR MECHANISMS

As previously stated the middle ear is responsible for two primary functions: (1) force amplification and (2) sound transduction. Both are interrelated as the primary goal of both functions is to convert incoming oscillations given off by the tympanic membrane in response to incoming acoustic signals, into similar yet amplified oscillations at the stapes to oval window interface.
2.2.1 FORCE AND PRESSURE AMPLIFICATION VIA OSSICULAR CHAIN LEVER ACTION

Sound transduction within the middle ear is accomplished via lever action of the ossicular bones, which is a mechanism used to impart force amplification. There are three ossicles in the middle ear. Attached directly to the tympanic membrane is the malleus which connects to the incus which in turn forms a flexible connection with the stapes. The flat portion of the stapes interfaces with the oval window of the cochlea much like a piston does. Because of these interconnects, when sound pressure enters the auditory canal, changes in surface pressure on the tympanic membrane cause ripple vibrations which in turn impart a motion to the malleus. The malleus is rigidly fixed to the incus who is in turn connected to the stapes via flexible joint. The stapes thus begins its piston like motion within the oval window creating pressure waves within the perilymph/endolymph filled cochlea. These three bones which link the tympanic membrane to the inner ear can be simplified mechanically to a system of two bars joined together by a third rigid member.

Figure 2.3 illustrates both an anatomical view of the ossicular bone placement as well as a simplified rigid lever arrangement. A small force $F_1$ (tympanic membrane vibration) is allowed to act through a larger distance $D_1$ (malleus) thus increasing its moment. Due to the rigid linkage (incus), the outcome is a larger force $F_2$ (motion of the stapes through the oval window) acting through a smaller distance $D_2$ (stapes). The resulting stapedial piston action occurs over a very small surface area when compared to tympanic membrane motion. Thus a very small pressure change on a large surface area (i.e. tympanic membrane) imparts a very large pressure change on a small area (oval window) and both sound amplification and transduction from acoustic energy to
mechanical motion is realized. This mechanical amplification via lever system can result in approximately 1.5 times the original pressure exerted on the tympanic membrane. The differences in acting surface area account for an additional 20 fold multiplicative factor thus the combined system imparts a 30 times amplification to the original force/pressure exerted on the tympanic membrane (Bear et al., 2001).

The need for such a transduction and amplification system arises from the fact that sound travels through the auditory canal using air as its medium. The inner ear cochlea is a fluid filled hydrodynamic system with a very strong pressure gradient acting on the back end of the oval window. If acoustic energy were to act upon the oval window directly this high internal pressure would cause most if not all acoustic energy to be reflected back out the outer ear. Without the function of the middle ear sound transmission is all but lost.

![Figure 2.3](image)

**Figure 2.3** (A) Anatomical illustration of ossicular components, (B) simplified mechanical analogue illustrating three bar rigid lever system. F2 imparts approximately 1.5 times the force exerted by F1. Due to the differences in surface area between the tympanic membrane (TM) and the oval window, the resulting force amplification is approximately 30 times that originally exerted on the TM (Adapted from Bear, Connors, Paradiso, 2001)
2.2.2 SOUND ATTENUATION VIA MUSCULAR CONTRACTION

The ossicles within the middle ear, have two muscular connections. The tensor tympani is connected to bone on one end and the malleus on the other. Likewise the stapedius muscle extends from temporal bone to the stapes. When the muscles contract involuntarily due to sudden loud sounds, the ossicles become extremely rigid and sound transmission is greatly attenuated. The tensor tympani pulls the malleus away from the tympanic membrane causing a decrease in compliance and admittance, and a rise in impedance on the surface of the eardrum. The stapedius muscle, in turn, pulls the stapes away from the oval window causing a physical disconnect between the middle ear transduction system and the inner ear. This is a natural reflex within these muscle groups which serves the purpose of protection from sudden loud, and potentially harmful noises. The attenuation reflex is much larger at lower frequencies than in high. Typical delays between stimuli and muscle contraction is typically 50-100ms. This natural delay explains why it is still possible to severely injure the cochlea if exposed to sudden loud sound levels of high intensity.

Later sections will discuss in more detail the diagnostic ramifications of testing the acoustic reflex within a patient. Diagnosis of reflex issues could point to middle ear pathologies or even neurological deficiencies. This study will also include this specific form of testing under variable pressure gradients to determine if the effect is altered in any way under loaded ear canal static pressure.

2.2.3 ACOUSTIC IMPEDANCE AND SOUND PRESSURE

The research presented in this work centers around static positive and negative pressure gradients within the ear canal and their effect on otoacoustic emission screening,
tympanometry and acoustic reflex diagnostics. The general presence of pressure, albeit
negative or positive, within the ear canal causes a decrease in tympanic membrane
compliance as well as a decrease in admittance. In fact this relationship is the basis for
tympanometry, a fundamental audiological test administered to diagnose specific middle
ear pathologies. More specifics of acoustic admittance screening will be covered in the
following chapter, but for the time being it is important to understand the basic
relationship between pressure, geometry and acoustic impedance.

When discussing acoustic impedance, pressure and volume velocity, it is best to
convert these physical characteristics to their electrical analogs. Static pressure is related
to voltage as volume velocity is to current thus the concept of acoustic impedance, which
is the inverse relationship to acoustic admittance just as electrical impedance is inversely
related to electrical admittance. The concept of characteristic acoustic impedance of any
surface is dictated by the relationship below:

\[ Z_0 = \frac{P}{U} \]  

(2.1)

where \( Z_0 \) is the characteristic impedance, \( P \) is the static pressure applied (or variable
pressure in the case of sound waves), and \( U \) is the volume velocity. Volume velocity can
best be described as the movement caused by a sound wave of a unit volume of a sound-
transmitting medium through a unit area per unit of time. With this construct a
relationship between acoustic impedance and the particular characteristics of air may be
formed, since this is the medium this study will be concentrating its research efforts on.
Thus, the relationship can be characterized as follows:

\[ Z_0 = \frac{\rho_{air} \cdot c}{A} \]  

(2.2)
where $\rho_{\text{air}}$ is the density of air and $c$ is the speed of sound, while $A$ is the cross sectional area of the surface in question. However while these may be accurate models for the acoustic response of a solid wall tube, these definitions alone fall short of completely identifying the effects of static pressure inside the human auditory canal.

Much research has been launched to determine accurate models for tympanic membrane impedance (e.g. Møller, 1965; Rabinowitz, 1981; Voss et al., 2000). Due to secondary effects such as geometry, sound absorption, reflectivity, anatomical asymmetricalities and ear canal volume, a much more sophisticated model of acoustic impedance is needed in order to determine the true impedance at the tympanic membrane. The major issue with the two definitions of acoustic impedance proposed in (2.1) and (2.2) is that they assume a purely resistive load and approach acoustic impedance with a DC-analysis type mindset. As discussed previously the ear canal is anything but a straight homogenous tube, thus many of its anatomical and physiological attributes lend frequency specific modalities to the impedance levels seen in the canal and tympanic membrane. A complete, frequency specific model of tympanic membrane impedance is better described in equation (2.3) (Voss et al., 2000):

$$Z_{TM} = Z_0 \cdot \frac{Z_{EC} - jZ_0 \cdot \tan(kt)}{Z_0 - jZ_{EC} \cdot \tan(kt)} \tag{2.3}$$

where $Z_{TM}$ is the acoustic impedance of the tympanic membrane, $Z_0$ is the characteristic impedance of the tympanic membrane as described in (2.1) and (2.2), $Z_{EC}$ is the acoustic impedance of the ear canal itself, $l$ is the length of the ear canal and $k$ is $2\pi f/c$ where $c$ is the speed of sound through air as described earlier in (2.2). This expression not only accounts for frequency modalities expressed in the anatomical structure of the outer ear, but it also provides the acoustic impedance of the tympanic membrane as a function of
the impedance throughout the ear canal which is a parameter most often measured with a hearing probe. The resulting expression (2.3) provides magnitude and phase information which is frequency specific. It can also be seen that the impedance function is also dependent on ear canal pressure due to its dependency on the characteristic value. Thus, it can easily be determined from equation (2.3) that increases in ear canal pressure, whether positive or negative will bring a proportional increase to the magnitude impedance of the tympanic membrane. Thus, it should not be surprising to see the characteristic tympanometric peak curve which plots pressure on the x-axis and acoustic admittance (inverse of impedance) on the y-axis. If the middle ear is functional and healthy the curve should peak at ambient pressure due to the fact that the Eustachian tube allows for pressure equalization of the middle ear cavity. An increase or buildup of pressure in either polarity will increase the impedance of the tympanic membrane and thus drop the acoustic admittance of the tympanic membrane surface.

2.3 INNER EAR MECHANISMS

The inner ear consists of the cochlea and vestibular organs. The vestibular system contributes nothing to the auditory process but rather acts as a accelerometer and tilt sensor for the body. It provides a person with their sense of balance and spatial orientation with respect to their position. For the sake of the research presented in this work this study will concentrate on the physiology of the cochlea.

The cochlea itself is a spiral shaped organ with a total length of approximately 32mm and an inner diameter of 2mm. The semi-hollow cross section of the cochlea is segregated into several chambers which run the length of the body. The walls and central pillar (modiolus) are made of thin bone. At the proximal base of the cochlea lies the inlet
and outlet ports where hydrodynamic pressure is generated and escapes; the oval and round windows, respectively.

2.3.1 COCHLEAR PHYSIOLOGY’S ROLE IN SOUND TRANSDUCTION

The cross section of the cochlea is divided between three independent fluid filled chambers: the scala vestibuli, scala media, and the scala tympani. These separate chambers are divided by two membranes, Reissner’s membrane separates the scala vestibuli from the scala media and the basilar membrane separates the scala tympani from the scala media. Lying on top of the basilar membrane is the organ of Corti, which contains neuronal auditory receptors called hair cells. Directly over the organ of Corti is the tectorial membrane. Because of the anatomy of the cochlea the scala vestibuli and the scala tympani join together at the apex (where the cochlea comes to a point) at a hole in the membranes called the helicotrema. Conversely, at the base of the cochlea the scala vestibuli and scala tympani are separated the most with each one meeting at a window membrane. The scala vestibuli meets at the oval window, and the scala tympani meets at the round window.

There are two types of fluid filling the cochlear walls. The perilymph is found in the scala, and the endolymph is found in the cochlear duct. These fluids as well as their ionic concentrations are almost as important as the cochlea itself. In the scala vestibuli and scala tympani the perilymph has an ionic concentration of low K+ and high Na+ (i.e. \( \approx 7:140 \text{mM} \) respectively). The scala media (cochlear duct) in turn has an ionic concentration of high K+ and low Na+ (i.e. \( \approx 150:1 \text{mM} \) respectively) thus the liquid is termed endolymph. The difference between endolymph and perilymph occurs because of the active transport of K+ across the concentration gradient and the as likely absorption
of Na+ by the stria vascularis, which lines the walls of the scala media. The difference in concentrations and the permeability of Reissner’s membrane generate a standing potential that is approximately 80 mV higher in the endolymph than in the perilymph, also termed the endocochlear potential (Tasaki & Spyropoulos, 1959); a crucial factor in sound transduction of electrical potentials within the cochlea. An illustration of the cross sectional anatomy of the cochlea is given in figure 2.4 for reference.


2.3.2 THE BASILAR MEMBRANE'S RESPONSE TO SOUND PRESSURE

The basilar membrane has two anatomical properties that determine its unique response to acoustic stimulation. The first property is the size of the membrane in respect to its location in the cochlea. The basilar membrane is wider at the apex (i.e. narrower portion of the cochlea) than at the base by a factor of 5. This is typically not shown in most anatomical illustrations due to the fact that it is most often illustrated in its natural coiled position. The second property deals with the stiffness of the membrane and how it
decreases from the base to the apex, with an approximate factor of 100 times. Due to the distribution of stiffness along the cochlea, the basilar membrane becomes naturally frequency coded by its anatomical location (von Bekesy, 1960). Figure 2.5 illustrates the natural tonal or frequency mapping of the cochlea.

![Figure 2.5 Illustrated view of tonal or frequency mapping of the cochlea along its long axis](http://www.cidpusa.org/I10-85-cochlea2.jpg, April 9, 2011)

When the stapes moves in and out of the cochlea, the movement causes a shift in endolymph which in turn bends the basilar membrane near the base. This bending motion starts a traveling wave which naturally propagates towards the apex. Depending upon the frequency of the sound the wave will travel a certain distance from the base to the apex. Higher frequency sounds will vibrate mostly at the base and quickly die out, while low frequency sounds will in turn have a much longer wavelength and will propagate a good way if not completely towards the apex.
2.3.3 THE HAIR CELL MICRO-MECHANISM AND ITS PHYSICAL IMPACT

From the auditory canal to the oval window sound has been converted from one form of mechanical energy to another in order to facilitate its transmission within the various regions of the ear. In order for the brain to appropriately process the audio information, this resulting mechanical energy must at some point be transduced into a form of electrical energy. This is the primary responsibility of the organ of Corti and its inner and outer hair cell system.

Auditory receptor hair cells, which interpret the mechanical energy of the middle ear into changes in membrane potential within the inner ear are located in the organ of Corti located on the wall of the scala media. The receptor hair cells are named so due to the fact that each cell contains around 100 stereocilia (Hudspeth, 1983). The hair cells are found in between the basilar membrane and the reticular lamina. Some supporting structures include the rods of Corti, which provide structural support. Hair cells positioned between the modiolus and the rods of Corti are called inner hair cells, all other hair cells are considered outer hair cells. Hair cells form neuronal connections whose cell bodies are in the spiral ganglion within the modiolus. Axons from the spiral ganglion enter the auditory-vestibular nerve (8th nerve).

The hair cells are incredibly susceptible to motion. The basilar membrane holds down the bottom of the hair cells, while the tectorial membrane holds the tips of the stereocilia found on the tops of the cells. Due to the fact that both membranes are somewhat physically independent from one another, a movement along the basilar membrane caused by the stapes, causes bending of the hair cells which are held in place by the tectorial membrane. To obtain a better grasp of the immensity of this transduction
system imagine that the loudest possible sound (without pain) moves the cilia about 20 nm to each side, then imagine that the faintest sound you hear would move the cilia by about 0.3 nm (Hudspeth, 1997).

Because each hair cell contains potassium channels at the tips of the stereocilia which open or close depending on the direction of ciliary movement, the slight motions trigger chemical voltage potential changes within each hair cell. In addition each channel is linked to another channel on the adjacent stereocilia by an elastic filament. When the cilia are straight (i.e. small tension on the filament), the K+ channels are partially open and thus K+ leakage into the hair cell occurs. Displacement to either side cause the filaments to stretch more or relax which triggers a full opening or closing of the K+ channels respectively. The entry of K+ into the hair cell causes a depolarization which triggers voltage gated Ca2+ channels to open and allow the influx of Ca2+, causing a neurotransmitter to be released. The release of this neurotransmitter activates the spiral ganglion fibers lying postsynaptic to the hair cell. Figure 2.6 shows the depolarization event within an individual hair cell via movement of potassium ions into the cell.

Figure 2.6 Depolarization of an individual hair cell as potassium rushes into the cell triggering the release of neurotransmitter. (adapted from Bear, Connors, Paradiso; 2001, pg. 365)
The output of hundreds of outer and inner hair cells ultimately converges to create the auditory nerve which is made of neural axons whose cell bodies are found in the spiral ganglion. The innervation of hair cells varies between inner and outer hair cells. One spiral ganglion fiber receives input from only one inner hair cell, while for outer hair cells multiple cells may be feeding one ganglion fiber. This is mainly due to the fact that outer hair cells outnumber spiral ganglions. It can thus be assumed that most sound information is processed by the inner hair cells. Conversely, outer hair cells provide something very important, a mechanical amplification of the basilar membrane’s response. Motor proteins found with outer hair cells provide changes in length for the stereocilia. This change in length pulls or pushes away the basilar membrane from the reticular lamina and tectorial membrane. These simple movements provide about a 100 times amplification of the basilar membrane’s response (Hudspeth, 1997; Ashmore & Kolston, 1994). Thus outer hair cell function is crucial for the detection of soft, low intensity sounds. This mechanism is also the reason why otoacoustic emissions exist.

2.4 OAE GENERATION

The physiological mechanisms previously discussed all have a hand in the elicitation and recording of otoacoustic emissions. In the following chapter we will discuss specific parameters and test conditions used when screening for the various forms of OAE, however for the time being it is sufficient to note that recording of these emissions involves all of the three major divisions of the ear as previously discussed. Sound stimulus enters through the outer ear, it must pass through the tympanic membrane and through the middle ear amplification path in route to the inner ear where the stimulus is frequency mapped and ultimately elicits a neurophysiological as well as an acoustic
response from the cochlea. The OAE must then make the same path in reverse in order for the emission to be perceived at the recording instrument. This bi-directional confirmation of the auditory pathway is why the OAE has become a staple when attempting to determine hearing health within an individual.

OAE generation has been associated with the amplification function of the cochlea and the hair cell structure which makes this amplification possible (Kemp, 1978). As previously discussed, stimulation of the cochlear hair cells elicits a physical motion. This motion in turn will move the fluids found within the cochlea. Motion of these fluids is imparted to the ossicles via the oval window interface which in turn vibrate the tympanic membrane causing the sound emission within the ear canal. Thus when presented with a stimulus, as is the case during evoked OAE screening, a healthy cochlear system will always respond with a frequency specific echo which the recording instrument collects as the OAE. In the case of spontaneous emissions, the sensitivity of the cochlear amplifier is very high and motion of the hair cells can be triggered without a stimulus event.

