

OBJECTIVE TINNITUS AND THE TENSOR TYMPANI MUSCLE

Erwin H. Rock, M.D., F.A.C.S.

ABSTRACT

Objective tinnitus (OT) may be caused by contraction of the tensor tympani muscle (TTM). The more forcefully the TTM contracts, the greater the intensity of the OT heard. Forceful closure of both eyelids can reflexively cause OT by contracting the TTM. The *Forceful Eyelid Closure Syndrome* (FECS) was reported at the Proceedings of the Second International Tinnitus Seminar in 1983.¹

FECS consists of several factors:

- (1) Objective tinnitus
- (2) An associated waning of hearing primarily of the lower frequencies, as much as 45 dB at 125 Hz, 30 to 40 dB at 250 Hz ascending to the patient's norm at 2000 Hz and approximately a 5 to 10 dB at 4000 Hz and 5 to 20 dB at 8000 Hz
- (3) Retraction of the manubrium and posterior mid-third of the tympanic membrane (TM) at the malleus-umbo area as seen under the otomicroscope (OM) in 25% (108) of 432 ears examined
- (4) These same ears were 75% (324) positive for increased impedance at maximum compliance with FEC. Of the patients studied, 25% had no response under the otomicroscope or by impedance audiometry.

ANATOMY AND MYOKINESIS

The length of the TTM is between 20 to 25 mm or approximately four times longer than the stapedius muscle. Both of these bipenniform middle-ear muscles increase impedance of the middle-ear mechanism to a sound wave by stiffening the TM, the ossicular chain, and its ligaments. The bipenniform anatomy creates a forceful muscular contraction with minimal displacement.

TTM arises from the cartilaginous part of the eustachian tube and an adjacent part of the greater wing of the sphenoid bone and also from its own bony semicanal. It is housed within this bony semicanal above the bony part of the eustachian tube and terminates into a thin tendon, which turns laterally around the

processus cochleariformis (also called trochleariformis) and attaches to the medial aspect of the root of the manubrium. The TTM is supplied by a motor branch coming from the mandibular division of cranial nerve (CN V). This branch, before passing through the otic ganglion without synapsing, sends a branch to the medial pterygoid muscle and finally innervates the TTM.² The name of the muscle is derived from the fact that it draws the handle of the malleus medially and forward and thus consequently tenses the TM. The contracting TTM also pushes the stapes more tightly into the oval window.

The stapedius muscle, the smallest skeletal muscle in the body, originates from a conical cavity in the pyramidal eminence and its tendon passes through an opening at the apex of this eminence to insert on the neck of the stapes. It is supplied by motor CN-VII. The muscle pulls the stapes posteriorly and tilts the posterior segment of the footplate into the oval window while the anterior part of the footplate is pulled outwardly. This action tightens the annular ligament and thus dampens the stapes' vibratory capacity below 2000 Hz.²

Stiffness is increased when both middle-ear muscles contract together more than with a singular response. The contraction of the two muscles is synergistic but not truly additive. The contracting stapedial muscle initially displaces the TM laterally and is minimally antagonistic to the medial retraction of the TM by the action of the TTM. Suffice to say each contracting muscle increases the acoustic impedance of the sound wave.³

FEC can initiate a reflex that causes contraction of the TTM. The normal eye blink rarely displays the forceful eyelid closure reflex (FECL). The reflex sequence is motor CN-VII activating the orbicularis oculi muscles relaying to motor CN-V to the TTM. A loud sound can elicit the well-known cochleo-palpebral reflex, affecting sensory CN-VIII to motor CN-VII. If the eyelid closure is forceful enough, the reflex can go on to cause TTM contraction. This is especially true if the initial sound created a

startle effect. When the FECS is active, the manubrium and the mid-third of pars tensa of the TM posterior to the malleus-umbo area toward the middle ear retract. The greater the force of the orbicularis oculi muscle contraction, the greater will be the degree of area circumference and intensity of TM retraction. Repeated forceful eyelid contractions eventually habituate the reflex. It may take seconds to several minutes of orbicularis oculi muscle relaxation before this reflex recurs.

