OPEN ACCESS GUIDE TO AUDIOLOGY AND HEARING AIDS FOR OTOLARYNGOLOGISTS

ACOUSTIC (STAPEDIUS) REFLEXES

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The ***acoustic reflex***, also known as the stapedius reflex refers to an involuntary muscle contraction of the stapedius muscle in response to a high-intensity sound stimulus. Due to ease of administration and information yielded, the acoustic reflex is considered one of the most powerful differential diagnostic audiological procedures.

The ***acoustic reflex*** and the ***tympanogram*** (see tympanometry chapter) are commonly used to ***assess middle ear function*** and are considered best practice. Each test yields invaluable information based on the delivery of acoustic energy (sound) to the ear canal. Thanks to microprocessors, the instruments used are now capable of rapid middle ear function assessment (typically <1min/ear).

Acoustic reflexes do not measure hearing threshold. Rather, they measure reflected energy which is a function of stapedius muscle contraction; it allows one to indirectly assess the middle ear, cochlea and neural innervation of the stapedius muscle.

As the acoustic reflex is involuntary and bilateral, it is ***replicable and provides valuable diagnostic information*** when comparing the amount of reflected energy according to signal intensity, as well as the presence of ipsilateral and bilateral acoustic reflexes.

Premise behind instrumentation

An acoustic stimulus is presented to the external auditory canal; energy is transferred from the ear canal through the middle ear, some of which is reflected and some absorbed by the tympanic membrane and external ear structures.

The premise behind the ***immittance instrumentation*** used to measure the acoustic reflex is the ability to ***accurately measure changes in reflected energy*** occurring from stiffening of the tympanic membrane as a result of contraction of the stapedius muscle (For the premise behind tympanometry see tympanometry chapter). The amount of reflected and absorbed energy varies depending on the admittance/impedance (flow/resistance) of energy within the system and the complex interaction between the ear structures.

The ***presence of reflexes within normal intensity limits*** is consistent with normal middle ear and brainstem function and suggests that auditory sensitivity is not significantly impaired. An ***elevated or absent acoustic reflex threshold*** is consistent with a middle ear disorder, hearing loss in the stimulated ear, and/or interruption of neural innervation of the stapedius muscle.

Anatomy & Physiology

Neural mechanisms mediate the acoustic reflex that results in involuntary stapedius muscle contraction stiffening the stapes within the middle ear. It is presumed that the physiologic reason for the reflex is that it serves as an inhibitory response to reduce the sound intensities reaching the inner ear by as much as 20 dB evoked when individuals vocalise (Møller, 2000).

As is illustrated in *Figure 1*, once a high intensity auditory stimulus is initiated and reaches the cochleae, neural impulses from the auditory nerves (CN VIII) ascend from both cochleae to each ipsilateral ventral cochlear nucleus (VCN). From VCN the reflex has two main neural pathways: one passes from the VCN directly to the ipsilateral facial motor nucleus (CN VII) that

directly innervates the stapedius muscle via the facial nerve and its stapedius branch; the other passes from the VCN to the superior olivary complex (SOC) before the impulses cross at the brainstem to innervate both ipsilateral and contralateral facial motor nuclei.

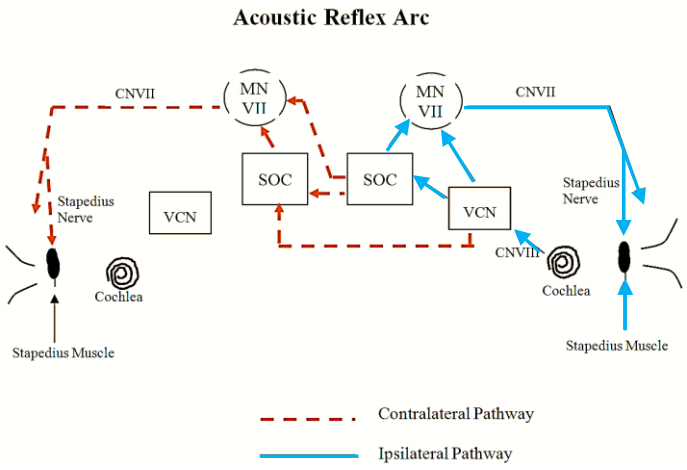

Figure 1: Schematic of the acoustic reflex neural pathways. CNVII (facial/seventh cranial nerve); CNVIII (auditory/eighth cranial nerve); MNVII (motor nucleus of seventh cranial nerve); SOC (superior olivary complex); VCN (ventral cochlear nucleus). Dashed lines represent the contralateral pathway and solid lines represent ipsilateral pathway of acoustic reflex

Contraction of the stapedius muscle stiffens the middle ear ossicles and tilts the stapes in the oval window of the cochlea; this effectively decreases the vibrational energy transmitted to the cochlea. Stapedius muscle contraction is clinically apparent by a marked change in the impedance properties of the middle ear.