As a result of their generation, otoacoustic emissions are frequency specific and frequency selective. This allows us to determine not only overall inner ear health but also if damage does occur to the inner ear, we may isolate the portion of the cochlea potentially damaged from the base to the apex. In general, only normal and healthy ears are capable of generating OAEs throughout the entire stimulated spectrum.
When a person is said to be experiencing hearing loss, the auditory deficit could be attributed to a number of factors. From the review in chapter two, several physiological conditions and situations could contribute to acute or chronic hearing loss. Cerumen or debris within the auditory canal, if not regularly cleaned, could potentially build up or become impacted. At its worst case excessive cerumen could press up against the wall of the tympanic membrane causing an increase in surface pressure, discomfort and a decrease in hearing sensitivity. With regard to the middle ear, the ossicles could become disconnected or fractured thus severing the transduction system necessary in interfacing the external ear with the inner ear. This specific pathology is referred to as ossicular disjunction or discontinuity, and is seen in cases resulting from severe head trauma. Within the inner ear region, a number of neurological issues could affect auditory nerve transduction. In order to better understand the root cause of hearing loss in patients, several diagnostic cues are needed so that false positives can be eliminated and proper diagnoses achieved.
The consensus of modern audiology is that no single test can truly determine the depth or nature of hearing loss. Thus, a fundamental test battery must be administered to the patient in order to properly screen infants, or adult patients complaining of hearing loss. The American Academy of Audiology (AAA) has been advocating the use of a test battery involving tympanometry, acoustic reflex, otoacoustic emissions, behavioral audiometry, and auditory brainstem response, with the hope of providing a strong testing base which would provide a complete picture of the pathology of the middle ear or inner ear and thus determine if the hearing loss is mechanical or neurological in nature.

This chapter will provide a review of modern OAE screening as well as basic tympanometry. The chapter will also discuss what the audiologist seeks to accomplish with each one, as well as technical information regarding proper interpretation of test results. The understanding of these basic core competencies within the clinical environment will serve us greatly as this study later explores pressure compensated testing variations for specific types of patients.

3.1 FUNDAMENTALS OF OTOACOUSTIC EMISSIONS SCREENING

One of the most fundamental hearing diagnostic tests performed by audiologists and hearing technicians today is the otoacoustic emission (OAE) screening. By definition, OAEs are low-level acoustic responses generated by the outer hair cells which are picked up by a sensitive low noise microphone placed within the auditory canal in the form of a probe. The OAE results from a natural active response from the cochlear amplifier and thus the lack of evoked OAEs can lead to the diagnosis of cochlear damage.

There are two general types of OAEs: evoked and spontaneous. Spontaneous OAEs (SOAEs) occur naturally in most of the population although the overall prevalence
is low. It occurs mainly in females and in children (Hall, 1999). The evoked OAEs can be divided into three sub groups: transient evoked (TEOAE), distortion product (DPOAE), and stimulus frequency (SFOAE). The last will not be discussed mainly due to its lack of use in clinical audiology. However, the transient and distortion product emissions are extremely important and have large prevalence in healthy human ears (i.e. \( \approx 99\% \)) making them a more than suitable test for determining cochlear integrity (Hall, 1999).

While not as complete or definitive an examination as a classical audiogram, the OAE diagnostic test is extremely attractive for one major reason. The entire test is completely objective and any elicitation of OAE signals is completely involuntary. Unlike an audiogram where the results depend on the cooperation of the patient, OAEs may be screened even without the patient’s knowledge, thus while hearing diagnostics such as an audiogram may be administered to a young child, OAE testing may be administered to newborns, thus, increasing the prevalence of screening infants for hearing loss and allowing the audiologist to implement the necessary protocols at an earlier age.

In order to protect against inaccurate testing results, there are two major checks a clinician performs before screening a patient for OAEs. The clinician must check the ear canal for occlusions or blockages. And the clinician must ensure that the probe has a good seal within the auditory canal while maintaining patient comfort. These two issues can dramatically alter the results of OAE testing and record false positives or negatives depending on the error.

In order to check the ear canal for abnormalities or obstructions an audiologist typically will perform an otoscopic inspection. According to Hall (Hall, 1999), there are four main reasons for this procedure. First, the clinician can determine if an OAE exam
will aid in screening the patient or if any abnormalities or ear canal pathology would render the test useless. Second, time lost in unsuccessful testing due to debris blockage of the ear canal can be avoided. Thirdly, information from the otoscopic inspection may yield information useful to the correct interpretation of the OAE data. Fourth, the determination and documentation of a normal and healthy ear canal can aid in the defense against claims that injury occurred because of testing.

The second check that must be addressed before an OAE test is administered is whether or not the probe has been successfully inserted. An improper probe fit can lead to erroneous data or no data at all, it is for this reason that most diagnostic software comes with a probe fit view on the main program screen which alerts the audiologist or clinician whether or not the probe has fit well into the auditory canal. A break in the probe seal would cause a mismatch of acoustic impedance at the entry boundary and this causes erroneous results in the diagnostic software.

### 3.1.1 TRANSIENT EVOKED OAE

Transient evoked otoacoustic emissions (TEOAEs) derive their name from the method of which OAE stimulation is achieved. These emissions are elicited from the ear in response to a sudden acoustic stimulus, either a click or tone burst. There is an inversely proportional relationship between the stimulus length and the range of frequencies the stimulus comprises, thus the shorter the length of the sound stimulus the broader the frequency spectrum affected. Typically a click stimulus lasting about 100 µs will have a broad band frequency response when viewed through a spectrum analyzer, while a pure tone burst, which lasts about 500 ms, will be limited to a single frequency. The main purpose of using a broadband tone is the idea of stimulating the cochlea from
the base to the apical end simultaneously. Because the cochlea is being stimulated from the base to apex, the patient should involuntarily respond with an OAE from low frequency to about 5000Hz. This assumes normal cochlear function, as well as the lack of middle ear pathology. The recorded OAE responses should also be elevated well above the noise floor of the recording system. Because of this fact many, if not all, TEOAE recording systems implement an averaging system with many rounds of click stimuli being elicited and their appropriate OAE recorded. Because the noise floor is ideally random the noise should in fact average out to zero while the recorded OAE echo will remain or grow with respect to the noise floor due to its repetitive elicitation and recording. Figure 3.1 shows a simplistic representation of a standard TEOAE system.

![Figure 3.1 Block diagram of a standard TEOAE recording system. Standard TEOAE testing uses an in-ear probe system fitted with one sound source, and one microphone. The acquisition system generates a multi frequency click stimulus through the sound source (receiver) which stimulates multiple areas of the cochlea at once. OAEs generated in response to the stimulus are propagated from the cochlea, back out to the ear canal and into the microphone which records the response.](image)

The diagnostic systems used in TEOAE analysis share some common themes. A display of the temporal view of the stimulus signal is usually standard, as well as transient and spectral views of the response as recorded by the microphone system. The
transient view provides the base to apical cochlear response if read from 0 to 20 ms, respectively, while the frequency spectrum contains the amplitude information with respect to the frequency of the stimulus. Both are useful when identifying potential regions of hearing loss. Also with regards to testing, there are two main types of stimuli used in TEOAE recordings: clicks, and tones. They can be delivered either as air or bone conduction stimuli. Each has a specific purpose and method of stimulation. In most current software packages a time domain view of the stimulus ring (meatal response) is shown to the technician, thus giving a visual affirmation of what the input to the patient’s ear is like. Excessive meatal ringing is typically due to excessive microphonic feedback and if indicative of a poor probe fit within the ear.

As previously mentioned, clicks contain a broad band of frequencies, which is most commonly limited by the transducer which is housed within the probe housing. In a perfect scenario, a click would contain all the frequency information at the same intensity levels across the spectrum. The closer to this performance, the better and more reliable the instrumentation becomes. It should be noted that although the click typically lasts about 0.1 ms electronically, acoustically the signal can endure for about 1-3 ms within the ear due to reverberation within the soft tissues of the canal as well as the middle ear cavities. Calibration of the speaker transducers (i.e. termed receivers) is normally done with minimal hardware filtering (i.e. band-pass or band-stop filtering) and sound level compensation via software equalizers.

Tone bursts, for all intents and purposes, are the exact opposite of clicks. They most usually consist of a single frequency and last about 3 to 5 times longer than the relatively short clicks. Tones can also be “overdriven”, a term that relates to the fact that
the intensity level can be taken above the transducers natural abilities. Such action produces distortions in the stimulus which are then of course recorded by the microphone making the data useless. Software typically protects from this by not allowing the SPL intensity of the stimulus to exceed certain predetermined levels.

3.1.2 DISTORTION PRODUCT OAE

Distortion product otoacoustic emission (DPOAE) testing stimulates the ear with two tones ($L_1$ and $L_2$) generated at close but different frequencies ($f_1$ and $f_2$). The DPOAE response is the distortion product that is created by the ear when stimulated by these closely spaced tones. Physiologically the cochlea and thus the basilar membrane are being stimulated at very close proximities therefore hair cell regions responsible for certain frequencies are overlapping in their initiated responses. The prominent DP arises at frequencies dictated by the simple relationship of $2f_1 - f_2$, other less significant DPs may also arise throughout the spectrum (Hall, 1999; Knight & Kemp, 2001).

The difference between the frequencies of the two tones is dictated by the frequency ratio. It is important to note that DPs will not be generated if the differences in tone frequency are too close or too far from this ratio. It has also been demonstrated that the intensity of the tones plays a crucial role in obtaining robust DPOAEs, as well as setting the intensity of the $f_2$ primary lower than the $f_1$ primary. Overall, an ideal clinical setting for obtaining DPOAEs would be a stimulus level between 55 and 65 dB with a relative intensity difference of 10 to 15 dBSPL, and a frequency ratio of 1.2 (Hall, 1999). With louder tones the largest DPOAE levels are recorded when both sound sources are of equal intensity. When stimulating levels decrease (i.e. less than 75dBSPL), optimal responses are produced when $L_2$ is lower in intensity than $L_1$ (Kummer et al., 1998).
Figure 3.2 shows a standard DPOAE system layout. Much like the TEOAE test, amplification of the low amplitude OAE signals is severely needed in order to observe any significant wave morphology as well as significant signal averaging for increased signal to noise ratios. For this reason most clinical testing packages come with signal conditioning software which accommodate for stimulus signal amplification as well as multi sweep signal averaging.

As in transient otoacoustic emissions, it is crucial for the microphone and probe assembly to have a very low acoustic as well as electronic noise floor. A noise floor higher than 0 dBSPL will most likely interfere in the recording of any OAEs and may render the data useless. Again time based and sweep based averaging helps but low noise components and filter designs are necessary for reliable data acquisition.
3.1.3 SPONTANEOUS OAE

The spontaneous OAE (SOAE) derives its name from the fact that they occur without any acoustic stimulus present. Due to its low prevalence in individuals with normal healthy hearing (i.e. 40-50% of the population is estimated to have these, Campbell, 1998), the presence of SOAEs is typically regarded as evidence of healthy cochlear function; however the absence of SOAEs does not act as a telling sign of abnormality or cochlear degeneration. Thus from a clinical perspective, the SOAE test is not regarded with primary concern to the audiologist. A part of this research will focus on determining if changes in middle ear pressure compensation results in the excitation of such naturally occurring elements. Figure 3.3 illustrates a typical SOAE test setup. Notice the simplified test setup due to the fact that no receiver or sound stimulus equipment is needed to perform this test.

Figure 3.3 Block diagram of a standard SOAE recording system. Standard SOAE testing uses an in-ear probe system containing a single microphone. The acquisition system does not generate any acoustical stimulus. If any OAEs are generated they are propagated from the cochlea, back out to the ear canal and into the microphone which records the response.

Much like the transient and distortion product tests the instrumentation consists of a low noise microphone system, data acquisition and filtering unit connected to a PC for
analysis and averaging. Unlike the DPOAE or TEOAE tests, no receivers (i.e. micro-miniature speakers) are needed within the diagnostic probe, although they usually are, on account that many systems which perform SOAE also perform other OAE screening functions as well as other core audiological tests.

SOAEs are generally composed of several narrow bands in the spectrum (i.e. bandwidth of 10-40Hz). There are two very similar methods of obtaining the SOAE signals. First, the instrumentation recording the signals may acquire a steady stream of audio signals from the microphone, convert the signals to the frequency domain via fast Fourier transform (FFT) and average this stream. Secondly, the instrumentation may time-lock the SOAEs to a click stimulus (Hall, 1999). The time locked signals may thus be averaged. These averaged signals may range between -5 and 15 dB thus the need for an extremely low noise probe becomes crucial as well as lowering the electronic and acoustic noise floors. In humans these SOAE peaks appear usually between 500 and 7000 Hz, and more specifically between the 1000 and 2000 Hz bands (Hall, 1999).

As previously stated, SOAEs, unlike TEOAEs and DPOAEs, do not have a high prevalence and may actually be tied to some intrinsic patient factors. Attributes such as gender, age, testing ear, body temperature, and genetic predispositions all affect the rate of occurrence of SOAEs in a patient.

### 3.1.4 OAE SCREENING AS A PASS/REFERRAL TOOL

The audiologist, when examining a patient and determining the presence of OAEs, assumes that middle ear function is operational (although a good tympanometry screening should be performed first to rule out that assumption, see section 3.2 for additional information). Sound must travel through the middle ear before affecting the
cochlea, and sound must again travel through the middle ear on its way out (i.e. as an otoacoustic emission) any pathology to the middle ear system would critically decline the ability to record any type of OAE. Thus, the primary function of the evoked OAE test is to determine if OAEs are in fact present. If this is true, two conclusions can be drawn: one, cochlear function is normal in terms of outer hair cell function, and two, the middle ear system is in healthy working condition.

The top five most common problems associated with middle ear dysfunction are negative middle ear pressure, tympanic membrane perforation, ventilation tube, otitis media, and otosclerosis. A well trained clinician can find enough information from an OAE screening test result to make a preliminary diagnosis of the source of the loss. The confirmation or elimination of these pathologies is mostly done through acoustic immittance measures of which will be discussed in the following section. Thus the typical result of a screening OAE session is a pass or referral rating based on the elicited OAE signal amplitudes. Referral ratings mean that additional testing must be performed to isolate the root cause of deficient OAE signal amplitudes.

If the disorder is a negative middle ear pressure OAEs can still be recorded unless the air to bone conduction gap exceeds 15dB. OAE amplitude is usually below normal limits, typically lower frequency components are affected more than higher frequency OAE values. In the case of tympanic membrane perforation, OAEs may be recorded until the perforation becomes too large or becomes a part of a larger problem (i.e. effusion, otitis media etc.). OAEs may be within the normal amplitude level from the range of 500-8000 Hz. In the case of eustachian tube pathology, OAEs are not very likely to be recorded at all. For TEOAEs, if the stimuli are set as clicks the temporal waveform may
show as an unusual triphasic pattern which starts at the expected level then drops and then rises again. Otitis media results in no OAEs being recorded as well as does otosclerosis. The differentiation between these pathologies most always will be referred to tympanometry for determination of the exact cause for the loss.

### 3.2 FUNDAMENTALS OF MODERN TYMPANOMETRIC SCREENING

The acquisition of OAEs from a patient typically defines normal and healthy cochlear as well as middle ear function. However what if the audiologist is not able to acquire OAEs from a patient? The middle ear along with its mechanical transduction system (the ossicles) must then be tested in order to determine if the hearing loss is truly due to middle ear pathology. Tympanometry is the dynamic measurement of acoustic immittance of the external ear and tympanic membrane as a function of pressure. A changing pressure gradient delivers negative and then positive pressure within the ear canal. Simultaneously a tone of single frequency is produced within the ear and its feedback level is recorded. Along with changes in acoustic immittance, there are physical changes to the surface of the tympanic membrane which cause differences in the signal intensity perceived at the recording microphone. Acoustic immittance is a general term referring to either acoustic impedance (i.e. the opposition of acoustic energy transfer), or acoustic admittance (i.e. the transfer of acoustic energy). Types of tympanometry range from single component tympanometry to multi-frequency, multi component tympanometry which provides a more substantial basis for middle ear diagnostics. This section will primarily delve into the basic function and diagnostic parameters of the tympanogram, as well as core theory used in the diagnosis of several middle ear pathologies.
Although the scheme behind basic tympanometry is rather simple, the implications of the test results can be extremely illuminating to an audiologist trying to determine the cause for a patient's hearing loss. In its most crude form a tympanogram does little more than present a constant pure tone to the ear canal, while simultaneously recording, via microphone, the audio feedback amplitude in the pressurized ear canal. The pure tone is typically set at 226 Hz and the pressure ranges from -300 to +300 daPa (Wiley and Fowler, 1997). The purpose of this is to record the amount of audio feedback from the tympanic membrane. When the ear canal is pressurized either negatively (vacuum) or positively (pressure) the tympanic membrane stiffness causing the wall of the tympanic membrane to become less compliant to incoming acoustic energy. Thus it is said that the acoustic admittance drops at the pressure extremes while reaching its sole peak (i.e. if the middle ear system is in fact healthy and complete) at a pressure equal to that of atmosphere (i.e. 0 daPa of external added pressure). Thus, the sound level measured from the microphone has an inversely proportional relationship with the acoustic admittance of the middle ear. That is, the larger the amplitude of the microphone signal recorded, the more acoustic energy was reflected back, thus the tympanic membrane has increased its stiffness and thus the acoustic admittance has dropped.

A tympanogram is the direct measure of the tympanic membrane’s acoustic admittance. It is this measurement that is plotted versus the pressure gradient administered inside the patient’s ear during tympanometry. In multi-component tympanometry multiple frequencies can be sent to the ear canal and thus various acoustic admittance curves (vectors) can be interpolated. The following figure 3.4 depicts a typical basic tympanogram with the four primary parameters labeled. The audiologist locates
these four characteristics and is thus capable of determining various discrepancies between an ideal tympanogram and the recorded one provided by the patient. These parameters include: tympanometric amplitude ($Y_{tm}$), tympanometric width (TW), tympanometric peak pressure (TPP), and equivalent ear canal volume (Vea).

![Figure 3.4 Example tympanogram with all major test parameters labeled (Wiley & Fowler, 1997)](image)

### 3.2.1 BASIC PARAMETERS OF THE TYMPANOGRAM

There are four major attributes of a tympanogram which must be analyzed in order to determine correctly if middle ear function is indeed normal and healthy. They are tympanometric curve amplitude, curve width, curve gradient or slope, and peak pressure. Each parameter will be identified and discussed individually in this section.