Impedance audiometry is three times more sensitive than observing the TM dynamics under the OM, at 10 power. Initially, 432 ears were examined routinely under the OM to note the presence or absence of FECS. All ears had an intact TM without tympanosclerosis. The TM retraction was positive in 25% of these ears whereas impedance audiometry was positive

for increased impedance in 75%. Increased impedance causes a hearing loss for as much as 45 dB at 125 Hz, 30 to 40 dB at 250 Hz and ascending to patient's norm at 2000 Hz and approximately a 5 to 10 dB at 4000 Hz and 5 to 20 dB at 8000 Hz (Fig. 1). Impedance audiometry is carried out at maximal compliance. American Electromedics' AE 83 and the AE 803 impedance audiometers were used in this study. In humans the TTM is not activated by sound *per se* as the stapedius muscle is. In other animals, however, both the TTM and stapedius muscle respond to acoustic stimuli.³⁻⁶

HISTORIC

Hitzig,⁷ Lucae,⁸ and Tschiasny⁹ in 1869, 1874, and 1946, respectively, described a

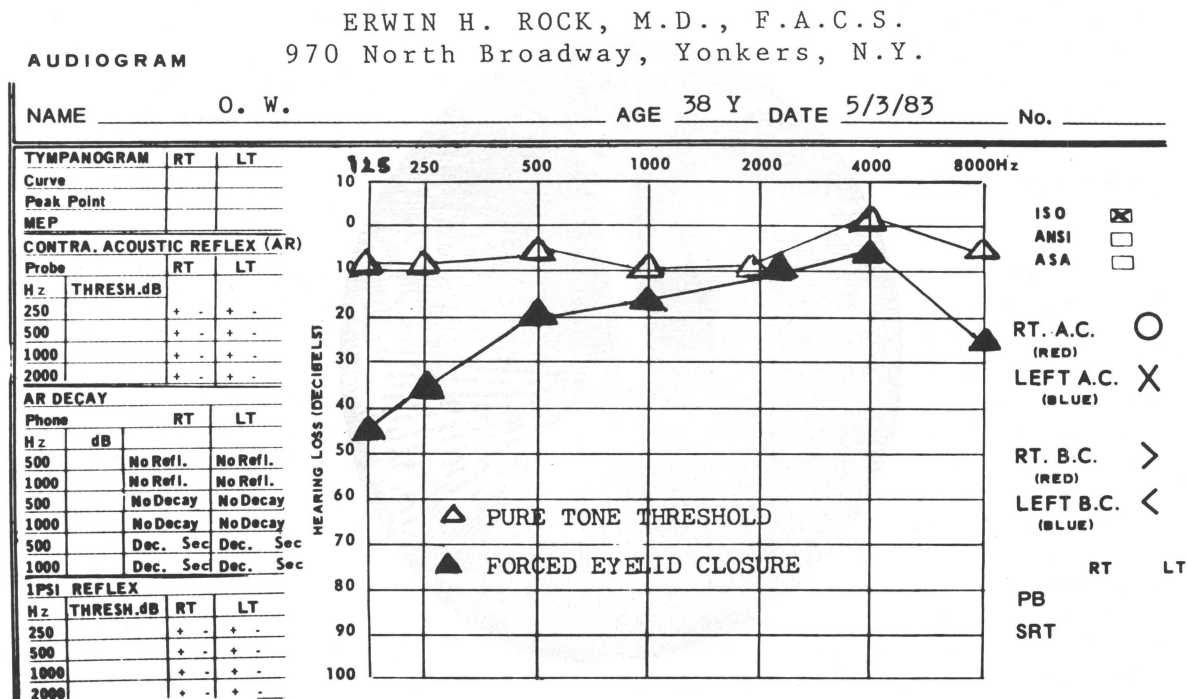


Fig. 1. Audiogram of patient O.W. with an active forceful eyelid closure reflex

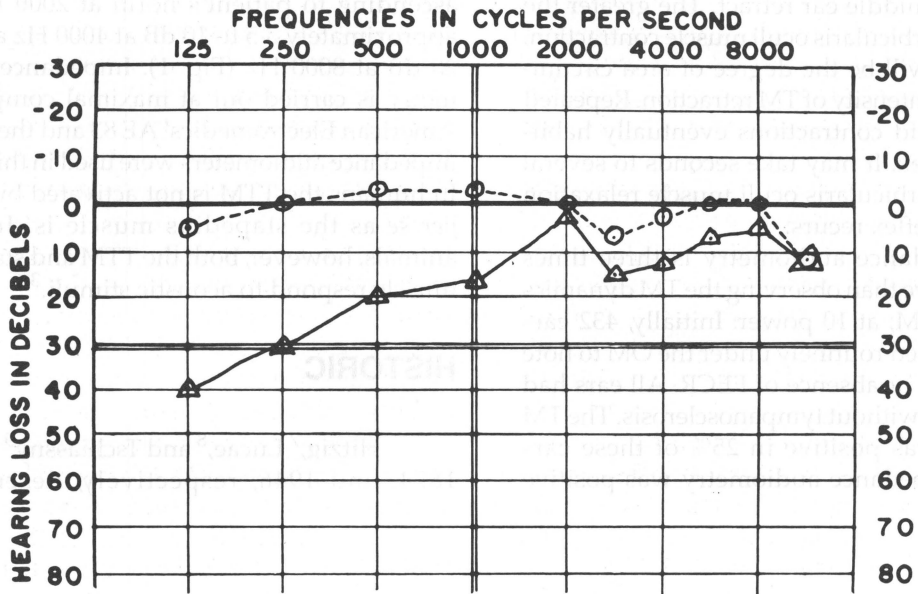


Fig. 2. o-----o average threshold sensitivity of both ears with middle ear muscles relaxed
 Δ——Δ average sensitivity of both ears during maximum voluntary contraction of middle ear muscles (one subject, after Smith)

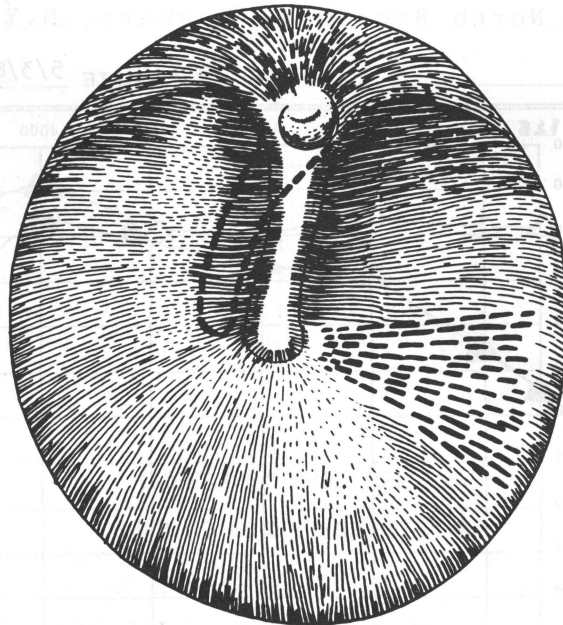


Fig. 3. Appearance of the membrane tympani during contraction of the tensor tympani muscle (after Smith, 1943)

blowing sound heard deep within the ear simultaneous with voluntary contraction of the periorbital muscles. They attributed this to stapedius muscle contraction. Forced eye closure (FEC) and changes in impedance have been observed by Geffcken,⁵ Klockhoff⁶ and Moller.³ Geffcken, however, in 1934, felt that the change in impedance secondary to FEC was caused by the TTM.⁵