It is important to realize that a ***bilateral acoustic reflex occurs*** *i.e.* stimulating one ear (ipsilateral ear) elicits an acoustic reflex in both the ipsilateral and contralateral ears.

Acoustic Threshold

An acoustic reflex threshold is the lowest intensity level at which an acoustic reflex is elicited at each frequency for each ear.

Stapedius muscle contraction increases as the stimulus intensity is increased; it is therefore possible to determine acoustic reflex thresholds by incrementally increasing the stimulus by 5 dB steps, beginning at 80 dB HL, until a contraction occurs *(Figure 2)*. With a normally functioning middle ear and cochlea, an acoustic reflex is elicited contralaterally at four frequencies (500, 1000, 2000, 4000 Hz), and ipsilaterally at two frequencies (1000, 2000 Hz) when stimulus intensities of 85-90 dB HL (hearing level) are delivered to the ear canal.

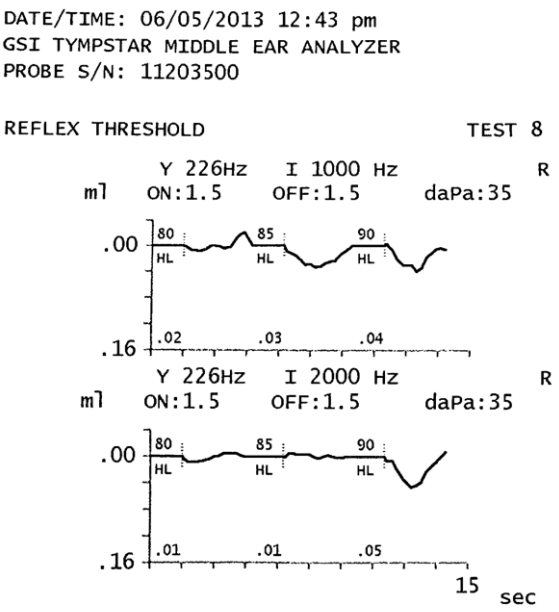

Figure 2: Example (right ear) of acoustic reflex threshold obtained from an immittance instrument. Results shown would indicate 85 & 90 dB HL thresholds for 1000 and 2000 Hz respectively, which would be expected for a normal ear

In a normal ear, after an initial contraction the strength increases with each sequential increase of signal intensity until a plateau is reached. Threshold is defined as the lowest acoustic stimulus intensity elicited with a deflection magnitude of at least 0.03ml. As seen in the magnitude of deflections in *Figure 2*, thresholds for both frequencies assessed clearly fall within the

criteria of at least 0.03ml deflection. To identify a threshold it is advisable to confirm the magnitude of the deflection with a repeat presentation. Once a threshold has been established at one frequency, the intensity is recorded, and the other frequencies are then assessed. During quick screening it is not unusual to assess an acoustic reflex threshold at only one frequency and a single set intensity (~95 or 100 dB HL. An ipsilateral acoustic reflex threshold tends to be attained at a slightly lower intensity stimulus than an acoustic reflex threshold elicited by a contralateral stimulus.