**TYMPANOMETRIC AMPLITUDE**

As seen in the previous figure 3.4 the maximum amplitude of a tympanogram is situated at or around the 0 daPa marking in a normal healthy ear (i.e. some fluctuations are normal and allow for swallowing or breathing by the patient). An audiologist is concerned with tympanometric amplitude mainly because certain pathologies involving the middle ear can decrease or increase the peak reading. Notice that the reading of the tympanic membrane acoustic admittance ($Y_{tm}$) is taken from the max peak to the tail of the tympanogram and not all the way to the bottom of the y-scale. This is done because
the tympanogram is a product of the microphone readings which does not discriminate between the effects of the tympanic membrane (TM) and the effects of the entire external ear system (i.e. ear canal + tympanic membrane wall). Thus the admittance of the ear canal (Yec) is subtracted by the audiologist by simply normalizing the peak value to the tail end of the tympanogram. This is why the tympanic amplitude is sometimes referred to as the compensated acoustic admittance.

While there is no typical value for what could be considered “healthy hearing”, an audiologist can read within a certain range to determine whether pathology is present or not, published literature and clinical testing has produced a set of such ranges which allow the audiologist to determine if their patient is indeed within the normal deviations of healthy middle ear function (Wiley and Fowler, 1997). For children the mean is a peak of 0.5 acoustic mmho (i.e. ohm spelled backwards is the standard unit of acoustic admittance), with a 90% range in between 0.2-0.9 mmho. For adults the average is 0.8 mmho with a spread of 0.3-1.4 mmho falling in the 90% prevalence category. Discrepancies between the norms in published journals are mainly attributed to testing conditions (Wiley and Fowler, 1997). Factors such as pump speed, age, gender, and tympanometric asymmetry can alter the recorded value of the peak. For example the faster the pump speed of a tympanometry system the larger the recorded peak response. The younger a patient is the lower the recorded amplitude will be. Women have lower Ytm values than men and the larger the asymmetry in a tympanogram the larger the discrepancy becomes when calculating Ytm from the positive or negative tail (Wiley and Fowler, 1997). To overcome this, the audiologist must choose a certain guideline and
standard and then make it their own responsibility to follow suit by implementing the same testing conditions as were followed in the literature.

**TYMPANOMETRIC WIDTH**

The tympanometric width (TW) is the width of the tympanogram measured at half the height from the peak to the tail, guidelines specified in ASHA (1990, 1997) (Wiley and Fowler, 1997) specifies that the tail value has to be estimated from the tympanogram at 200 daPa. Literature concerned in documenting clinical recordings of TW has reported a low correlation between tympanometric amplitude and width. The low correlation between TW and Ytm bolsters the fact that while one particular pathology may affect TW it may not directly affect Ytm. Thus because of this low correlation, variations in tympanometric width may indicate pathology which may have gone undiagnosed simply by a cursory review of the curves amplitude. Later sections will showcase how this parameter becomes crucial in the identification of key factors which identify certain middle ear pathologies.

**TYMPANOMETRIC GRADIENT**

The tympanometric gradient is a measure of the slopes of the sides of a tympanogram. The most common method of approximating the tympanometric slope is to calculate the difference between the peak and the average acoustic admittance at 50 daPa on the positive and negative sides. This measure has been all but abandoned in current clinical audiology because of the high correlation between gradient and peak. Thus the audiologist most often chooses to use the peak value because of its clear reference and ease in determination. Also because of its high correlation the gradient provides no additional information regarding middle ear pathology unlike the width.
Tympanometric gradient is a parameter which could simply be used to reinforce the clue provided for by the tympanometric amplitude.

**TYMPANOMETRIC PEAK PRESSURE**

Tympanometric peak pressure (TPP) is a measure from the peak value to the bottom of the y-axis. The benefit of measuring this value instead of just the peak amplitude is that the audiologist can detect the presence of negative pressure in the middle ear, which is typically caused by a blocked eustachian tube. Thus monitoring the TPP of a patient suffering from a middle ear effusion can allow the audiologist to view the restoration of normal middle ear function (i.e. as the TPP resolves back at 0 daPa). This is perhaps the most valid function used today by audiologists today. Its screening purposes have of course been replaced by the more robust tympanometric amplitude discussed previously.

**EQUIVALENT EAR CANAL VOLUME**

Although not a parameter found on the tympanogram itself, the equivalent ear canal volume (Vea) is an estimate of the volume between the probe tip and the tympanic membrane (assuming a tight fit between the probe and the auditory canal walls). This value can be shown on the graph as an acoustic admittance measurement due to the simple conversion between acoustic admittance and the volume of air. Thus the selection of the stimulating frequency at 226 Hz proves to be crucial for the conversion, because at this frequency 1 cc of a column of air has an acoustic admittance of 1mmho (Wiley and Fowler, 1997). Thus if the admittance on the curve is 2 acoustic mmho, the equivalent volume in the ear canal is approximately 2 cc.
These volume estimates are best measured from the height of the lower tail (either positive or negative) to the floor. The measure assumes that at the lowest value of acoustic admittance the tympanic membrane has stiffened up so much that it has altogether ruled itself out of the admittance measurement, thus the effects of the middle ear and tympanic membrane can be assumed to be a non-issue. The nominal range for Vea depends on the age of the patient. According Wiley and Fowler (Wiley and Fowler, 1997) the 90% prevalence value ranges from 0.3-0.9 cc. in young children and in adults it ranged from 0.63-1.46 cc. Vea becomes critically important when discriminating between three major pathologies, namely: tympanic membrane perforation (flat tympanogram with large volume), effusion of the middle ear (flat tympanogram with normal volume), and cerumen blockage (flat tympanogram with small volume). The following Figure 3.5 depicts all three pathological situations.

Figure 3.5 Middle ear pathologies and their effect on the tympanogram (Wiley & Fowler, 1997)
3.2.2 TYMPANOMETRY AS A DIAGNOSTIC TOOL

When diagnosing patients subtle deviations in Ytm are to be expected in normal, healthy ears from one patient to the next. It is not these small changes in acoustic admittance from the established norm that an audiologist is looking for when trying to establish their diagnoses. While there may be minor discrepancies in the final values of Ytm depending of certain testing conditions, there are major contradictions between a normal, healthy tympanogram and one which was recorded in an ear with middle ear pathology. The most common abnormality in a tympanogram is flat-lining, where instead of a well-defined peak the audiologist recovers a flat line from the diagnostic session. This suggests that the tympanic wall, at no time during pressurization, became more susceptible to incoming acoustic energy (i.e. no changes in acoustic admittance were observed). There are four major causes for flat-lining: otosclerosis, middle ear liquid, cerumen, and tympanic membrane perforations.

Otosclerosis is the growth of spongy bone around the stapes, the primary ossicle in the middle ear system. This growth effectively anchors the stapes thus damping the natural mechanical transduction system of the ossicular chain. This is viewed by the audiologist as a reduction in the peak Ytm. The presence of middle ear liquid would also dampen the effects of the middle ear by reducing the volume of air. This, in effect, reduces the entire middle ear into a hard-wall approximation where most if not all acoustic energy will be reflected back and thus the audiologist sees a completely flat tympanogram. The same can be said of cerumen in the ear canal which blocks and reflects acoustic signals back towards the microphone, or a tympanic perforation in which the admittance, as seen by the probe, no longer just involves the tympanic membrane and
the ear canal but also the middle ear volume which again becomes a hard-wall approximation. In summary any pathology which would lead to acoustic signals being mostly reflected back to the microphone diaphragm during testing would see a typical flat line condition on the tympanogram which is starkly different than the typical peak curve.

Conversely there are pathologies which can be diagnosed given a tympanogram with an increased peak $Y_{tm}$. Such pathologies always involve an adding of mass to the middle ear system and include ossicular discontinuity, external otitis, and cerumen or water adhesion to the tympanic membrane. In ossicular discontinuity, the ossicles which remain attached to the tympanic membrane add mass to the membrane’s wall. The same may be said of external otitis and cerumen and or water adhesion, which may add pus, debris or water to the tympanic membrane. The increased mass at the tympanic membrane causes an increase in acoustic admittance at peak pressure due to the increased sound absorption qualities artificially enhanced by the additional mass. This is something not seen in a healthy middle ear system and easily diagnosed when viewed on a tympanogram.

All the previously discussed pathologies involve stark variations in tympanometric amplitude, however stiffening pathologies such as otosclerosis can also narrow the TW value. The purpose of the TW value is in determining pathologies which increase the width such as otitis media (typical inflammation caused by infection of the middle ear region). Some of the most recent guidelines (ASHA, 1997, (Wiley and Fowler, 1997)) stipulate that the cutoffs for abnormal function are 235 daPa in infants, 200 daPa in older children, and 300 daPa in children with a high prevalence for middle ear infections.
3.3 FUNDAMENTALS OF ACOUSTIC REFLEX SCREENING

While tympanometry is an assessment of middle ear health in regards to sound transmission through the middle ear, acoustic reflex is a specific inquiry to the health of the muscular system surrounding the ossicles. As previously reviewed in chapter 1, the two muscles within the middle ear cavity, namely the tensor tympani and the stapedius muscle, contract due to a neurological trigger (trigeminal and facial nerves respectively) which is activated by the onset of a loud sound. This acoustic reflex mechanism is crucial for sound attenuation of sound levels which are at or around the pain threshold for a human being. For the audiologist this reflex also brings about a change in acoustic immitance, and more specifically in acoustic admittance. The acoustic reflex itself is bilateral, meaning that if the reflex occurs in one ear it will occur simultaneously in the other ear. Thus ipsilateral and or contralateral measurements may be taken with respect to the ear in which focused stimulation is occurring (as in the case of probe measurements). The activator (acoustic signal which is louder than or equal to the threshold signal) may be administered to either the same ear or the opposite ear that the probe is recording from. The reflex response will always be greater in the ipsilateral ear due to interaural shadowing and intensity attenuation due to a difference in distance. It is regular practice to record the acoustic reflex function both ipsilaterally and contralaterally due to the neuronal pathways excited by such testing.

The preceding sections will focus first on the parameters audiologists look to when recording and utilizing acoustic reflex data. The chapter will then shift focus to the clues testing results give clinicians with respect to physiological or neurological hearing damage. This later section will focus on the important variations between the neuronal...
pathways in both ipsilateral and contralateral stimulation as well as provide a diagnostic view of the acoustic reflex threshold and the reflex adaptation parameters which an audiologist analyzes when diagnosing a patient. Section 3.3.2 will discuss various pathologies in the auditory system and what their effect on the acoustic reflex would be.

### 3.3.1 BASIC PARAMETERS OF THE ACOUSTIC REFLEX

Much like tympanometry, the acoustic reflex is a measure of the tympanic membrane's (and to some extent the auditory canals) acoustic admittance. Unlike tympanometry, the acoustic reflex is a measure of acoustic admittance with respect to changes in sound pressure level (SPL) as a function of time. There is no dependency on static tympanic membrane pressure outside of that induced by changes in sound levels. When referring to acoustic reflex tests the audiologist is primarily concerned with two parameters: (1) acoustic reflex threshold and (2) acoustic reflex adaptation.

#### ACOUSTIC REFLEX THRESHOLD

The principle idea when testing a patient’s reflex threshold is in determining the lowest intensity signal that will trigger a time-locked change in acoustic immittance (meaning the stapes has moved due to contractions of the stapedius muscle and the reflex is clearly indicated by a decrease in admittance). For this reason the most common method of displaying the acoustic reflex is in the time domain. Because the stapedius muscle is a graded muscle, increases in activator intensity (dB HL) directly affect the contraction of the stapedius muscle, the hardening of the stapes and thus the decrease or notching in the admittance reflex curve. Figure 3.5 demonstrates this decrease in admittance with respect to various intensities of activator signals. In most humans reflex thresholds have various levels depending on the stimulus frequency, due to the fact that
the ear has a natural equalizer which imparts the perception that certain frequencies sound louder than others. However the response usually falls between 70-80 dB above the hearing threshold for each frequency.

![Figure 3.6 Typical acoustic reflex plot indicating changes in admittance (from Wiley & Fowler, 1997)]

Because of the involuntary nature of the stapedial contraction, acoustic reflex is often used to determine the hearing sensitivity of newborns as well as patients suspected to be faking a hearing loss. The main reason being that it is impossible to have behavioral thresholds (e.g. as recorded with patient driven audiograms) higher than the acoustic reflex thresholds for tone activated signals.

**ACOUSTIC REFLEX ADAPTATION**

Acoustic reflex adaptation is defined as the post stimulatory increase in acoustic admittance for a sustained session of acoustic stimulation. This is due to the relaxation of the stapedius muscle during a sustained acoustic stimulus. Much like any other muscle fiber, the stapedius muscle if contracted for a long duration will slowly fatigue until it can no longer bear its contractual load. As with the acoustic reflex threshold, measurements of the acoustic reflex adaptation are made along the time domain noting changes in acoustic admittance during a period of loud acoustic stimulation which does not change
intensity level or frequency. A clinical measure of this admittance change is known as the adaptation or decay half-life, and is made from the negative peak to half the value of the negative peak. A typical response and measurement is shown below in figure 3.6.

![Graph showing acoustic reflex adaptation and its half-life](image)

**Figure 3.7 Acoustic reflex adaptation and its half-life (from Wiley & Fowler, 1997)**

During the recording of acoustic reflex adaptation, stimulation is left on for anywhere between 5-30 seconds, with the sound source typically calibrated for either a 500 Hz or 1 kHz pure tone. The use of low frequency for the activator is based on the fact that most healthy patients who experience no hearing loss will not experience much decay in the lower frequencies (i.e. 500-1000 Hz) whereas both hearing loss and non-hearing loss patients have been recorded as having substantial decay in the higher frequencies (i.e. 2000-4000 Hz) (Wiley and Fowler, 1997).

### 3.3.2 Acoustic Reflex as a Diagnostic Tool

As stated previously, the contraction of the stapedius muscle is a bilateral reflex, meaning both muscles on either side of the head contract at the same time during the reflex, and contraction is not dependant on the location of the stimulus. The contraction of the muscle is brought upon by motor neurons in the facial nerve. It is also crucial to understand that while both ipsilateral and contralateral pathways share similar structure,
variations in the neuronal structure directly produce different measurements for both ears. Knowledge of these pathways and their differences is crucial for the correct diagnosing of the reflex measurement by the audiologist.

The ipsilateral pathway consists of the cochlea (COCH), eighth nerve (VIII), ventral cochlear nucleus (VCN), superior olivary complex (SOC), facial motor nucleus (FMN), and motor (stapedial) branch of cranial nerve VII (facial nerve) which directly innervates the stapedius muscle (S) in the middle ear (ME).

The contralateral pathway varies somewhat and includes the cochlea, eighth nerve, ventral cochlear nucleus, contralateral superior olivary complex, contralateral facial motor nucleus, and the contralateral motor (stapedial) branch of the facial nerve which then innervates the stapedius on the opposite side. Because the activator is positioned in the opposite ear the neuronal pathways must cross, unlike those during ipsilateral stimulation. Figure 3.7 shows both neuronal pathways and the major difference between the two.

Figure 3.8 Schematic representation of neurological pathway differences in ipsilateral and contralateral signal flow paths
Understanding the differences between the two conductive pathways is critical for applying their variances while determining the site of neurological disorder causing hearing loss in some patients. It is important to note that while the signal flow of the neuronal pathways is vital in understanding the stapedial reflex, the clinician performing everyday diagnostics mainly looks for the three parameters previously discussed in a reflex test result. Mainly the presence/absence of the acoustic reflex, the acoustic reflex threshold and the acoustic reflex adaptation.

Clinically speaking the results of an acoustic reflex test may hold the clues for the proper diagnoses or middle ear trauma, various neurological conditions, or cochlear damage. In almost all cases a patient suffering from middle ear pathology will most likely not exhibit any contralateral or ipsilateral acoustic reflexes in the affected ear. If the pathology is bilateral then of course no acoustic reflexes will be obtained at all, this of course is due to the nature of the pathology. Most middle ear pathology, as discussed previously, is essentially a hardening or stiffening of the middle ear either due to otitis media, ossicular dislocation, or other root pathology. The stiffening of the tympanic membrane of course brings about a decrease in the acoustic admittance of the ear. Thus because of the already low acoustic admittance of the middle ear, any reflex by the stapes would not produce a sufficiently large enough change such that the audiologist would be able to differentiate between normal and activated responses. Thus the patient is said to have no acoustic reflex. The nature of the pathology is also the reason why an audiologist may diagnose a patient as having acoustic reflex in the right ipsilateral (recording right ear, stimulating right) and left contralateral (recording right ear, stimulating left) but no
left ipsilateral or right contralateral. In this case the audiologist can determine that the patient is suffering some form of middle ear disorder is in the left ear.

Because the stapedius muscle is innervated by cranial nerve VII, the acoustic reflex may be used to diagnose certain problems relating to paralysis. Thus if it has been previously determined that there exists no middle ear disorder (i.e. perhaps through OAE testing) the audiologist may help diagnose a neurological hearing loss due to the absence of a stapedial reflex in a certain ear. This technique may also be used to monitor VII nerve function and postoperative recovery. The side of the neurological disorder may be diagnosed by the ear in which the audiologist is not able to record any acoustic reflex.

Portions of the neuronal pathways connecting the stapedial reflex involve parts located in the inferior portion of the brain, most importantly the cochlear nucleus and the peripheral auditory system. Central auditory pathologies proximal to the brainstem may manifest themselves as abnormal stapedial reflex patterns, while central disorders above the brainstem have no effect on stapedial reflex function. This fact can assist the audiologist in discriminating between a central and peripheral neurological disorder.