Voluntary contraction of the TTM was recognized by Luschka and Politzer over 100 years ago. Politzer (1909)¹¹ reported the following about a patient, "during the contractions the hearing distance was diminished and deep tones became deadened and indistinct and the high tones ascended one quarter of a tone."

Smith (1943), a captain in the United States' Army Medical Corps, reported on the audiometric findings of a lieutenant who could voluntarily contract his TTM.¹² At 8 to 9 years of age he became aware that he could produce a low-pitched roar in both ears when he voluntarily contracted the muscles, and simultaneously diminished hearing enough to block out ordinary conversation (Fig. 2).

Boring conversations could be blocked out at will by voluntary contraction of the TTM. Smith does not state how the lieutenant precisely carried out this voluntary contraction. Maximum intensity of retraction could be maintained for 20 to 30 seconds. Smith showed a diagram of the TM during contraction of the TTM (Fig. 3).

Reger's (1960) audiometric measurements on eight ears during maximum voluntary contraction of their middle ear muscles demonstrated that maximum contraction could be controlled for 20 to 30 seconds. A marked reduction in loudness for environmental sounds was noted as well as a simultaneous fluttering noise in their ears. He attributed this noise to middle ear muscle tremors. The average hearing level for these eight selected ears plotted by Reger (Fig. 4), approximated Smith's audiogram with similar losses greater for the lower tones at 125 Hz and leveling off at 2000 Hz.¹³

Jepsen et al.¹⁰ observed in 1963 that in about 50% of people with normal hearing, FEC caused contraction of the middle ear muscles. He used an impedance bridge in carrying out this observation.