Factors affecting acoustic reflexes

- ***Ipsilateral conductive hearing loss***: Anything that obstructs transmission of a sound signal to the cochlea in the stimulated ear results in reduced or absent contraction of the stapedius muscles. Middle ear disorders therefore easily mask an acoustic reflex. Forty percent of patients with conductive hearing loss of >20 dB HL do not have an acoustic reflex. Once conductive hearing loss reaches 40 dB HL, about 80% of patients do not have a reflex (Jerger, Anthony, Jerger and Mauldin, 1974)
- ***Ipsilateral sensorineural hearing loss:*** This may obstruct transmission of a signal beyond the cochlea of the stimulated ear and usually causes reduced or absent contraction of the stapedius muscles. However, because of recruitment of loudness in cochlear sensorineural hearing loss, acoustic reflexes may occur even within the expected normal or partially elevated intensity range in the presence of mild or moderate-to-severe sensorineural hearing loss.
- ***Stapes fixation:*** The stapes footplate is more-or-less fixed to the surrounding bone with otosclerosis (and sometimes with tympanosclerosis). This may interfere with the acoustic reflex in two ways: it causes a conductive hearing loss; and reduced mobility of the stapes prevents stiffening of the tympanic membrane when the stapedius muscle contracts
- ***Ossicular disarticulation:*** As with otosclerosis, it causes a conductive hearing loss, and if located lateral to the stapes, prevents stiffening of the ossicles and tympanic membrane when the stapedius muscle contracts
- ***Middle ear effusion:*** This causes a conductive hearing loss, and reduces compliance of the tympanic membrane and middle ear structures and may mask the presence of an acoustic reflex
- ***Tympanic membrane perforation:*** This causes a conductive hearing loss. Furthermore, due to the perforation, changes in compliance caused by contraction of the stapedius muscle cannot be measured
- ***Negative/abnormal middle ear pressure:*** To maximize the likelihood that a reflex will be detected, the pressure on either side of the tympanic membrane has to be equal for the tympanic membrane to be at or near the point of maximum compliance (see tympanometry chapter for further explanation). This necessitates one to match the peak pressure of the tympanogram to the actual middle ear pressure so that valid acoustic reflex threshold values may be obtained
- ***Facial nerve dysfunction:*** The stapedius muscle is innervated by the facial nerve. Contraction of the stapedius muscle has to occur for both ipsi- and contralateral acoustic reflexes.

Interpretation of acoustic reflexes

Acoustic reflexes may be reported as:

- ***Ipsilateral:*** Reflex recorded in ear to which auditory stimulus is presented
- ***Contralateral:*** Reflex recorded in ear contralateral to which auditory stimulus is presented
- ***Partially present:*** Reflex present at some frequencies and absent at others
- ***Elevated threshold:*** Reflex thresholds elicited >100 dB HL
- ***Absent reflex*****:** No reflex elicited

Partial or elevated reflex thresholds may indicate the presence of hearing loss at the frequencies where they are specifically absent.

Absent reflexes have also been observed in individuals with normal or near-normal hearing, which may then indicate middle ear disease or neurological involvement of the 8th cranial nerve, such as in pontine angle tumours and auditory neuropathy (see below), or neurological involvement of the 7th cranial nerve. With facial nerve paralysis, the absence of acoustic reflexes in the presence of normal middle ear function suggests a lesion in the neural pathway proximal to the stapedius nerve, whereas the presence of an acoustic reflex in patients with facial nerve paralysis suggests that the lesion is distal to the origin of the nerve. A very small percentage of people will have normal auditory sensitivity and absent acoustic reflexes across all frequencies without other identifiable pathology.

Interruption of Neural Transmission

As is illustrated in *Figure 1,* both ipsi- and contralateral acoustic reflex pathways pass through the SOC of the lower brainstem before proceeding to the facial nerve. Consequently, brainstem lesions can interrupt transduction of neural impulses resulting in an absence or reduction of acoustic reflexes elicited with ipsi- and/or contralateral stimulation.

Reflex Pattern	Reflex	Probe Right	Probe Left	Description	Interpretation
Normal	Contra			Normal	Normal
	Ipsi				
Vertical	Contra		■	Abnormal whenever probe in affected ear	Mild middle ear disorder (left ear)
	Ipsi		■		CN VII (facial nerve) disorder (left side)
Diagonal	Contra	■		Abnormal with sound to the affected ear	Right nerve disorder (left side)
	Ipsi		■		Severe cochlear loss (left ear)
Inverted L-shape	Contra	■	■	Crossed stimulation abnormal in both ears; uncrossed stimulation abnormal in affected ear	Unilateral middle ear disorder (left ear)
	Ipsi		■		Intra-axial brainstem disorder eccentric to one side (left side)
Horizontal	Contra	■	■	Abnormal to crossed stimulation on both ears	Extra-axial and/or intra-axial brainstem disorder
	Ipsi				
Unibox	Contra	■		Abnormal with sound to affected ear on crossed stimulation only	Extra-axial and/or intra-axial brainstem disorder
	Ipsi				

Red box = Absent reflex

Table 1: Six patterns of acoustic reflex thresholds based on presence or absence of each ipsilateral and contralateral reflexes, based on Jerger & Jerger (1977). This example depicts left sided involvement.