In general, patients with some form of brainstem disorder often demonstrate normal ipsilateral reflexes, while conversely generating abnormal contralateral recordings. The crossed portions of the superior olivary complex are primarily affected. As a general rule, a brainstem lesion is to be suspected if contralateral reflexes are absent and the patient has been cleared of middle ear problems and has normal hearing sensitivity. This type of diagnosing of course can only give suspicion as to what the source of the neurological disorder is. Further, more precise testing is always needed to exactly determine the cause.
Acoustic reflex testing may also serve to find pathological issues within the auditory nerve. The most common test result leading to the diagnosis of an auditory nerve (VIII) disorder is an absence of an acoustic reflex with activating signals being presented to the affected ear. Referring back to figure 3.7 which illustrates the two major neurological pathways of the acoustic reflex, the path leading to the cochlear nucleus is taken by the auditory nerve (assuming the patient has a healthy middle ear system and cochlea). Referring back to chapter 2, the inner and outer hair cells terminate at various ganglion bunches which in turn become the auditory nerve. Thus a severing, or any other damage to nerve VIII, would cause an absence of reflex for that ear being stimulated. Thus if the auditory nerve branch in the right ear was completely severed any ipsilateral stimulation in that ear would produce no stapedial reflex. Using the same reasoning an audiologist would come to the conclusion that no acoustic reflexes would be obtained in the left ear using contralateral right stimulation, because once again the activator has to pass the severed VIII nerve. Conversely, if there is auditory nerve damage but the audiologist continues to record reflexes (i.e. as in the case of a partial pathology) then it is common to observe an increase in the acoustic reflex threshold while at the same time notice rapid reflex decay. The acoustic reflex decay is so pervasive that half lives of less than 5 seconds for 500 and 1000 Hz tones are often diagnosed. It is this decay time that differentiates an auditory-neurological disorder from a cochlear pathology. In most cases cochlear disorders give rise to reflex decays that are much slower.

This brings us to the use of acoustic reflex in diagnosis of cochlear pathology. Acoustic reflexes in ears with a cochlear pathology are determined mainly by the level of sensorineural hearing loss. Patients suffering from a 50 or 55 dB HL loss will more than
likely demonstrate normal acoustic reflexes, while those patients with an 80 dB or greater loss will observe low amplitude reflexes or nothing at all (Wiley and Fowler, 1997). The relationship then becomes completely proportional in terms of hearing loss and reflex amplitude. It should also be noted that acoustic reflex decay may sometimes be a part of the test result; however this decay is substantially slower than the decay experienced in patients suffering from an auditory nerve disorder, and can easily be differentiated.

3.4 FUNDAMENTALS OF WIDEBAND ACOUSTIC REFLECTANCE

Wideband acoustic reflectance is a relatively new technique which relies on the measurement of reflected acoustic energy at the tympanic membrane to make certain correlations with the health of the middle ear space. It has been used to examine normal middle-ear function, and to obtain immittance measurements at lower (safer) sound levels than those typically used during acoustic reflex.

As sound energy enters the meatus and travels to the tympanic membrane, not all of the acoustic energy is transferred into the middle ear space and to the cochlea. The tympanic membrane absorbs a certain percentage of the incident sound pressure wave and reflects the rest back into the ear canal. The ratio between the reflected energy and the original incident power is the energy reflectance ratio (Keefe et al., 1993). The ratio is scaled from 1 (complete reflection) to 0 (all energy absorbed). This ratio gives an indication of the amount of successful transfer into the middle ear space and has been proposed as a tool for the clinical diagnosis of middle ear disorders (Feeney et al., 2003).

Feeney et al. (2003) describes the relationship between reflected energy and the presence of common middle ear disorders. The amount of energy reflected is a function of change in middle ear impedance. Low frequency reflectance increases in the presence
of disorders that lower middle ear stiffness reactance and decreases in the presence of disorders that increase the middle ear stiffness reactance (Feeney et al., 2003). Examples of decreased stiffness reactance are otosclerosis, Otis media with effusion and elevated middle ear pressures. Increases in the mass reactance component of the middle ear would make energy transfer more difficult, while also increasing reflectance of high frequency stimulus components. An example of this would be ossicular discontinuity, where much of the power would be absorbed by the middle ear, but very little would actually make it to the cochlea. Another example would be perforation of the tympanic membrane, where the impedance at the tympanic membrane increases or decreases with the middle-ear volume. Currently there is an effort underway to model these middle ear disorders with their corresponding acoustic reflectance spectral responses. The hypothesis of the reflectance method is that these middle ear disorders manifest themselves with a specific reflectance signature which is frequency specific and consistent between patients of all backgrounds.

Feeney et al (2003) describes finding such patterns including several reflectance signatures such as elevated low-frequency reflectance for cases of otosclerosis, and a deep low frequency reflectance notch in cases of ossicular discontinuity. In the presence of negative middle-ear pressure, wideband reflectance at ambient pressure was elevated, which is consistent with the tympanometric understanding of admittance versus varying pressure gradients.

Figure 3.9 shows the resulting patterns elicited in the article presented by Feeney et al. (2003). The plots show a normal reflectance baseline curve, sensorineural hearing loss, otitis media with effusion, otosclerosis, ossicular discontinuity, hypermobile
tympanic membrane, perforation of the tympanic membrane, and sensorineural hearing loss with elevated middle ear pressure. As is evident, a specific reflectance pattern can be seen for each type of middle ear disorder.

Figure 3.9 Acoustic reflectance patterns as determined during a 50 subject test study (Feeney et al., 2003). The normative reflectance pattern (A) is represented in each graph (shaded grey region) and represents the 5th to 95th percentile of 40 healthy hearing subjects. Reflectance measurements were taken in 10 subjects which demonstrated (B) sensorineural hearing loss, (C) otitis media with effusion, (D) otosclerosis, (E) ossicular discontinuity, (F) hypermobile tympanic membrane, (G) perforated tympanic membrane, and (H) sensorineural hearing loss w/ elevated middle ear pressure

3.4.1 WIDEBAND REFLECTANCE AND THE ACOUSTIC REFLEX

A rising trend in the academic and commercial audiological sectors has been the surge of broadband acoustic reflex testing. As previously discussed, basic tonal acoustic
reflex testing explores the temporal relationship between activator signal and the recorded drop in tympanic membrane admittance. Because the recording probe is only emitting the constant 1000Hz tone, the test typically relies on very high stimulatory sound levels particularly when dealing with patients who may have sensorineural hearing loss. The proposed alternative testing methods use a broadband chirp signal (250-8000Hz) to measure changes in energy reflectance, admittance, and power absorption (Feeney & Keefe, 1999; Feeney et al., 2003). One of the main objectives when introducing a wide band probe signal is to significantly and consistently lower the reflex threshold by sampling the frequency region most capable of significant change in middle ear admittance.

There have been previous findings that indicate different probe frequencies can yield lower acoustic reflex thresholds (Porter, 1972; Wilson & McBride, 1978, Burke & Herer, 1973), however it is still not evident that an optimal stimulating frequency can be applied that would yield the lowest and most sensitive and efficient reflex thresholds among adults and infants alike. Due to sensorineural differences among patients, recording at a less than optimal probe frequency may mean having to increase the stimulating tone higher than clinical devices are allowed to reach by ANSI standards. Testing with a broadband probe stimulus increases the chances of sampling the most sensitive frequency regions in any given patient, and thus allows for significantly lower stimulatory threshold levels. The results of lower threshold levels are clinically significant. Lower thresholds would allow for acoustic reflex testing in patients who have already demonstrated mild or moderate sensorineural hearing loss. Lower thresholds would also mean the increased ability to administer acoustic reflex testing in young
infants, where traditional acoustic reflex measurements are avoided due to the high sound levels typically used during the test battery.

**REFLECTANCE TESTING METHODOLOGY**

Feeney et al. (1999, 2003) have published several papers concerning a specific wide band reflectance testing methodology originally developed in 1992. Typically two individual tonal activator stimuli are tested, one at 1000Hz the other at 2000Hz. A recording probe is inserted into the acquisition ear canal complete with sound source and recording microphone. The test begins with a chirp signal consisting of frequencies between 250 to 8000Hz. On some occasions, such as the 2003 publication in JSLHR, the bandwidth of the chirp was truncated in order to determine variations within the recorded reflex thresholds. During the baseline chirp emission, the microphone response is monitored to determine that the patient is still and physically ready for testing. The activator stimulus is presented contralaterally and the reflex induced change in admittance pre and post activator is recorded. This admittance shift is then analyzed in both the time and frequency domains. As the activator level is decreased the admittance shift approaches zero, as does the cross-correlation value which indicates that response shifts are only due to baseline variability and thus uncorrelated (Feeney et al., 2003).

Results published in 2001 and 2003 from Keefe, Feeney et al were consistent in finding repeatable, contralateral reflex thresholds using this same wideband reflectance method. In both cases acoustic reflex thresholds were measured in multiple subject demographics which were on average 12-18dB lower than the predicate clinical system. In some cases thresholds were obtained as low as 24dB below the clinical reflex threshold. The results as originally published in 2003 are presented in table 3.1.
In addition to the benefits discussed above, it should also be mentioned that the overall chirp sound level used was 65dBSPL Typical ANSI standards call for a 226Hz probe tone \( \leq 90 \)dBSPL. This ANSI specified probe level could in turn produce an ipsilateral acoustic reflex prior to the activator stimulus producing a false positive during contralateral testing. The significantly lower chirp sound level eliminates this.

### 3.5 TEOAE IN CLINICAL APPLICATIONS

As discussed earlier the TEOAE (and the OAE in general) is an acoustic signal originating from the cochlea in response to an acoustic event originating outside of the external ear. The fact that the acoustic signal not only has to pass successfully through the external auditory canal., tympanic membrane, middle ear space and basilar membrane (via the oval window) to elicit the response from the cochlea, but that it must also navigate the reverse path so that the TEOAE may be recorded by the diagnostic probe, suggests strong diagnostic indicators which can help in diagnosing the root cause of
sensorineural hearing loss. A subject with perfectly normal hearing but suffering from swimmer’s ear or otitis media for example would exhibit increased middle ear pressure. This increased MEP would artificially raise the acoustic impedance of the tympanic membrane and should thus decrease the TEOAE amplitude. However, unlike severe ossicular chain dislocation some level of TEOAE (although reduced) should be recorded since the transmission path remains intact. The same may be said for subjects suffering from eustachian tube dysfunction such as tube blockages or swelling within the areas around the eustachian tube. These dysfunctions could result in increased middle ear pressure as the subject is not able to self-compensate by swallowing or clearing the tube of the pressure build up.

Zhao and associates (2003) recorded TEOAE signal responses within patients exhibiting various middle ear disorders. These disorders included tympanic membrane aberrations, otitis media, tympanic membrane perforation, otosclerosis and ossicular chain dislocation. The results of the study characterized specific TEOAE frequency amplitude changes for each type of middle ear disorder. Of the disorders most negatively affecting TEOAE amplitudes were the effects of otitis media. The amplitude of the TEOAE signal was characterized by the subject’s hearing level and middle ear mobility which is directly correlated with the resonant frequency of the middle ear cavity which in turn is greatly affected by the excessive buildup of fluid within the middle ear space. In the end subjects exhibiting chronic otitis media were all absent of the presence of significant or recordable TEOAEs. It is important to note that later on we will analyze the effects of positive ear canal pressure on the TEOAE. While positive ear canal pressure can simulate similar effects of naturally occurring negative middle ear pressure (Sun,
2012), we should note that non-linear effects of fluid buildup within the middle ear cavity and along the interior lining of the tympanic membrane are difficult to replicate by solely applying external ear canal pressure. Thus, the effects of external ear canal pressure compensation on a subject with a healthy and naturally occurring negative middle ear TPP shift would be different than that on a subject experiencing negative middle ear pressure due to a preexisting middle ear disorder such as otitis media.
In order to validate the effect of pressure gradients on otoacoustic emissions, the study needs two major acquisition tools: (1) OAE recording software complete with an off the shelf probe capable of recording low noise OAE emissions, and (2) a syringe pump which can be controlled and synchronized with the aforementioned probe and data acquisition software. For the volunteer trials this study will be using Intelligent Hearing System’s (IHSYS) SmartTrOAE DSP based software/hardware platform. This OAE recording system is capable of performing standard click TEOAE testing, spontaneous OAE testing, automatic probe fit checks, and works with standard OAE probes such as the Etymotic 10D and 10B+ probes. This platform will be paired with a custom built, automated syringe pump designed with this specific research in mind. The purpose of this chapter will be to provide detail with regard to the mechanical and electrical design of this particular syringe pump with special emphasis given to syringe volume calculations, motor selection, aspects in reliability, position sensing, pressure monitoring, and patient...
safety. This chapter will spend some time covering various hardware and system features aiding in patient safety as it will be crucial to ensure performance confidence before the study progresses to the volunteer trial evaluations.

4.1 OVERVIEW OF MECHANICAL DESIGN (CAD)

The target areas the study focused on during the design of the syringe pump were (1) low friction design, (2) small footprint or size envelope, (3) reliability, (4) precise linear motion, and (4) linear position sensing built within the pump assembly. Figure 4.1 provides an overall isometric view of the pump as designed in Pro/Engineer. In the figure all major components have been labeled as well as identified by part number.

![Figure 4.1 Isometric view of syringe pump with component part numbers and BOM detail](image)

In general the system is comprised of three stages: motor block stage, travel stage, and termination stage. The motor block stage is where the DC motor is mounted and where the front of the syringe is encased and clamped to the front stop. This section of
the syringe pump also houses the start switch, an SPDT micro switch which when pressed by the travel plate relays the starting position of the syringe pump. The travel stage is a subassembly which houses the linear bearings which in turn travel along the alignment shafts during operation. This stage also retains the back end of the syringe during operation and is responsible for the operation of the pump. The termination stage is where the lead screw terminates and is supported by a radial bearing as well as where the stop switch is housed, a secondary micro switch which operates identically to the start switch but relays the end position of linear travel.

Figure 4.2 below provides an isolated view of the motor block stage where the coupling between the motor shaft and lead screw is shown. A precision, rigid style stainless steel coupling provided by SPDI was used. The coupling consisted of two precision machined 3mm bores on both ends of the part, along with set screw fasteners which secure both the motor’s shaft as well as that of the actuating lead screw. The DC motor has a standard 3mm shaft and the lead screw was machined down on one end to 3mm to provide the insert to the rigid coupling.

Figure 4.2 Isometric view of the syringe pump’s motor block stage, complete with mounted motor and syringe (additional components have been removed for the sake of clarity)
It should be noted that during the first iterations of the pump design there were several severe alignment issues due to the fact that the initial couplings were prototyped using SLA materials or improperly toleranced machined metals. Improper coupling between the shaft of the motor and the lead screw, however minor, causes elliptical wobbling during high speeds. This wobbling, when translated through the threaded portion of the lead screw, places a large non-axial mechanical load on the lead screw during its rotation, which in turn causes the motor to stall.

Figure 4.3 below provides an isolated view of the syringe pump's travel stage. As previously mentioned this stage is responsible for pressure generation through movement of the syringe plunger. The travel stage employs two precision linear bearings which run along two precision machined shafts supported on each end by the motor block stage and termination stage. The linear bearings aid in the reduction of system friction, while also maintaining strict linearity during the pump’s motion. The benefit of such a design is increased motor efficiency and speed. Great care was taken to implement a low friction, highly linear motion. Because of this, the motor, which performs the work load, does not have to be over-specified which allows us to maintain a small footprint design. It is also important to note that the presence of these alignment rods also reduces the amount of mechanical noise during motion. Other types of syringe pump systems currently on the market rely on friction and interference to prevent rotation of the translating member, which is not preferred. As is evident in figure 4.3, the backend of the syringe plunger is captured by a stop piece which can be removed by two screws. Removal of the stop allows for servicing and/or changing of the syringe plunger.
Figure 4.3 Isometric view of the syringe pump’s travel stage which shows the linear bearings and syringe plunger (additional components have been removed for the sake of clarity)

Figure 4.4 shows the termination stage where the stop position switch and radial bearing are mounted. The radial bearing is used to allow rotation at the lead screw’s end, while still supporting the far end of the lead screw during its rotation. If the lead screw was set within a static holder (i.e. hole/slot) it could provide sufficient friction to provide long term damage or reliability issues over time.

Figure 4.4 Isometric view of the syringe pump’s termination stage which shows the supporting radial bearing and stop position switch (additional components have been removed for the sake of clarity)
One of the primary goals when designing the syringe pump was to minimize its size envelope so that it would be possible to visualize the pump unit as a portable self-contained system which could be mobile during the evaluation of patients and volunteers during the study. Figure 4.5 provides overall dimensions of the pump system.

4.1.1 SYRINGE VOLUME SELECTION

Because the research proposed in this study will culminate in the evaluation of several adult volunteers, we have based our pump's volume specification on average ear canal volumes within the adult population. Typical adult ear canal volumes trend between 0.8-1.9cc. Multiple research efforts with varying methods of calculating volumes have been published in the field. One such publication (Ojala et al., 1982) compared a liquid filling method (invasive) to compliance measurements (non-invasive). The results showed a high variability between the 123 cadaver and living ears tested. However all of
the results remained within the previously disclosed volume range. Using this information, the syringe pump was fitted with a plastic (low friction, and replaceable) 5cc syringe. The design ensured that the full stroke of the syringe could be used during both positive and negative pressure trials. The 5cc volume allows us plenty of margin while conducting pressure compensated testing. During testing it will be crucial to establish a tight probe fit within the ear canal. However, small leaks causing steady changes in system pressure during a multiple sweep OAE session should be expected, even under the best circumstances. The additional volume will allow us to consistently trigger the syringe pump in either direction to ensure that pressure within the close loop system remains within a tight tolerance during testing.