Salomon and Starr (1963)¹⁶ studied the electromyography (EMG) of the middle ear muscle activity of the TTM and stapedius muscle on two patients. One patient was observed before labyrinthectomy and the second through a TM perforation. They found that the TTM responded for up to 15 milliseconds before eye closure and increased both frequency and amplitude of TTM potentials. With relaxed eye closure, no tensor tympani activity was found. TTM activity occurred 20 to 60 milliseconds after periorbital activity using an air jet blown against the eyes. Voluntary FEC created a more consistent tensor activity than that caused reflexively by the orbital air jet. Habituation occurred rapidly. Jaw muscle activity did not activate the TTM.

TTM activity was always elicited before or simultaneously with talking or humming. Therefore this represents a non-acoustic function because of the loss of the normal skeletal muscular latency period. Forceful voluntary eyelid closure or orbital air jet activity was not associated with any changes of stapedius muscle activity.

Visual observation using an OM demonstrated stapedius muscle activity during vocalization, coughing, but no activity with eye closure. The TTM remained active for the duration in a subject lying supine who lifted the head and maintained a flexed head-neck position.⁴

Djupesland also carried out EMG studies on the middle ear muscles and concluded that weak voluntary contraction of the periorbital muscles caused no activity in either muscle. With complex movements, however, such as FEC, opening of the mouth, teeth clenching, swallowing, and speaking, there were accompanying impedance changes which seem to be caused by both middle ear muscles. With the above maneuvers, both CN-V and CN-VII were activated.⁵

METHOD

In 1980, a 16-year-old male patient with normal hearing and without any past history of any serious ear problems and with normal TMs presented with the prime complaint of being aware of a fluttering in his ears when he forcefully blinked his eyelids.

The relationship between observing the TM movement under the microscope when the patient closed his eyelids in this manner and had a simultaneous objective tinnitus became very apparent. At that time, I was totally unaware of the fact that as early as 1869 Hitzig already was cognizant that voluntary contraction of the periorbital muscles cause a sound to be heard within the ears.

A total of 432 ears was examined according to the following protocol. The TMs were essentially anatomically normal. During routine ear examination, the patients were asked to forcefully close both eyelids, then clench their teeth tightly without closing the eyes. They were asked what sound, if any, they heard with each maneuver. The patient's hearing status was not considered to be a factor for this study. Otomicroscopy with 10x and 16x was positive, showing retraction of the TM as already described in 25% (108 ears), whereas impedance audiometry was positive with an increase in impedance in 75% (324) of these total ears. Of the ears, 25% (108) showed no response either by otomicroscopy or by impedance measurements. The sensitivity of impedance audiometry over otomicroscopy is obvious.

One patient did not forcefully close both eyelids but did so with minimal force only and had a positive FECR with both impedance and TM retraction observed under the OM. Thus, an individual sensitivity for the FECR exists. This patient also was aware of a head noise that was observed to be simultaneous with the TM retraction. She was aware that the head noise resulted from eyelid closure before my examination, but unaware of its significance.

Teeth clenching using the masseter and temporalis muscles both innervated by motor CN-V was likewise negative for the FECR. A smile-activating motor CN-VII likewise showed no measurable response for the FECR either by the otomicroscope or impedance audiometry. Tinnitus was heard only with the FEC and not with any of the other maneuvers.

A positive FECR did not change an existing subjective tinnitus, rather the objective tinnitus brought on by the FEC became an additional tinnitus. Practically all patients with a positive FECR had a simultaneous objective tinnitus. The objective tinnitus is best heard with a Toynbee auscultation tube with both the

patient and examiner in a soundproof booth. All types of objective tinnitus were described, for instance, "crackling, scratching, fluttering (like a bird flutters), moth fluttering, swishing, pressing high-pitched sound, like bones are moving, water flowing, ting, hissing, wind blowing, and an audible vibration." The objective tinnitus is most likely related to the contraction and subsequent relaxation primarily of the tensor tympani muscle. The retraction of the malleus's tensing the TM and ossicular ligaments along with ossicular joint motion may also contribute to the objective tinnitus.

Most patients in this study who showed a positive FECR were totally unaware of this capability. They never realized beforehand that FEC could cause a head noise. The reflex mechanism was explained to the patient and its relative insignificance.

DISCUSSION

The TM is tensed when the malleus handle is retracted by tensor tympani muscle contraction. The annulus at the footplate is likewise stiffened and the intralabyrinthine pressure is also increased. An increase in stiffness of the middle ear enhances the response to the higher frequencies, whereas the stiffened ligaments impede mass (ossicles) mobility for the lower frequencies. Thus, a low tone hearing loss is expected because of the stiffness factor created in the middle ear mechanism. The more the tensor tympani muscle contracts, the greater the resulting hearing loss. An audiogram (see Fig. 1) of a patient with an active FECR compares similarly with those of Smith and Reger (see Figs. 2 and 4).