Table 1 is a system to classify acoustic reflex thresholds according to the presence or absence of ipsilateral and contralateral reflexes; it has great diagnostic value (Jerger and Jerger, 1977). The system categorises acoustic reflex thresholds into six distinct patterns based on presence or absence of ipsilateral and contralateral reflexes. Cochlear, CN VII, CN VIII, or brainstem sites of lesion are represented by vertical, diagonal, inverted L-shape, horizontal, and unibox patterns.

Wideband reflectance technique

This is a newer measuring technique (Feeney, Douglas and Sanford, 2004) to assess acoustic stapedius reflex thresholds by using complex wideband (125 - 10,000 Hz range) reflectance with a stimulus resembling "chirplike" sounds instead of a single probe tone frequency. The wideband reflectance technique reportedly yields more reliable results than the single probe tone technique, and appears to hold promise as a clinical procedure for measuring acoustic reflexes for normal-hearing subjects who fail to demonstrate reflexes with the standard clinical procedure.

Acoustic Reflex Decay

This is another powerful differential diagnostic test and has a high degree of sensitivity to identify retrocochlear pathology due to *e.g.* tumours of the cerebellopontine angle. However magnetic resonance imaging is considered the examination of choice to identify retrocochlear pathology. In order to examine acoustic reflex decay, an extended duration signal (≥10 seconds) is presented, using low-frequency stimuli (500, 1000 and 2000 Hz) contralateral to the probe (test) ear. Muscle contraction of the acoustic reflex is sustained in the normal ear at maximum or near-maximum amplitude throughout the entire duration of the stimulus.

The standard procedure is to present the stimulus contralaterally at 10 dB SL (threshold of acoustic reflex of test ear), or between 90 and 105 dB HL for 10 seconds. Abnormal reflex decay is evidenced by an inability of the stapedius muscle to maintain full contraction (amplitude) for the duration of the stimulus signal. When reflex decay occurs within 5 seconds or at frequencies less than 1000 Hz, it is suggestive of 8th nerve dysfunction.

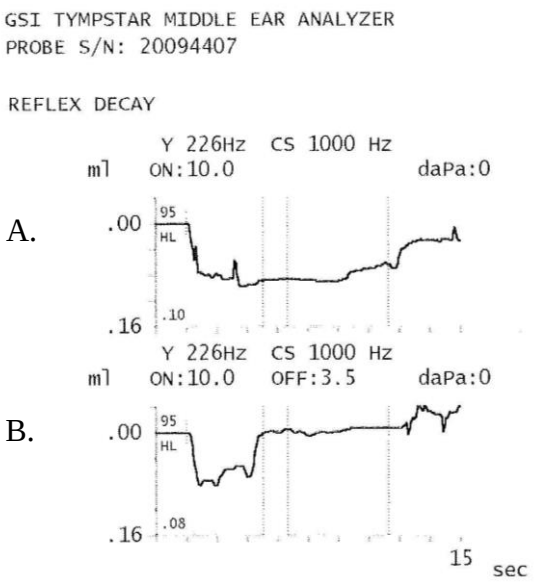

Figure 4: (A) Normal and (B) abnormal findings on acoustic reflex decay test

Concerns have recently been raised about causing temporary or permanent auditory threshold shifts as a consequence of presenting acoustic stimuli for extended periods of time at exceptionally high intensities. For this reason, clinicians are cautioned to administer acoustic reflex decay tests judiciously and never to exceed 105 dB HL presentation levels at any frequency.

Caveats with acoustic reflex measures

It is important to recognize that there are some limitations with acoustic reflex measures.

- ***Correct probe placement*** may become challenging for those patients with external auditory meatal openings being larger than the available probe tips (as seen in the elderly)
- Conversely, the ***very small canal*** openings (as seen in infants) may not be compatible with any of the available probes
- In addition, care must be taken that the patient is capable of ***sitting quietly***. Any jaw movements that occur during

coughing, crying, talking, swallowing, or jaw clenching will result in artifact and provide fallacious results.

- Reportedly, collapsed canals due to placement of ***supra-aural headphones*** on the pinna of the contralateral ear may also result in fallacious findings

Concluding remarks

Despite the few limitations with acoustic reflex threshold testing, the advantages far overshadow those limitations. Although few new discoveries have been made with acoustic reflex threshold testing, it is still considered one of the most powerful differential diagnostic tools that should be within the standard battery of tests attempted with all patients. Within a very brief time period it allows one to identify middle ear pathology; cochlear or retro-cochlear pathology; evaluate neural transmission efficiency; while providing a general indicator of magnitude of hearing loss.

References

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