The geometry of the syringe itself was also selected to ensure quick flow rates during operation. Typical tympanogram rates are typically around 200dAPA/s. As we will see in the following section, some assumptions were initially made regarding the linear speed of the pump system, such as the assumption that a minimal linear speed of 0.125in/s (or 3.2mm/s) should be specified so as to design a pressure system which would be as equally fast if not faster than commercially available systems. From this initial constraint we can develop an expression for volume displacement within the syringe as seen in (4.1).

\[ V_{\text{displaced}} = L(\pi r^2) \]  

(4.1)

The expression above is an applied version of the simple relationship describing volume within a cylinder. Because the syringe we have chosen has a circular cross section, as the plunger moves within the syringe body, the volume displaced, is
characterized by the length the plunger travels \((L)\), and the cross sectional radius of the syringe body \((r)\). Replacing the constraint previously discussed, we obtain the following:

\[
V_{\text{displaced}} = \frac{1}{8} \pi \cdot 0.236^2
\]  

Equation (4.2) yields \(0.022\text{in}^3\), or 0.36cc. Thus if our linear actuation speed from the motor coupling to the lead screw, and ultimately the translation stage remains at 0.125in/s, we will be displacing approximately 0.36cc every second while the pump is in motion. Boyle's law may be used to estimate the transitional relationship between this displaced volume and the resulting pressure change.

\[
P_1 V_1 = P_2 V_2
\]  

\[
\therefore P_2 = \frac{(10132.5)(5)}{(5 - 0.36)} = 10918.6 \text{ dAPA}
\]  

\(P_2\) as described by equation (4.4), is the new pressure within the enclosed (and presumed perfectly sealed) syringe barrel after the syringe plunger moves 0.125in. \(P_1\) is ambient pressure (expressed in dAPA), \(V_1\) is the initial syringe volume (pre-test) which is 5cc, and \(V_2\) is the new volume post motion. The change in pressure within this quick motion is expressed as \(\Delta P\), and is expressed in (4.5) below.

\[
\Delta P = P_2 - P_1 = 786.1 \text{ dAPA}
\]  

On first inspection, equation (4.5) suggests that the pump system will be almost 3 times as fast as clinical testing systems. This is a partially misleading. While this development of volumes and pressures is crucial during the planning phases of product development, it can only serve as a liberal estimation of performance. In reality there will be boundary leaks present within the syringe itself, coupled with boundary losses due to a less than optimal probe fit. In addition, frictional loses are not being considered at the motor or travel stage portions of the pump (although as previously stated these should be
minimal). Even if the system were to perform at 40% efficiency, due to these losses, a pressurization rate of 314.4dAPA/s would still outperform existing tympanometry screeners. In order to ensure that we can attain these parameters a suitable motor was selected, as will be discussed in the following section.

### 4.1.2 MOTOR SPEED CALCULATIONS AND CONSIDERATIONS

The syringe pump designed for this study used a graphite commutated DC motor. There were several key factors which had to be calculated before selecting an appropriate motor. Initial parameters are tabled below:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>VAR</th>
<th>Value</th>
<th>Units</th>
<th>Importance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Voltage</td>
<td>Vdc</td>
<td>15</td>
<td>VDC</td>
<td>Motor operating voltage</td>
</tr>
<tr>
<td>Linear Speed</td>
<td>s</td>
<td>3.2</td>
<td>mm/s</td>
<td>Linear speed desired (converted from [in/s])</td>
</tr>
<tr>
<td>Force Displaced</td>
<td>F</td>
<td>22.2</td>
<td>N</td>
<td>Push/pull force required to overcome syringe plunger + friction</td>
</tr>
<tr>
<td>Screw Type</td>
<td>None</td>
<td>#1/4-20</td>
<td>None</td>
<td>Necessary for determining sub parameters such as lead and eff.</td>
</tr>
<tr>
<td>Screw Lead</td>
<td>l</td>
<td>1.27</td>
<td>mm/rev</td>
<td>Calculated based on the single start lead screw used for the design, (converted from [in/rev])</td>
</tr>
<tr>
<td>Screw Eff.</td>
<td>$\eta_s$</td>
<td>30</td>
<td>%</td>
<td>Estimated based on standard, high tolerance machined lead screws</td>
</tr>
</tbody>
</table>

Using the design inputs listed in table 4.1, it is now possible to calculate three major motor parameters: (a) load torque, (b) power, and (c) loaded current draw. These parameters will narrow which motors are suitable for use in the design, as well as
determine the power supply used to power the DC motor. The first parameter which must be calculated is the load torque required at the lead screw. Equation (4.6) shows the relationship between linear force and rotational torque \( M \) as seen by the motor shaft.

\[
M = \frac{FL}{2\pi \eta_s} \tag{4.6}
\]

\[
\therefore M = \frac{(22.2)(1.27)}{2\pi(30)} = 14.957 \text{ mNm} \tag{4.7}
\]

Linear speed may be converted into its rotational counterpart through the simple relationship expressed in (4.8) below.

\[
RPM = n = 60 \left(\frac{s}{I}\right) \tag{4.8}
\]

\[
\therefore n = 60 \left(\frac{3.2}{1.27}\right) = 151.2 \tag{4.9}
\]

From the load torque provided in (4.7), we can calculate the power required at the motor shaft. The relationship between load torque, rotational speed and power is shown in (4.10). This specific relationship has a unit conversion factor thus torque and speed must be expressed in [oz-in] and [RPM] respectively.

\[
P_{\text{motor}} = \frac{nM}{1350} \tag{4.10}
\]

\[
\therefore P_{\text{motor}} = \frac{(151)(2.12)}{1350} = 0.24W \tag{4.11}
\]

From the DC motors readily available from MicroMo, the 2342012CR was chosen. The motor provides a nominal loaded torque rating of 16mNm, and has unloaded speeds up to 8100RPM. Once loaded the speed rating will drop substantially, however there is a considerable amount of margin compared to what the functional criteria of the pump is. There are several parameters listed on the motor datasheet, two of which are the torque constant \( k_m \), and the no-load current \( I_o \). From these two parameters we can
conclude the worst case current draw as seen across the terminals of the motor. The relationship between motor torque and current consumption is shown in equation (4.12).

\[ I = \frac{M}{k_m} + I_o \]  
\[ \therefore I = \frac{2.12}{13.4} + .075 = 0.23A \]  

4.1.3 SYSTEM CONSIDERATIONS FOR RELIABILITY

During initial testing of the assembly shown in figure 4.1, there were several times that the travel assembly showed excessive binding and chucking, especially during startup from either extreme. It was also noticed that sometimes, primarily at the end of its stroke, the travel stage would sometimes jam against either the termination or motor block stages. A solution which was implemented to temper both situations was the addition of two compression springs on both sides of the travel stage. The compression springs were coiled around the syringe barrel and the lead screw respectively. The cushioning effect towards the end of travel eliminated binding and jamming, as well as presented a pre-load force to the travel plate which aided in the reduction of binding during startup.

Another consideration which was taken into account was the lubrication of the entire system. A silicon based lubricant was applied to the inside barrel of the syringe as well as on the lead screw and linear bearings. These points were determined to be the primary sources of friction and noise during evaluation. These adjustments will greatly aid in securing the operational reliability during clinical evaluations.
4.2 OVERVIEW OF PUMP CONTROL CIRCUIT DESIGN

There are four major sections which comprise the syringe pump electrical hardware design: (A) digital I/O interface, (B) motor driver circuit, (3) pressure transducer circuit and (4) patient safety release valve circuit. The pump control system acts as a slave to the IHS digital acquisition system. The IHS system is a commercially available OAE diagnostic system, which for the sake of this study, had custom software written expressly for the sake of obtaining pressure compensated OAEs. The IHS system provides power as well as input signals generated by the DSP in response to the windows software interface. A complete system schematic is provided in the appendix section.

4.2.1 DIGITAL I/O INTERFACE

As previously mentioned the syringe pump designed for this study is a slave system to the IHS DSP acquisition system. The main interface between the two units is a 12 pin Fischer connector which brings +15VDC, GND, and several DSP output lines along with one ADC input line which is used to monitor voltage levels from the pressure transducer. All digital lines are driven through an octal buffer chip (SN74F541DW). The resulting circuit portion is depicted in figure 4.6.

![Figure 4.6 Digital I/O interface circuit depicting pin outs for 12-pin input Fischer connector](image-url)
The DSP system provides a total of five digital inputs into the pump control board which control various functions and triggers within the syringe pump's hardware. Table 4.2 describes all of the digital inputs and their functions.

**Table 4.2 List of all DSP generated syringe pump inputs and their functions**

<table>
<thead>
<tr>
<th>Input</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>D0</td>
<td>Motor control line (PWM, 1=Forward, 0=Reverse)</td>
</tr>
<tr>
<td>D3</td>
<td>Digital switch control (A/D, Read, 1=Pressure, 0=Switches)</td>
</tr>
<tr>
<td>D4</td>
<td>Clock Control (CLK, 1=Enabled, 0=Disabled)</td>
</tr>
<tr>
<td>D5</td>
<td>Motor Shut Off (MTR, 1=Off, 0=On)</td>
</tr>
<tr>
<td>D6</td>
<td>Release Valve (RV, 1=Open, 0=Closed)</td>
</tr>
</tbody>
</table>

**4.2.2 DC MOTOR DRIVING CIRCUIT**

The syringe pump's DC motor is driven by a DMOS full bridge motor driver IC (A3950). DSP control lines D0, D4, and D5 control certain aspects of the motor driver chip, as was previously mentioned in Table 4.2. The IC controls DC motor function via pulse width modulation (PWM) and can work with supply voltages up to 36VDC. The phase and enable pins, shown in figure 4.7, control the motor's directionality as well as speed. The enable pin on the motor driver chip is controlled via an external clock signal generated on a standalone board. Early on in the development of the syringe pump there were several issues regarding consistent PWM control directly from the DSP, thus a standalone solution was created using a standard 555 timer design with potentiometer control over duty cycle and frequency. The A3950 includes internal protection for supply shorts, ground shorts, thermal shutdown, overvoltage monitoring, and cross-over current protection. The resulting circuit portion is depicted in figure 4.7.
4.2.3 PRESSURE TRANSDUCER CIRCUIT

The syringe pump uses a highly sophisticated dual port pressure transducer provided by Honeywell (ASDX001D44D). The transducer is a piezoresistive sensor, with amplified output and built in signal conditioning. The IC works from a +5VDC supply and acts as a differential gauge. In an atmospheric condition the analog output is about 2.5VDC. Positive pressure changes result in voltage increases until the transducer reaches 1PSI at which time the unit reaches its top most limit (5VDC). Negative pressure difference have the exact opposite effect whereas the bottom limit is 0VDC. The resulting circuit portion is depicted in figure 4.8, along with its isolating output buffer.

Figure 4.8 Pressure circuit stage with low pass output filter and output isolation buffer
4.2.4 PATIENT SAFETY RELEASE VALVE CIRCUIT

The core component responsible for delivering patient safety is the solenoid triggered release valve provided by LeeCo (LHDA053115H). The output pressure line from the syringe pump feeds directly into a T-coupling where one output is directed at the main patient interface panel mounted at the front of the system. The orthogonal output of the T-coupling is fed to the input port of a Y-coupling. The dual port output of the Y-coupling connects to two distinct safety and diagnostic systems. One output of the Y-coupling connects to the input port of the pressure transducer, where the pressure within the closed loop pneumatic circuit is monitored and reported. The other output is connected to the normally open port of the LHDA release valve.

As shown in figure 4.9, triggering of the valve is initiated in one of three ways: (a) position switch triggering, (b) direct software trigger or (c) comparator output trigger. Position switch triggering occurs in two different events. The first is during initiation, when the pump finds its home position. The release valve is automatically triggered so as to start testing without a preload. The second instance would happen in the unlikely scenario that the pump is allowed to reach its end position. Because the syringe volume is several times larger than a typical ear canal volume this would immediately indicate a severe leakage issue and the valve would open to drain any slight pressure buildup.

The other two potential triggering events would be a direct software trigger made available to the audiologist in the windows computer interface. This allows venting if the audiologist feels that a test should be stopped or repeated, and is controlled via D6. The comparator circuit output shown in figure 4.6 provides the final triggering event. As seen in figure a high and low pressure threshold can be set via POT adjustment. This allows
for very fine adjustment of the testing conditions and pressure ranges the volunteer is allowed to experience. During pressure compensation or immittance screening these same pressure thresholds are monitored and compared with the output from the pressure transducer. The pump is slowed down or stopped before it reaches these programmable thresholds, however this physical layer provides a redundant protection scheme and ensures that testing does not exceed the predetermined range.

Figure 4.9 Patient monitoring and safety circuits including release valve and comparator circuits. Notice that both start and stop switch outputs have switch de-bouncing circuits implemented so as to avoid false valve triggers which could ruin clinical testing.
4.2.5 ADDITIONAL PATIENT SAFETY CONSIDERATIONS

It is our imperative that patient safety remains as our top priority. As such the system's relief valve system was designed to operate the valve in its normally open state. Triggering of the valve to close is accomplished via a high level TTL signal at the base of the power transistor which drives the solenoid valve. If any or all of the safety mechanisms mentioned previously were to fail, power to the system could be quickly interrupted via power switch at the rear of the device, thus immediately restoring the system back to ambient pressure.

It should also be noted that all system specifications including pressure range conform to predicate devices which already have FDA clearance and are registered under the exemption class II audiometer code EWO.

4.3 ACOUSTIC ADMITTANCE SYSTEM CALIBRATION METHODS

Before proper admittance measurements are taken, it must be ensured that the system design is properly calibrated, so that metrics such as ear canal volume, admittance and compliance measurements can be taken accurately, repeatedly and with complete confidence. In order to obtain proper correlations between ear canal volume, sound level and admittance, several base line measurements must be taken so that a relationship can be established. A volumetric test assembly was designed for the calibration of acoustic imittance instrumentation. The main purpose of the assembly is to present several precise cavities with predetermined volumes to a probe with imittance capabilities. The probe should then be calibrated (in software) in such a way that via compliance measurements, it will be able to correctly assess the volume of the cavity in which it is in.

In order to be judged valid, the volume coupler must conform to a specific
standard enforced by the Acoustical Society of America, and was thus designed with certain criteria in mind. During the design of the volumetric tester, standard ANSI S3.39-1987 (ASA 71-1987), Specifications for Instruments to Measure Aural Acoustic Impedance and Admittance, was followed. Specifically section 10.2 provides exact design guidelines and is referenced in table 4.3 below. The standard provides several criteria for the dimensioning of each volume, as well as guidance on which specific volumes can be used and how many volumes should be used in a typical calibration assembly. Additional important information comes in the form of acceptable machining and manufacturing tolerances as well as satisfactory diameter-length ratios.

<table>
<thead>
<tr>
<th>ANSI S3.39-1987 Section #</th>
<th>Description</th>
<th>Criteria Met</th>
</tr>
</thead>
<tbody>
<tr>
<td>10.2.1</td>
<td>At least three calibration cavities shall be provided with the instrument. These three required cavities shall have volumes of: 0.5, 2.0, and 5.0 cm$^3$. Additional volumes if provided shall have volumes chosen from: 1.0, 1.5, 2.5, 3.0, 3.5, 4.0, or 4.5 cm$^3$</td>
<td>Yes</td>
</tr>
<tr>
<td>10.2.2</td>
<td>All calibration cavities shall have hard, non-porous, acoustically rigid surfaces</td>
<td>Yes</td>
</tr>
<tr>
<td>10.2.3</td>
<td>The volume of all calibration cavities shall be accurate to $\pm 2%$ or 0.05 cm$^3$, whichever is greater.</td>
<td>Yes</td>
</tr>
<tr>
<td>10.2.4</td>
<td>For cylindrical calibration cavities, the ratio of the length (depth) of the cavity to its inside diameter shall range between 1 and 3. The draft angle shall not exceed 3°.</td>
<td>Yes</td>
</tr>
<tr>
<td>10.2.5</td>
<td>The cavities and the probe shall be designed in such a way that, when connected, the indicated volume of the cavity shall be obtained, with an air tight fit. The probe tip shall not enter the cylindrical volume of the cavity, and the tip shall not be more than 1 mm above the top circular plane of the cavity.</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Table 4.3 ANSI standards for the design of calibrated volumes (ref. ANSI S3.39-1987)
The volumes which were chosen for the assembly were the mandatory 0.5, 2.0, and 5.0 cc as well as 1.0 cc. Figure 4.10 shows several views of the coupler assembly (note: volumes are by ascending order from left to right).

![Figure 4.10 (a) Front, (b) lateral and isometric views of the V-coupler](image)

A minimum of 0.1” of material thickness was left in between each individual volume cavity in order to provide proper insulation and acoustical rigidity as stipulated in the ANSI standard. The coupler was machined out of 2024Al.

Referring back to the basis of acoustic immittance testing, if a proper tone frequency is chosen, the admittance of the middle ear can be directly proportional, or in the case of a 266Hz tone, equal to the volume from the tip of the probe to the tympanic membrane. Thus, if a method for determining the volume of a cavity could be deduced, than accurate immitance measurements can be obtained. Depending on the volume of a cavity sound intensity either increases (small volumes) or decreases (large volumes). A relationship between recorded receiver output (dBSPL) and cavity volume was easily established by allowing the probe to produce a 266 Hz tone at 70 dBSPL and placing the probe in the four calibrated volumes found in the volumetric assembly. The resulting output (dBSPL) as recorded by the microphone was plotted versus the volume of the cavity (4.11).
Figure 4.11 Linear response between calibrated volumes and their respective recorded responses.

Figure 4.11 also shows the linear relationship which was determined between the decreases in dBSPL output for every volume increase. Equation 4.14 shows the simple linear formula which determines the volume of cavities in response to the intensity perceived by the microphone (i.e. reference intensity equal to 70 dBSPL). This algorithm was programmed so that volume changes could be monitored and ear canal volumes recorded arriving at true tympanometric readings.

\[
Volume_{cc} = \frac{Intensity_{dBSPL} - 72.6}{-3.11}
\]  
(4.14)
4.4 PUMP DESIGN VALIDATION

For the purposes of the design portion of this research, it was desired to validate if our pressure system was actually successful in three main aspects: (1) positive pressure compensation, (2) negative pressure compensation, and (3) acoustic immitance measurements. In order to validate the system's function and integration with the existing DSP acquisition system, several validating tests were conducted including a positively compensated TEOAE screen, a negatively compensated TEOAE screen, and an informal tympanometric scan. These tests were designed exclusively to validate the success of the pressure compensation system design and were not controlled or included within the results discussed in chapter 6. The results of these preliminary tests are covered in the following sections.