Anything that initiates a forceful orbicularis oculi contraction (i.e., a startle which can cause a forceful eye blink, essential blepharospasm, blepharospasm in hemifacial spasm, or psychogenic blepharospasm) may cause the hearing deficit along with synchronous objective tinnitus. Drugs that act either directly or as a side effect can increase muscular irritability and act as a catalyst in initiating the FECR.

One patient complained of an intermittent click in her ears and was anxious and concerned because of not knowing the cause of

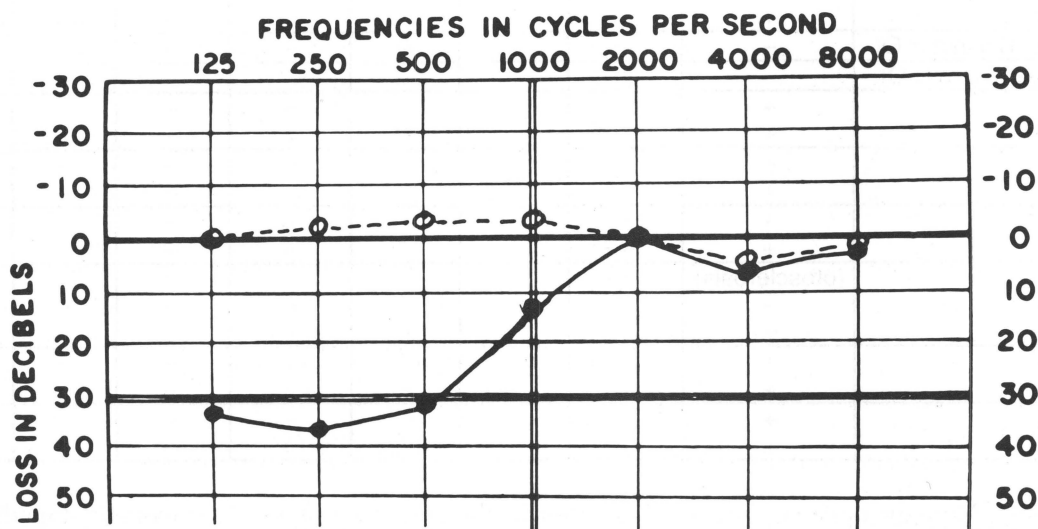


Fig. 4. o-----o average hearing level of eight selected ears
 •-----• average hearing level of above ears during maximum voluntary contraction of middle ear muscles (after Reger, 1960)

her head noise. She was recently placed on anhydrous theophylline (Elixophyllin [Forest], a methylxanthine chemically related to caffeine) because of a pulmonary problem. Muscle twitching, central nervous system stimulation, and reflex hyperexcitability are three of the adverse effects of the drug. This patient had an active eyelid closure reflex, both under the OM and on impedance audiometry. Here a routine and not truly forceful eyelid closure recreated the patient's head noise. The theophylline was discontinued with subsequent relief of the head noise. Any drug that can increase muscle irritability or cause tremors (myokymia) and increase the sensitivity of the FECR has to be considered as etiogenic, for instance, any synthetic sympathomimetic amine.

A normal ossicular chain shows the greatest amplitude of TM movement and increase in impedance when the FECR is active. If, however, the malleus is ankylosed, the TM movement will be lost as well as the presence of any impedance measurements. If only the

stapes is ankylosed, the malleus will still move but with less magnitude and a positive FECR, albeit with less amplitude, may be measured by impedance audiometry.⁶ Thus, it is wiser to forewarn the patients when carrying out acoustic stapedial muscle reflex testing, that they should not be surprised or startled when hearing the rather loud sound. This is especially true with the ear demonstrating recruitment. "Do not close your eyes when you hear the sound." The patient with ankylosis of the stapes, or one with necrosis of the distal third of the incus who has an active FECR, may falsely show a positive acoustic stapedial reflex if he or she should forcefully close the eyelids simultaneous with the sound heard. Here, a true negative acoustic stapedial muscle reflex can be mistakenly interpreted as a positive one because of the FECR. Table I is an impedance audiometric comparative study of the presence or absence of the FECR and the acoustic stapedial reflex along with other various head, neck and body kinetics.