4.4.1 FIRST VALIDATED TYMPANOMETRIC RECORDING

Once calibrated, we performed a self-administered tympanometric screening. Because the subject was known to have healthy hearing and had been screened in prior instances with a commercially available tympanometer, the expected TPP was at or very close to 0daPA. This was the baseline to which the accuracy and effectivity of the newly designed system was compared to. Figure 4.12 shows the final test result as recorded on April 1, 2011. The graph shows the relationship between recorded probe intensity and pressure shift from the ambient pressure. The peak is inverted when compared to a standard tympanogram due to the fact that sound level intensity has an inversely proportional relationship with acoustic admittance or ear canal volume which is typically shown on a tympanogram. However figure 4.12 clearly indicates a TPP right at the ambient pressure which is what was expected (shown as the red demarcating line).
Figure 4.12 First validated tympanometric screening result. A TPP at 0daPA is clearly indicated.

The system software could be further developed so that ear canal volumes will be automatically generated based on the correlation shown previously in equation (4.14). This will provide a more standard indication of pressure shifts which must be compensated for during testing.

4.4.2 PRESSURE COMPENSATED TEOAE TEST RESULTS

A normal condition (i.e. ambient pressure) TEOAE was administered twice to serve as a baseline when comparing pressure compensated tests. The first was administered in a quiet room with no additional ambient noise present. The second was administered under the same test conditions with the exception of running the syringe pump continuously on without being connected to the probe system. This was done to determine if pump noise
would have any result in recorded TEOAE levels. The results of these baseline tests can be seen in figure 4.13.

Figure 4.13 Normal (ambient pressure) TEOAE screening results. The result shown on the left is a standard TEOAE without any additional ambient noise. The result on the right is a second TEOAE screening result which was administered while the motor pump was being turned on and off without being connected to the probe. The resulting deviations in TEOAE signal amplitude were negligible.

A positive pressure compensated OAE test was administered. The pressure was raised to a predetermined value as seen by the A/D. This value was then sustained and the OAE was administered. The result of this recording is shown in figure 4.14. This test result proved to be unreliable and should have been repeated. Stimulus artifacts can be seen in the meatal response, thus there is an artificially high level of higher frequency signal amplitudes which were not previously seen in the other recordings. During the volunteer study a test result such as this one would be rejected as it will have very little test-retest value among the high frequencies within sessions performed on the same subject. However, for the purposes of design validation this test proved that a TEOAE session
could be successfully administered while a sustained positive pressure was applied via the syringe pump designed for this research.

![Figure 4.14 Positive pressure TEOAE screening result. The testing was administered to the same volunteer as shown in figure 4.13](image)

A negative pressure compensated OAE test was administered. The pressure was lowered to a predetermined value as seen by the A/D. This value was then sustained and the OAE was administered. The result of this recording is shown in figure 4.15. Once the testing data was collected, TEOAE signal amplitudes were compared to each other in order to determine if similar results as obtained in the literature could be seen. The results of this comparison are given in table 4.4, which clearly indicate significant effects on recorded TEOAE amplitudes throughout the recorded frequency range.
Figure 4.15 Negative pressure TEOAE screening result. The testing was administered to the same volunteer as shown in figure 4.13.

The table 4.4 below shows while there were negligible differences between the two ambient pressure TEOAE tests, there were quantifiable drops in signal amplitude observed for both positively and negatively compensated testing sessions.

Table 4.4 TEOAE signal amplitude results for normal, positive compensated, and negative compensated testing conditions

<table>
<thead>
<tr>
<th>F [Hz]</th>
<th>TEOAE Signal Amplitude [dBSPL]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>1000</td>
<td>3.09</td>
</tr>
<tr>
<td>1500</td>
<td>-2.84</td>
</tr>
<tr>
<td>2000</td>
<td>-3.16</td>
</tr>
<tr>
<td>3000</td>
<td>-9.34</td>
</tr>
<tr>
<td>4000</td>
<td>-15.89</td>
</tr>
</tbody>
</table>
It should be known that the only purpose of these initial recordings was to establish a first successful OAE screening session while compensating for pressure and thus validate the functionality of the syringe pump system. In the end this data will not be included with the data collected during the volunteer study and was only performed to determine the success of the engineered syringe pump system working in tandem with the SmartTrOAE software package.
Chapter 4 of this dissertation focused on the design aspects of a software controlled syringe pump. The pump was capable of administering a constant ear canal pressure set by a software control module which would allow TEOAE screening simultaneously while the pressure gradient was controlled and stabilized. The design showed evidence that such a device could be incorporated in a clinical setting and could in fact contribute to pressure compensated TEOAE screening. In this chapter we now shift our focus to subject testing. An important aspect of this work is the determination of the actual effects of ear canal pressure on the recorded TEOAE. As discussed during the introduction some past studies have been conducted on the effects of pressure on the frequency bands of collected TEOAEs but there is still more to be analyzed and reviewed. Does pressure alter the meatal portion of the TEOAE signal more so than the high frequency components? What are the effects of pressure on the time domain? Does the TEOAE shift as pressure increases within the ear canal? The individual effects of
EAC pressure in both the time domain and frequency domain must be understood so that the benefits of a compensated procedure can be more richly appreciated.

This chapter will focus on the IRB sanctioned volunteer study implemented in 2012 for the sake of TEOAE signal collection under various EAC pressures. Due to the regulations of the IRB, devices without FDA pre-market approval are not allowed to be used on subject volunteers and thus the software controlled syringe pump was replaced with a manual method developed so that the study may be performed. An overview of the experimental design will be presented as well as the test battery performed on every volunteer. The remaining portion of this chapter will focus on signal collection and analysis as we present correlations found during the IRB study.

5.1 PURPOSE OF THE STUDY

The purpose of this study was to collect TEOAE signals from a total of 10 subject volunteers. TEOAE signals were collected from both the right and left ear, thus at the end of the study a sizeable pool of TEOAE signals under a variety of conditions were organized from a total of 20 ears. This study focuses on additional affects not currently found in the existing research. Data was collected and analyzed for the sake of studying the relationship between EAC pressure and the performance of the TEOAE signal in both the frequency and time domains. Beyond that scope the study also set out to segregate the effects of pressure on the meatal portion of the TEOAE and the effect on the higher frequency components (i.e. those typically processed and used for TEOAE screener programs). Of great interest was whether it could be demonstrated that the effect of pressure was polarity insensitive. That is are the TEOAE signal affects for ear canal pressures at +100daPa and -100daPa statistically significant from each other or are both
TEOAE signals affected in largely the same manner. The study provided the opportunity to analyze the effects of multiple ear canal pressures first hand and draw correlations which directly impact the possible interpretation of TEOAE screener results.

5.2 OBTAINING VOLUNTEER CONSENT

As mandated by the University of Miami internal review board, an oral and written explanation of the test procedure was delivered to each volunteer. Within the consent form (see figure 5.1 or the sample consent form in the appendix section) the following major items were addressed in writing and explained to the subject volunteer: purpose, procedure, risks, benefits, alternatives, compensation for injury, confidentiality, right to withdraw, and costs associated with the study.

![Figure 5.1 Three page volunteer consent form as executed during the recruitment of all IRB study volunteers at the University of Miami. The complete document may be found in the appendix section of this dissertation.](image)

Each volunteer was made aware that the purpose of the study was to record OAE signals (i.e. explained as low level acoustic signals emitted by the inner ear) under a variety of pressures. The risk was reported as extremely low due to the fact that the study co-investigator would be constantly monitoring the amount of pressure induced within
the ear canal and that the test battery itself did not call for any pressure outside the range typically experienced during standard tympanometric screening.

The procedure and test battery was then explained to the volunteer in common terminology and the fact that participation was strictly on a volunteer basis and that they could in fact withdraw at any time was also reiterated. The following is an excerpt from the consent form which summarizes the common procedural explanation provided to the volunteer before obtaining their consent to proceed with testing.

A small probe containing a miniature microphone, sound port, and pressure port will be gently inserted into your outer ear canal. With the probe in place, you will hear a clicking sound that will vary from soft to moderately loud. You will also be presented with a range of ear canal pressures during testing which may present minor and temporary discomfort. Though the level or rate of sounds will vary as will the pressure within the ear canal, the effects will never be harmful to your hearing. Testing may take up to one hour. All you will be required to do during this time is lie quietly or preferably sleep. Occasionally we may ask the subject (you) to come back for a second test. The procedures used in the second test will be identical to the first test. This study will use a commercially available, FDA approved hearing screening device called SmartTrOAE. This device will be responsible for both the presentation of sounds and for the recording and acquisition of the feedback those sounds produce within the ear. In addition to this we will also be fixing an incoming pressure line to the probe. This portion of the study is investigational. The pressure within this line will be manually controlled via syringe adjustment by the experimenter and stop cock. Once the pressure has been adjusted to the correct value the stop cock will be activated thus stabilizing the pressure within the line. Pressure regulation will be maintained via U-tube manometer. Pressures applied within the ear canal will be small and well within the scope of standard FDA devices. The U-tube manometer will ensure that the ear canal can never be over or under pressurized.

Once the co-investigator completed the oral explanation of the volunteer consent form the co-investigator required a verbal confirmation that all portions of the consent form were understood by the volunteer. Once verbal confirmation was give the volunteer
was asked to sign their consent and date the document. The co-investigator was also required to sign and date the consent form bearing witness that both written and oral explanations of the study’s scope had been satisfactorily provided to the subject volunteer.

5.3 TEST BATTERY PERFORMED (IRB TEST SUMMARY)

A specific test battery was created prior to volunteer testing. The test battery was designed to acquire a variety of pressure ranges at two distinct gain settings while trying to limit the duration of testing to one hour (which was deemed crucial as volunteer fatigue can be a potential source of testing error). The rationale for collecting all data at two distinct gain settings was due to the fact that our study focuses on the effects of pressure on both the meatal response and the higher frequency TEOAE components. Typical TEOAE screener programs (such as that used during data acquisition) have built in AUTO gain modes which elevate the microphone or post-processing gains to accentuate the higher frequency components of the TEOAE. Because the relative amplitude of the TEOAE is orders of magnitude smaller than those of the meatal response, the meatal portion of the recorded signal clips and thus distorts the time and frequency domain data. For this reason most if not all TEOAE screener programs blank out the meatal portion of the TEOAE signal and focus solely on the later portion of the TEOAE signal. It was hypothesized that a LO gain recording would allow us to collect clean meatal signals (i.e. without clipping artifacts) and would thus allow us to analyze the time domain and most importantly the frequency domain without artificial non-linear effects. A HI gain recording would provide amplification of the high frequency portion of the TEOAE in the case that resolution of the LO gain signal proved to be an issue when
analyzing the signals in post process. This approach was cumbersome but necessary as all of the analysis was performed post-test and the availability of the volunteers for a second round of testing at a later date was not guaranteed.

The image below is an excerpt of the test summary checklist. This checklist was used by the co-investigator during volunteer testing and data collection. It established a form which could track the subjects name and demographic data as well as provide a checklist for the co-investigator as tests were completed. The spaces provided next to each test were provided to indicate the collected file number associated with each test. In addition the reader can see that a code identifier was used for each subject. The code (i.e. S01-S10) was assigned to each participant prior to testing and was later used as the sole subject reference in all data analysis work sheets and statistical packages.

![IRB Test Summary & Checklist](image)

*Figure 5.2 IRB Test Summary & Checklist as used during volunteer studies and data collection*
The test battery shown in Fig. 5.2 explored the pressure range between +200 daPa and -200 daPa. For each ear a baseline TEOAE recording was taken without any additional ear canal pressure (i.e. the pressure system was open and exposed to the room’s ambient pressure). Discrete pressure values of ±50 daPa, ±100 daPa, and ±200 daPa were used during individual recording sessions at the two gain settings previously described. Overall the test battery constituted of 28 individual tests. Each test from start to finish typically lasted 60 seconds to perform (i.e. 1024 sweeps @ 19.3/s stimulus rate). This did not account for stop/restart issues due to the lack of proper sealing at the probe to ear interface. Overall testing duration varied from subject to subject but on average the complete battery lasted approximately 1 hour per individual. The subject was instructed during both the verbal and written consent times that he or she was within their rights to stop or discontinue their participation at any time before, during, or after testing.

### 5.4 TEST PROCEDURE SUMMARY

The purpose of this section is to quickly provide a summary of the test procedure used on each of the 10 volunteers. The itemized list below provides the outlined steps taken with each subject:

1. The subject was given and read a consent form explaining the purpose and nature of the study (including specifics of how testing would be conducted).

2. The subject was placed in a natural, upright sitting position on a comfortable chair located within a sound isolation chamber.

3. The sound isolation chamber was closed and the subject was given a selection of new ear tips to use with the probe tester.

4. The probe (10B+) contains two open tube fittings which are typically used for sound sources. For the experiment, one tube was fitted with an ER3A sound source and the other with a dedicated pressure line.
5. The IRB test summary (discussed in detail in the previous section) was used to conduct testing on the subject. The test battery included TEOAE acquisition in both HI & LO gain modes. Ambient pressure served as the baseline. Pressures of ±50 daPa, ±100 daPa, and ±200 daPa were manually administered and TEOAE signals were collected in both Right and Left ears.

It must be noted that for the sake of continuity all tests were administered continuously and the subject was asked to maintain the same seating position while the probe was attached to the seat behind the subject (i.e. the subject was not asked to hold the probe in their hand). Although these precautions were taken to ensure uniformity and consistency between the various TEOAE recording sessions, it must be noted that several times multiple subjects had to remove and accommodate the probe within their ear canal. Most of these situations were necessary due to a lack of a proper seal around the probe/ear interface and thus the re-accommodating of the probe was initiated by the co-investigator. In other situations the subject simply became weary of the probe within their canal and requested an interlude during testing. This will be discussed more as we explore the results obtained in later sections. Figure 5.3 below shows depictions of procedural steps 2-4.

Figure 5.3 (a) Subject seated in test chair with attached probe assembly (b) probe assembly with integrated pressure line (c) sound isolation chamber used during volunteer testing.
5.5 TEST SETUP SUMMARY

As previously mentioned IRB regulations prohibit the use of non-FDA approved test equipment during an IRB sanctioned test study, thus a manual method was developed to administer a positive and/or negative static pressure within the ear canal. A manual syringe pump controlled by the operator (co-investigator) was constructed according to the schematic shown in Figure 5.4. The operator, via action on the syringe, was in control of the system pressure which was monitored using the calibrated digital manometer. Relief valve 1 was used by the operator to expose the pressure line to ambient (typically before baseline tests and/or after a round of extensive testing). The pressure line was connected to a 10B+ probe which was then fixed to the subject’s chair where all testing was conducted. An additional venting control was given to the subject in case they felt any discomfort during testing. Subjects were also instructed that they could, at any time, remove the probe from their ear in case of extended discomfort.

![Figure 5.4 Schematic representation of the manual pump system used during the IRB study](image-url)
Commercially available test equipment (i.e. IHS SmartTrOAE software module used in combination with a SmartUSB system) was used with a 10B+ probe to present the auditory stimulus and record the TEOAE signals from the volunteer. All data was collected, sorted and then analyzed post-test.
This chapter will focus exclusively on the time domain and frequency domain analysis of the collected meatal responses and TEOAE signals. Individual signal collections will be presented as well as mathematically averaged responses which are derived from the responses of all valid subject ears. We present the effects of pressure on both the meatal response as well as on the TEOAE as it pertains to the various differences from the baseline response within frequency bands (as publicized in prior articles). We also review modifications to the signals morphology in the time domain which has not been previously covered within the literature. Statistical significance of the results is also illustrated and discussed.

6.1 INITIAL REVIEW OF SUBJECT SCREENER RESULTS

Prior to data analysis, all baseline TEOAE screener results were reviewed for every volunteer. The purpose of this initial screening was to segregate potential outlying test results before the data was analyzed in further detail. Results were removed if the
subject tested with poor baseline TEOAE results. These poor results typically exhibited mid to hi frequency components submerged within the noise floor. These low amplitude TEOAE results would provide limited insight into the affect that pressure variation has within the ear canal.

The other main source of exclusion from the study was on subjects that had great difficulty in retaining a seal at the probe-canal interface. These cases led to multiple test-retest scenarios as the probe was removed from the ear and placed again repeatedly during the entire test battery. These results were excluded from the analysis as it was believed that multiple variations of probe placement could eventually degrade the significance of the affects seen exclusively from the variation of EAC pressure. The table below shows the entire pool of volunteers and their exclusion/inclusion status. Right and left ear test results were treated independently although for most subjects, poor performance in one ear typically indicated poor performance in the other. This was true for both probe sealing issues and poor TEOAE performance.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Right</th>
<th>Left</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>S01</td>
<td>HEALTHY</td>
<td>HEALTHY</td>
<td>Normal</td>
</tr>
<tr>
<td>S02</td>
<td>HEALTHY</td>
<td>HEALTHY</td>
<td>Border line refer results for baseline in the Right Ear (weak HF)</td>
</tr>
<tr>
<td>S03</td>
<td>POOR</td>
<td>POOR</td>
<td>Perforated TM on (L), poor TEOAE baseline results (special case)</td>
</tr>
<tr>
<td>S04</td>
<td>POOR</td>
<td>HEALTHY</td>
<td>Right ear LO gain collection contained clipped meatal results</td>
</tr>
<tr>
<td>S05</td>
<td>HEALTHY</td>
<td>HEALTHY</td>
<td>Border line refer results in both ears (weak HF)</td>
</tr>
<tr>
<td>S06</td>
<td>POOR</td>
<td>POOR</td>
<td>Subject had poor TEOAE recordings and difficulties retaining a seal</td>
</tr>
<tr>
<td>S07</td>
<td>HEALTHY</td>
<td>HEALTHY</td>
<td>Very large TEOAEs, subject did not adjust their probe during testing</td>
</tr>
<tr>
<td>S08</td>
<td>POOR</td>
<td>POOR</td>
<td>Subject had several probe fit issues, difficulties retaining a seal</td>
</tr>
<tr>
<td>S09</td>
<td>HEALTHY</td>
<td>HEALTHY</td>
<td>Trouble retaining +200daPa seal (CI had to compensate quickly)</td>
</tr>
<tr>
<td>S10</td>
<td>POOR</td>
<td>POOR</td>
<td>Subject has poor TEOAE results in the (R) &amp; Clipped meatal in (L)</td>
</tr>
</tbody>
</table>

Table 6.1 Subject inclusion/exclusion status for all 10 volunteers: HEALTHY status indicates that the data was included in the analysis portion while a POOR rating indicates the exclusion of the test results from the data analysis.
6.2 EXTRACTING THE RAW TEOAE SIGNAL

As previously mentioned all TEOAE recordings were obtained using the IHS SmartTrOAE system. Due to the fact that this system is a commercially available clinical screening tool there are many filters applied in post processing to ensure the collected signals are sorted in a specific manner to where the software can make a quantifiable decision with regards to the PASS/REFER rating it provides the clinician. One of the techniques the software employs is the predetermined “blanking” of the meatal response so that additional amplifying filters can be applied to the higher frequency components of the TEOAE without distorting the FFT conversion of the signal (which is displayed in real time and during review). This is important for the clinician but for the purposes of this study it was crucial that all analysis be performed from the RAW microphone output so that the meatal portion could be extracted and separated from the higher frequency components and analyzed individually. Fortunately the SmartTrOAE filters are not destructive and the raw microphone output is kept intact within the data file. Figure 6.1 illustrates the extraction process. Once the RAW A/D values were extracted from the data file (via export to text functionality provided by the software suite), the values were placed in excel and converted from the A/D values to an actual voltage level. Conversion factors were based on the resolution of the A/D as well as the rail to rail operating voltage the unit performs at. The FFT of this entire signal (meatal response + TEOAE) was then taken to obtain the frequency content for use during meatal analysis. The same process was implemented when analyzing just the TEOAE portion of the signal with the additional step of blanking the first 2ms of audio prior to taking the FFT. This effectively
removed the large meatal effects from the lower amplitude, higher frequency TEOAE components.

Figure 6.1 Representation of raw MEATAL and TEOAE signal preparation from the original

The raw signals described above were then used to perform the data analysis we will cover in the upcoming sections. It is important to note that for the sake of comparison all signals were collected and compared using the same gain level applied during initial collection.

6.3 MEATAL FREQUENCY DOMAIN ANALYSIS

For meatal signal recordings, frequency analysis was performed not on the direct FFT signal derived from the original microphone output, but on frequency bins or frequency bands predetermined and used for all recordings. Frequency band analysis was then used to derive a simplified (and summarized) graphical depiction of the average band values associated with the subject’s signal spectrum. These bands, and the center frequency they represent, were selected due to their appeal in the clinical setting. The benefit of this method is twofold. First, frequency bands introduce averaging when
comparing spectra together. Averaging within bands introduces stability and repeatability and helps to mitigate intra-test variations subjects can produce during recordings (i.e. swallowing, breathing, probe removal/reinsertion, etc.). Secondly, the resulting graphical depiction greatly reduces the complexity of the test basis and facilitates the use of statistical measures when trying to establish causal effects. Figure 6.2 illustrates the frequency bands selected along with the center frequency they represent.

![Figure 6.2 Frequency bands and center frequencies used for individual and averaged bin analysis](image)

**Figure 6.2** Frequency bands and center frequencies used for individual and averaged bin analysis

### 6.3.1 INDIVIDUAL SUBJECT FREQUENCY BIN ANALYSIS (MEATAL)

Frequency band plots were derived for each ear. In addition to the averaged frequency content, plots were separated into three distinct areas representing ±50 daPa, ±100 daPa and ±200 daPa. Error bars representing the minimum and maximum values within the specific band were shown to provide context around the averaged value representing the center frequency. It should be noted that what we see depicted in the following plots are not the amplitudes of the subject FFT, but rather the normalized
response based on the subjects baseline. Each subplot also contains a P-stat and correlation coefficient so that statistical significance as calculated between the two plotted series can be quickly reviewed. Figure 6.3 shows a typical frequency bin plot.

![Figure 6.3 Typical meatal frequency bin plot as used during bin analysis. The error bars signify the maximum and minimum values within the specified band. P-stat and correlation coefficients provide a statistical representation of the comparison between trends the pressure values shown above.](image)

As previously mentioned the responses below are all normalized to their respective baseline. For all subjects the pressurized FFT was subtracted from the baseline (non-pressurized) FFT. This resulted in a difference or DIFF. The impact of pressure on the meatal response could then be compared relatively easily as a fluctuation from the baseline response. This also allowed us to achieve an overall averaged response based on
the total 11 ears reviewed. Figures 6.4 and 6.5 provide views of all 11 individual frequency bin plots.

Figure 6.4 Meatal frequency bin plots for ears S01(R), S01(L), S02(R), S02 (L) and S04(L)
Figure 6.5 Meatal frequency bin plots for ears S05(R), S05(L), S07(R), S07 (L), S09(R), and S09(L)

One of the first things most noticeable and consistent among the plots is the fact that overall trending (i.e. the shape of the bin curve) remains (in most cases) consistent regardless of the polarity of the static ear canal pressure. Another striking feature is that
although the general trending remains similar, in most cases the negative pressure polarity contains content as high as 10dB higher than its positive polarity counterpart. Phrased in a slightly different way, overall frequency bin plot shapes are pressure polarity independent, however there seems to be a statistically significant difference between positive and negative pressure polarity and their effects on the meatal response.

### 6.3.2 AVERAGED FREQUENCY BIN ANALYSIS (MEATAL)

Comparing the individual bin plots from subject to subject indicated a pattern which could be further reinforced by obtaining an “average” response for all ears. This average response derived from 11 ears would thus help further reinforce the trends we could perceive while reviewing the individual plots. Figure 6.6 shows the resulting averaged response. The individual pressure and frequency specific bins for all 11 ears were averaged together to arrive at a new averaged response. The minimum and maximum values were obtained using the same data ranges and were displayed as error bars to signify the variability between subjects for any particular frequency band. Again, P-stat and correlation coefficients were used to demonstrate both similarities and differences between opposing static pressures plots. In addition to this the averaged plots contain P-stat criteria for each frequency band. That is, the effects of pressure polarity were tested for statistically significant differences at each major frequency band.

The averaged plots re-enforced what was evident in most of the individual frequency bin plots. While the trends for each plot were highly correlated and were not affected by the polarity of the static pressure, the values of all negatively pressurized meatal responses were typically 3-5dB greater in most cases resulting in a statistically significant difference between meatal responses collected under positive pressure and
those under the directly opposing negative pressure (i.e. ±50daPa, ±100daPa, and ±200daPa).

![Image of a graph showing frequency band difference with respect to baseline recording, all subjects averaged. The graph includes a legend indicating statistical significance with asterisks.](image)

**Figure 6.6** Averaged mental response frequency bin plots (N=11)

### 6.3.3 MEATAL P-STAT COLOR MAP

The averaged frequency bin plots in figure 6.6 provide statistical relevance to equal and opposing static pressures however they do not compare the effect between all static pressure values within the range tested. Is there a statistically significant difference between the frequency bin values at +50daPa as opposed to +200daPa or -200daPa? Exploring this question amplifies our understanding of static pressure’s effect on the mental response.

Figure 6.7 shows a three dimensional color map of all P-stat values collected in the analysis matrix below. All T-Tests were conducted on the averaged bin responses.

<table>
<thead>
<tr>
<th></th>
<th>+200daPa</th>
<th>+100daPa</th>
<th>+50daPa</th>
<th>-50daPa</th>
<th>-100daPa</th>
<th>-200daPa</th>
</tr>
</thead>
<tbody>
<tr>
<td>-200daPa</td>
<td>0.006123521</td>
<td>2.26821E-05</td>
<td>1.38386E-07</td>
<td>3.19955E-06</td>
<td>0.000962689</td>
<td>1</td>
</tr>
<tr>
<td>-100daPa</td>
<td>0.116618409</td>
<td>0.000315435</td>
<td>3.14245E-06</td>
<td>0.000100914</td>
<td>1</td>
<td>0.000962689</td>
</tr>
<tr>
<td>-50daPa</td>
<td>0.474541084</td>
<td>0.015126996</td>
<td>0.000557945</td>
<td>1</td>
<td>0.000100914</td>
<td>3.19955E-06</td>
</tr>
<tr>
<td>+50daPa</td>
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<td>0.642438817</td>
<td>1</td>
<td>0.000557945</td>
<td>3.14245E-06</td>
<td>1.38386E-07</td>
</tr>
<tr>
<td>+100daPa</td>
<td>0.022727814</td>
<td>1</td>
<td>0.642438817</td>
<td>0.015126996</td>
<td>0.000315435</td>
<td>2.26821E-05</td>
</tr>
<tr>
<td>+200daPa</td>
<td>1</td>
<td>0.022727814</td>
<td>0.010440054</td>
<td>0.474541084</td>
<td>0.116618409</td>
<td>0.006123521</td>
</tr>
</tbody>
</table>

**Table 6.2** Mental P-Stat cross matrix: T-Tests performed on the averaged frequency bin data P-stat < 0.05 (statistically significant difference between mental responses @ relative static EAC pressure)
6.4 MEATAL TIME DOMAIN ANALYSIS

The second aspect of our analysis centers around the effects of pressure on the meatal response in the time domain. Not a great deal has been written in the past concerning meatal response time domain effects but our study has yielded very compelling results which we will illustrate in the following sections.

Similar to the frequency bin analysis performed in earlier sections of this chapter, subject responses were grouped into positive/negative static pressure pairings. This was done so that a distinction between polarity dependent and polarity independent affects could be witnessed. Once the meatal responses were plotted, the time and amplitude
values of the response’s two most prominent features (i.e. the dip and the peak) were recorded. These values were then plotted against the static pressure they were collected at. The following sections showcase both the individual subject results as well as the averaged result.

6.4.1 INDIVIDUAL AMPLITUDE & PHASE SHIFT ANALYSIS (MEATAL)

Time versus amplitude plots were created for each ear, strictly focusing on the meatal portion of the collected signal (i.e. 0 – 2ms). Meatal responses were separated into three distinct groups representing \( \pm 50\text{daPa} \), \( \pm 100\text{daPa} \) and \( \pm 200\text{daPa} \). The subject’s baseline response was included in each group for a visual indication of how pressure was affecting the subject’s meatal signature. To further enhance the time domain analysis, time and amplitude information for all major dips and peaks were extracted and plotted separately on dual axis plots so that amplitude and phase shift effects could be reviewed.

![Figure 6.8 Individual subject meatal responses (\( \pm 50\text{daPa}, \pm 100\text{daPa} \) and \( \pm 200\text{daPa} \)) with accompanying dip and peak dual axis plot]
The interpretation of both graphs together becomes very telling. In the example provided in Figure 6.8 it is evident by inspection of the time domain plots that the following effects are being demonstrated: (1) consistent amplitude elevation of the meatal response at the negative static pressure value (2) increase in relative peak delay which seems to be pressure polarity insensitive yet very linear as the absolute value of the EAC pressure increases (3) very minor change to the meatal dip although as with the peak, negative polarity induced a larger amplitude. These effects are again represented in the dual axis plot however their values are now quantifiable and extracted for averaging among subjects collected. The overall dip and peak amplitudes in Figure 6.8 are represented by the column chart and use the axis on the left side of the plot. Time shifts from the original baseline response are represented as a scatter line plot and measured with the axis on the right side of the plot. As was evident during inspection, the relative shift from the original baseline recording increases linearly as the absolute value of the EAC pressure increases in this particular subject.

Among all subjects the relationships above seemed to repeat over and over. The amplitudes of the meatal responses collected in the negative pressure polarity were either the same amplitude as those obtained in the positive polarity or were noticeably larger. Overall positive phase shifts (signal lag) tended to increase linearly with elevated pressures within the ear canal regardless of the polarity of that pressure. It was also noted that in most cases even though the overall phase shift increased, lag was typically greater for meatal responses collected with positive ear canal pressure than the negative counterpart. Overall all subjects either demonstrated an amplitude increase in the major peak, a phase shift or both as the absolute value of the EAC pressure increased. Figure
6.9 and figure 6.10 showcases the meatal responses of all 11 collected ears while figure 6.11 and figure 6.12 illustrate the subjects’ extracted dip and peak dual axis plots.

Figure 6.9 Meatal responses from ears S01(R), S01(L), S02(R), S02 (L) and S04(L)
Figure 6.10 Meatal responses from ears S05(R), S05(L), S07(R), S07 (L), S09(R), and S09(L)
Figure 6.11: Metal Dip/Peak Amplitude & Phase Shift Plots from ears S01(R), S01(L), S02(R), S02(L) and S04(L) (amplitudes for both dips and peaks are plotted in columns while phase shifts for both dips and peaks are represented in the scatter line plots.)
Figure 6.12 Meatal Dip/Peak Amplitude & Phase Shift Plots from ears S05(R), S05(L), S07(R), S07(L), S09(R), and S09(L) (amplitudes for both dips and peaks are plotted in columns while phase shifts for both dips and peaks are represented in the scatter line plots)
6.4.2 AVERAGED AMPLITUDE & PHASE SHIFT ANALYSIS (MEATAL)

Although the overall morphology changes as seen in section 6.5.1 are compelling, of most interest in this study are the quantifiable effects on amplitude and signal phase in the time domain. From this information we can begin to determine the effects of pressure on the meatal response. The extracted dip and peak amplitude and phase shift metrics were averaged for all 11 meatal responses according to the static pressure in which they were collected. The resulting average plot is seen below in figure 6.13.

![Figure 6.13 Averaged (N=11) Meatal Dip/Peak Amplitude & Phase Shift Plot](image)

From figure 6.13 several insights into the effect of pressure on the meatal response can be clearly seen. On average there is very little or significant change to the amplitude of the meatal dip, however positive pressures on a whole create slightly shallower responses. The inverse can be said on the performance of peak amplitudes. On average as the absolute value of EAC pressure increases so does the amplitude of the meatal response. However it is evident that responses elicited under negative pressure were larger amplitude than those elicited under the same valued positive pressure. With regards to phase shifts, pressure does not seem to have any significant effect on the
relative position on the dip. However as noted previously, there is a direct relationship
with meatal signal lag (i.e. positive phase shift) and the increase of EAC pressure
regardless of its polarity. This relationship is clearly indicated in the averaged plot shown
in figure 6.13. In addition it’s important to note that while the overall phase shift
increases as the absolute value of pressure increases, phase shifts for meatal responses
collected at positive pressures were on average slower than those of the negative
counterpart. This discrepancy in phase shifts grew as the absolute value of the EAC
pressure increased.

6.5 TEOAE FREQUENCY DOMAIN ANALYSIS

Due to the fact that the amplitude levels for the LO gain recordings were obtained
above the noise floor and exhibited well defined high frequency content, we were able to
use the same recordings for the 11 subjects previously tested thus the HI gain recordings
were not chosen for data analysis. In order to remove the influence of the meatal response
from the more delicate TEOAE signal, the time period of 0-2ms was blanked out with 0’s
before the signal FFTs were taken. As in the case of the meatal responses, frequency
analysis was performed not on the direct FFT signal, but on the frequency bands
previously explained in section 6.4. The same type of simplified graphical depiction of
the average band values associated with the subject’s signal spectrum was obtained for all
TEOAE signals.

6.5.1 INDIVIDUAL SUBJECT FREQUENCY BIN ANALYSIS (TEOAE)

As was the case for the meatal responses, frequency band plots were derived for
each ear. In addition to the averaged frequency content, plots were separated into three
distinct areas representing ±50daPa, ±100daPa and ±200daPa. Error bars representing the
minimum and maximum values within the specific band were shown to provide context around the averaged value representing the center frequency. As previously stated the frequency content depicted is not the raw FFT of the signal but rather the normalized response based on the subjects baseline. Each subplot contains a P-stat and correlation coefficient. Figures 6.14 and 6.15 provide views of all 11 TEOAE frequency band plots.

Figure 6.14 TEOAE frequency bin plots for ears S01(R), S01(L), S02(R), S02 (L) and S04(L)
Figure 6.15 TEOAE frequency bin plots for ears S05(R), S05(L), S07(R), S07 (L), S09(R), and S09(L)
Of the things most noticeable and consistent among the plots is the fact that unlike the meatal responses, increased ear canal pressure seemed to have less of a dramatic effect on the frequency bands of the TEOAE signal. Changes from baseline tend to be measured between 1-5dB as opposed to the larger swings seen from the meatal responses. Overall the signals maintain their high correlations with each other although as the P-stat metrics indicate, there seems to be no real statistically significant difference between the effects on the TEOAE recordings based on the polarity of the EAC static pressure.

6.5.2 AVERAGED FREQUENCY BIN ANALYSIS (TEOAE)

As with the meatal response an average of all 11 TEOAE signals was used to further reinforce the overall effect of pressure on the TEOAE. Figure 6.16 shows the resulting averaged response. The minimum and maximum values reflect the min and max subject responses within that particular frequency band and were displayed as error bars to signify the variability between subjects for any particular frequency band. Again, P-stat and correlation coefficients were used to demonstrate both similarities and differences between opposing static pressures plots. In addition to this the averaged plots contain P-stat criteria for each frequency band. That is, the effects of pressure polarity were tested for statistically significant differences at each major frequency band.

The averaged plots re-enforced what was evident in most of the individual frequency bin plots. Unlike the meatal response the effect of pressure was not as large; however one does have to take into account the originating signals small amplitudes. Overall there were no statistically significant differences between the effect of +/- P on the TEOAE amplitude, thus indicating that the effect of EAC pressure on the TEOAE
amplitude is polarity indifferent as indicated by the evenly distributed and matched MIN/ MAX values (i.e. error bars).

![Image of TEOE Frequency Band Difference With Respect to the Baseline Recording: All Subjects Averaged](image)

**Figure 6.16 Averaged TEOAE response frequency bin plots (N=11)**

### 6.5.3 TEOAE P-STAT COLOR MAP

The averaged frequency bin plots in figure 6.16 provide statistical relevance to equal and opposing static pressures however they do not compare the effect between all static pressure values within the range tested. As with the meatal responses a P-stat color map was developed to ascertain the statistical significance between averaged TEOAE frequency band values obtained and compared among all of the static pressures tested.

Figure 6.17 shows a three dimensional color map of all P-stat values collected in the analysis matrix below. All T-Tests were conducted on the averaged bin responses shown in figure 6.16.

<table>
<thead>
<tr>
<th></th>
<th>+200daPa</th>
<th>+100daPa</th>
<th>+50daPa</th>
<th>-50daPa</th>
<th>-100daPa</th>
<th>-200daPa</th>
</tr>
</thead>
<tbody>
<tr>
<td>-200daPa</td>
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<td>0.118520661</td>
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<td>0.159750185</td>
<td>0.363785248</td>
<td>0.363785248</td>
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<td>-100daPa</td>
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<td>0.331788257</td>
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<td>1</td>
<td>0.363785248</td>
</tr>
<tr>
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<td>0.615865384</td>
<td>0.032627058</td>
<td>1</td>
<td>0.469200757</td>
<td>0.159750185</td>
</tr>
<tr>
<td>+50daPa</td>
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<td>0.253726951</td>
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<td>0.032627058</td>
<td>0.039986032</td>
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</tr>
<tr>
<td>+100daPa</td>
<td>0.726781959</td>
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<td>+200daPa</td>
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<td>0.424981365</td>
<td>0.242162477</td>
<td>0.090070534</td>
</tr>
</tbody>
</table>

**Table 6.3 TEOAE P-Stat cross matrix: T-Tests were performed on the averaged bin data P-stat < 0.05 (statistically significant difference between TEOAE responses @ relative static EAC pressure)**
Figure 6.17 TEOAE difference P-Stat color map: the graph depicts regions of statistically significant differences between TEOAE responses at various static EAC pressures (refer to Table 6.2).

For the TEOAE signals a further step in analysis was taken. Due to the fact that we were not able to find as compelling results or statistically significant variations within the various static pressures, the next logical step was to question if statistically significant differences could be found between the raw baseline FFT and the various other TEOAE FFTs collected at the various test pressures. The resulting RAW TEOAE FFT P-stat color map is presented in figure 6.18. As shown in the color map the most significant differences were relegated to the extreme pressure differences (i.e. between +200daPa and -200daPa).
Figure 6.18 RAW TEOAE FFT P-Stat color map: the graph depicts regions of statistically significant differences between TEOAE responses at various static EAC pressures.

6.6 TEOAE TIME DOMAIN ANALYSIS

In the following sections we will be focusing on the effects of pressure on the TEOAE response in the time domain. Again, not a great deal has been written in the past concerning TEOAE response time domain effects but we will be reviewing the effects on both amplitude and phase shifts for the 11 ears collected. Again subject responses were grouped into positive/negative static pressure pairings. This was done so that a distinction between polarity dependent and polarity independent affects could be witnessed. Once
the TEOAE responses were plotted, the time and amplitude values of three prominent and repeatable peaks were recorded. These values were then plotted against the static pressure they were collected at. The following sections showcase both the individual subject results as well as the averaged result.

6.6.1 INDIVIDUAL AMPLITUDE & PHASE SHIFT ANALYSIS (TEOAE)

Time versus amplitude plots were created for each ear, strictly focusing on the TEOAE portion of the collected signal (i.e. 2.25 – 22.25ms). TEOAE responses were separated into three distinct groups representing ±50daPa, ±100daPa and ±200daPa. The subject’s baseline response was included in each group for a visual indication of how pressure was affecting the subject’s TEOAE signature. In addition to the time domain plots dual axis plots tracking amplitude and phase shift metrics were once again implemented. Figure 6.19 provides an example.

![Figure 6.19 Individual subject TEOAE responses (±50daPa, ±100daPa and ±200daPa) with accompanying dip and peak dual axis plot](image)
In the example provided in figure 6.19 it is evident by inspection of the time domain plots that the following effects are being demonstrated: (1) consistent amplitude degradation of the TEOAE response (i.e. all peaks measured) as the absolute value of EAC pressure increases (2) increase in relative peak speed which seems to be pressure polarity insensitive yet very linear as the absolute value of the EAC pressure increases. These effects are again represented in the dual axis plot however their values are now quantifiable and extracted for averaging among subjects collected. The overall peak amplitudes in Figure 6.19 are represented by the column chart and use the axis on the left side of the plot. Time shifts from the original baseline response are represented as a scatter line plot and measured with the axis on the right side of the plot. The arrow indicators are used as a convenient method of identifying which peaks we are reviewing for each TEOAE signal. As was evident during inspection, the relative negative shift (i.e. signal speed increases) from the original baseline recording decreases linearly as the absolute value of the EAC pressure increases in this particular subject.

Among all subjects the relationships above seemed to repeat over and over. TEOAE signal amplitudes by in large decreased as the absolute value of EAC pressure increased. Overall negative phase shifts (signal speed) tended to decrease linearly with elevated pressures within the ear canal regardless of the polarity of that pressure. It was also noted that in most cases even though the overall phase shift increased, negative shifts were typically greater for TEOAE responses collected with positive ear canal pressure than the negative counterpart. Overall all subjects either demonstrated an amplitude decrease in at least one peak, a phase shift or both as the absolute value of the EAC pressure increased. Figure 6.20 and figure 6.21 showcases the TEOAE responses of all 11
collected ears while figure 6.22 and figure 6.23 illustrate the subjects’ extracted peak amplitude and phase shift plots.

Figure 6.20 TEOAE responses from ears S01(R), S01(L), S02(R), S02 (L) and S04(L)
Figure 6.21 TEOAE responses from ears S05(R), S05(L), S07(R), S07 (L), S09(R), and S09(L)
Figure 6.22 TEOAE Peak Amplitude & Phase Shift Plots from ears S01(R), S01(L), S02(R), S02 (L) and S04(L) (amplitudes for all peaks are plotted in columns while phase shifts for peaks are represented in the scatter line plots)
Figure 6.23 TEOAE Peak Amplitude & Phase Shift Plots from ears S05(R), S05(L), S07(R), S07 (L), S09(R), and S09(L) (amplitudes for all peaks are plotted in columns while phase shifts for peaks are represented in the scatter line plots)
6.6.2 AVERAGED AMPLITUDE & PHASE SHIFT ANALYSIS (TEOAE)

As with the meatal responses, TEOAE peak amplitude and time shift metrics were collected for the purpose of quantifying a linear relationship existing between either of these metrics and the variation of pressure within the ear canal. From this information we can begin to make some initial determinations regarding the effects of pressure on the TEOAE response. The extracted peak amplitude and phase shift metrics were averaged for all 11 TEOAE responses according to the static pressure in which they were collected. The resulting average plot is seen below in figure 6.24.

From figure 6.24 several insights into the effect of pressure on the TEOAE response can be clearly seen. On average TEOAE amplitude is increasingly degraded as the absolute value of EAC pressure is increased, positive pressures tend to slightly decrease TEOAE amplitudes more; however on average the effect is essentially independent of the pressure polarity.

With regards to phase shifts, a very interesting relationship was discovered. Much like the meatal response, there is a direct relationship with TEOAE phase shift and the
increase of the absolute value of EAC pressure. This relationship is clearly indicated in
the averaged plot shown in figure 6.24. Much like the plot discussed in figure 6.12, there
exists a linear relationship between signal phase shift and increased pressure. Unlike the
meatal response this linear effect induces a negative phase shift when compared to the
baseline TEOAE signal. In addition it’s important to note that while the overall phase
shift decreases as the absolute value of pressure increases, phase shifts for TEOAE
responses collected at positive pressures were on average faster than those of the negative
counterpart. This discrepancy in phase shifts grew as the absolute value of the EAC
pressure increased.
Due to the fact that this study was segregated into two facets (i.e. design proposal and research investigation) it’s only fitting to bring conclusion to both areas of the study individually. This chapter is thus separated into two sections each covering a summary of the findings and results of the design and research aspects of the study.

**7.1 FINDINGS: PRESSURE COMPENSATING SYRINGE PUMP**

Although the IRB limitation regarding the use of non-FDA approved test equipment precluded the use of the prototype syringe pump during the volunteer research study, several facets of the design were tested and validated. The syringe pump was used successfully to generate both positive and negative pressures within the ear canal and a software module was created in which TEOAE signals could be collected while simultaneously generating the EAC static pressure. Although only a prototype, the early success of the device provides insight that such a device is commercially viable and could be easily integrated with some modification into a test module in clinical software packages such as SmartTrOAE. The addition of more complicated software functions
such as automatic tympanogram screening to set optimal pressure thresholds which would be automatically used during TEOAE screening of the same subject are completely viable and would be the next logical step from the more basic prototype constructed for the purposes of this dissertation.

7.2 FINDINGS: ANALYSIS OF PRESSURE COMPENSATED TEOAES

Our testing of 11 distinct ears yielded results in four separate areas of analysis: (a) frequency domain analysis of the meatal response, (b) time domain analysis of the meatal response, (c) frequency analysis of the TEOAE responses (with eliminated meatal component) and (d) time domain analysis of the TEOAE response (also with the eliminated meatal component).

Averaged meatal frequency bin analysis yielded amplitude shifts from -1.92 to 0.5dB for +50daPa as compared to the original averaged baseline response. In comparison responses collected at -50daPa contained 0.76 to 1.83dB amplitude increases. The negative pressure polarity seemed to amplify the signal response while the opposite positive pressure seemed to decrease the response. Reviewing the results in chapter 6 we can see this trend continue as the absolute value of the static pressure increased. Amplitude shifts of -2.71 to 0.67dB were seen at +100daPa while the meatal response was amplified by 2.34 to 4.02dB at -100daPa. Similarly at +200daPa we see shifts of -0.67 to 4.12dB while amplitude increases of 3.94 to 6.06dB across the spectrum can be seen for -200daPa. As discussed in chapter 6, P-stat values indicate a statistically significant, non-zero difference between the meatal responses collected in positive pressure versus those collected in negative pressure thus indicating that the effect on the meatal response is pressure polarity sensitive. Upon introspection this correlates with
what we know about the external ear and the tympanic membrane. As the absolute value of static pressure increases the tympanic membrane stiffens and thus begins to reflect more energy and transmit less due to restrictions in the vibration of the membrane. Thus as pressure increase and reflected energy increase the meatal response as collected by the microphone increases in amplitude.

When analyzing the time domain effects of pressure on the meatal response, we can see from the results obtained that although the relative position of the dip remains approximately constant, there is a slight amplitude decrease for negative static pressures when compared to their positive counterparts. Also it was determined that on average, as the absolute value of EAC pressure increased a steady increase of positive peak phase shift could be observed. This included slightly larger peak delays for positive pressures. The delta between positive and negative pressure induced phase shifts increased as the absolute value of the EAC pressure increased.

In reviewing the results obtained for our TEOAE analysis we can determine similar yet opposite results for the frequency bins reviewed. Averaged TEOAE frequency bin analysis yielded amplitude shifts from 0.06 to 1.23dB for +50daPa as compared to the original averaged baseline response. In comparison responses collected at -50daPa contained -0.04 to 0.77dB amplitude increases. The positive pressure polarity seemed to amplify the signal response while the opposite negative pressure seemed to decrease the response or not amplify as much as the positive pressure signals. Reviewing the results in chapter 6 we can see this trend continue as the absolute value of the static pressure increased. Amplitude shifts of -0.14 to 1.52dB were seen at +100daPa while the meatal response was amplified by -0.49 to 1.21dB at -100daPa. Similarly at +200daPa we see
shifts of -0.17 to 2.04dB while amplitude increases of -1.3 to 1.37dB across the spectrum can be seen for -200daPa. P-stat values indicate that the results were not statistically significant thus pointing to the result that effects on TEOAE amplitude are not sensitive to the polarity of pressure induced within the canal, but certainly as the absolute value of pressure increases within the canal so does the amplitude attenuation of the TEOAE signal. The exception was consistent amplification throughout the spectrum at +50daPa. This correlates with the knowledge that most subjects averaged a reconstructed TPP shift at approximately +50daPa. The data leads us to believe that the TEOAE signal is potentially being optimized (on average) at +50daPa where the tympanic membrane is allowed to vibrate the most and thus transmit the cochlear signals the greatest. This theory is reinforced as we see larger attenuations for individual frequency bin plots at higher absolute pressures.

When reviewing the results for the time domain analysis of the TEOAE signals it is clearly indicated that on average TEOAE peak amplitudes decreases as the absolute value of EAC pressure increases. This effect is for the most part polarity indifferent. However we also see a very interesting trend develop with regards to phase shifts. On average increases in negative peak shifts occur as the absolute value of pressure increases. Positive pressures had a noticeable and repeatable difference (i.e. larger negative shifts) when compared to their negative counterparts.
REFERENCES


APPENDIX
(A) Pressure Compensating Syringe Pump Schematic
INFORMED CONSENT
Adults

PROJECT TITLE: Recording of Otoacoustic Emissions with Outer Ear Pressure Compensation

PRINCIPAL INVESTIGATOR: Jorge Bohorquez, Ph.D.

PURPOSE:
You are asked to participate in a research study regarding otoacoustic emissions (OAEs). OAEs are faint sounds produced by the ears of many people, either spontaneously or when they hear certain sounds. This study will focus on the effects of ear canal pressure on the recorded OAEs. These responses are measured by inserting a small probe containing a miniature microphone into the outer ear canal in a manner similar to that used to fit a hearing aid. The responses are obtained by computer processing of the microphone measurements.

PROCEDURE:
A small probe containing a miniature microphone, sound port, and pressure port will be gently inserted into your outer ear canal. With the probe in place, you will hear a clicking sound that will vary from soft to moderately loud. You will also be presented with a range of ear canal pressures during testing which may present minor and temporary discomfort. Though the level or rate of sounds will vary as will the pressure within the ear canal, the effects will never be harmful to your hearing. Testing may take up to one hour. All you will be required to do during this time is lie quietly or preferably sleep. Occasionally we may ask the subject (you) to come back for a second test. The procedures used in the second test will be identical to the first test.

This study will use a commercially available, FDA approved hearing screening device called SmartTmOAE. This device will be responsible for both the presentation of sounds and for the recording and acquisition of the feedback those sounds produce within the ear.

In addition to this we will also be fixing an incoming pressure line to the probe. This portion of the study is investigational. The pressure within this line will be manually controlled via syringe adjustment by the experimenter and stop cock. Once the pressure has been adjusted to the correct value the stop cock will be activated thus stabilizing the pressure within the line. Pressure regulation will be maintained via U-tube manometer. Pressures applied within the ear canal will be small and well within the scope of standard FDA devices. The U-tube manometer will ensure that the ear canal can never be over or under pressurized.

RISKS:
The risks of recording pressure compensated otoacoustic emissions are minor. Discomfort from the small probe/microphone is minimal and comparable to routine ear examinations. Whereas most of the test sounds are soft, the more intense sounds are comparable in level to what would be experienced by listening to someone who was talking in a moderately loud voice. There is an extremely low risk of hearing damage with the procedure itself.

BENEFITS:
No benefit can be promised directly to you from participating in this study. However, the results of these experiments are of potential value to society by developing the use of pressure compensated otoacoustic emissions as a part of a new clinical test procedure.

UNIVERSITY OF MIAMI HEALTH SYSTEM
Miami, FL 33136
(305) 243-4009

CLINICAL RESEARCH CONSENT FORM

NAME: ______________________

MRN: ______________________

AGE: _____ DOB: _____ / _____ / _____

PAGE 1 OF 3
ALTERNATIVES:
You have the choice of deciding not to participate in this study. In addition, you may withdraw from this study at any time.

COMPENSATION FOR INJURY:
Should you be injured because of participation in this study, treatment in most cases will be available. If you have insurance, your insurance company may or may not pay for these costs. If you do not have insurance, or if your insurance company refuses to pay, you will be expected to pay. Funds to compensate for apparent pain or discomfort due to an injury, or your lost wages, are not routinely available.

CONFIDENTIALITY:
By signing this consent, you authorize the Investigator(s) and his staff to access and gather your information as may be necessary for purposes of this study. The investigator and his assistant will consider your records confidential to the extent permitted by law. Your test results will not be identified as pertaining to you in any publication without your express written permission. The US Food and Drug Administration (FDA), the US Department of Health and Human Services (DHHS), and authorized University staff may review these research records. If this happens, the FDA or DHHS request will be granted. Your records may also be reviewed for audit compliance purposes by designated University of Miami employees or other agents who will be bound by the same provisions of confidentiality.

RIGHT TO WITHDRAW:
Participation in this study is strictly voluntary. You are free to remove yourself from this study at any time after telling the investigator in charge of the test. You are also aware that the investigators can remove you from the study without your consent if your recordings are not suitable for the study at hand. You are free to ask the investigators any questions that you may have regarding the study. You have the right to refuse to participate at any time.

You have been informed of the procedures to be employed in the otoacoustic emissions study conducted by Dr. Ozcan Ozdamar and his co-investigators, and you agree to participate in this experiment as a subject. The testing will take place in the Neurosensory Engineering Laboratory at the McArthur Engineering Building, University of Miami, Coral Gables Campus.

If you have further questions about the procedures, you may call co-investigator Moises Perez at (305) 753-4489 (day). If you have any questions about your rights as a research participant, you may contact the Human Subjects Office of the University of Miami at (305) 243-3195.

COSTS:
There are no costs to you as a result of participating in this study. You will not be paid for your participation in this study.
CONSENT TO PARTICIPATE IN THIS STUDY:
I have read the above information. The content and meaning of this information has been explained to me.

I hereby voluntarily consent and offer to take part in this study. I understand that the information contained in my test records will be kept confidential to the extent permitted by law. I agree that the results of these tests may be published for scientific purposes provided my identity is not revealed.

I have received a copy of this informed consent agreement.

Signature of Participant ___________________________ Date ___________ Time ___________

Signature of Person Obtaining Consent ___________________________ Date ___________ Time ___________