Tympanogram	Acoustic stapedial reflex	Forceful eyelid closure	Normal eyelid blink	head flexed toward chest	Ipsilateral tactile antero-tragal	Head rotating right	Head rotating left	Trunk flexion
As	+	+	-	-	-	-	-	-
A	+	+	-	-	-	-	-	-
A	+	+	-	-	-	-	-	-
A	+	+	-	-	-	-	-	-
As	+	+	-	-	-	-	-	-
Ad	+	+	-	-	-	-	-	-
C (-175 mm H ₂ O)	-	+	-	-	-	-	-	-
C (-200 mm H ₂ O)	-	+	-	-	-	-	-	-
Ad	+	+	-	-	-	-	-	-
Ad	+	+	-	-	-	-	-	-
A	+	+	-	-	-	-	-	-
A	+	+	-	-	-	-	-	-
A	+	+	-	-	-	-	-	-
A	+	+	-	-	-	-	-	-
As	(otosclerosis) -	+	-	-	-	-	-	-
A	+	+	-	-	-	-	-	-
As	+	+	-	-	-	-	-	-
As	+	+	-	-	-	-	-	-
Ad	+	+	-	-	-	-	-	-
Ad	+	+	-	-	-	-	-	-

Table I. Note the positive change in impedance when there was a negative acoustic stapedial reflex

- + Change in impedance
- No change in impedance

The FECR, as stated, is a consensual reflex. If one ear shows a positive FECR under the OM with the expected TM excursion and the opposite ear minimal to no movement, one should consider this latter ear as suspect. The physician should be on guard for an impairment of mobility of the ossicular chain, for example, fixed malleus, ankylosed stapes, or tympanosclerosis binding an ossicle or ossicles.

Knowing the source of their head noise and of its relative insignificance was emotionally therapeutic for most patients. Some who complained that their hearing waned at times may have an active and/or more sensitive FECR. Thus, normal hearing may be temporarily worsened; those subjects with an already pre-existing mild hearing loss may increase to a moderate hearing loss.

Do not necessarily classify all these patients as being neurotic when routine causes of an intermittent type hearing loss cannot be found. Muscle relaxants can be tried or tenotomy of the TT muscle may be plausible if

necessary. The FECS is a practical, albeit uncommon, clinical entity and can be easily missed in routine clinical practice.

SUMMARY

A syndrome brought on by FEC usually initiates objective tinnitus. Hearing may wane simultaneously with the FEC. TM observation with the OM disclosed that in 25% of patients, the mid-third of pars tensa posterior to the handle of the malleus-umbo area retracts toward the middle ear along with the manubrium. This observation was present in 25% (108) of 432 ears examined for the FECR. A positive change in impedance was found in impedance audiometry in 75% (324 ears) in the same study. When there was a positive TM change, there was always a concurrent positive impedance change. In 108 or 25% of the ears, no change was noted in either TM contraction or impedance. The more forceful the FEC, the greater the TM retraction observed during otomicroscopy.

REFERENCES

1. Rock E: Forceful eyelid closure syndrome. Proceedings of the Second International Tinnitus Seminar, New York City: June 1983, pp. 165-69.
2. Williams P, Warnick R, Dyson M, Bannister L: The auditory and vestibular apparatus. In Gray's Anatomy. Philadelphia: Lea & Febiger, 1989, pp. 1227-28,
3. Moller A: Physiology of hearing. In Head and Neck Surgery (Self Instructional Package). American Academy of Otolaryngology, 1981, pp. 24-31.
4. Salomon G, Starr A: Electromyography of the middle ear muscles in man during motor activities. *Acta Neurol Scand* 39:161-68, 1963.
5. Djupesland G: Advanced Reflex Considerations. In Handbook of Clinical Impedance Audiometry. Jerger J, Ed., 1975, pp. 90-93.
6. Klockhoff I: Middle ear muscle reflex tests for otologic diagnosis. *Transactions Am Ophthalmol Otolaryngol* 67:777-84, 1976.
7. Hitzig E: Beiträge zur Kenntnis der Peripheren Lähmungen des Facialis. *Berl Klin Wochenschr* 6:18-20, 1869.
8. Lucae A: Die Accommodation und die Accommodations-Störungen des Ohres. *Berl Klin Uschr* 11:163-65, 1874.
9. Tschiasny K: The site of the facial nerve lesion in cases of Ramsay Hunt's syndrome. *Ann Otol Rhinol Laryngol* 55:152-74, 1946.
10. Jepsen O: Middle ear muscle reflexed in man. In Jerger J: *Modern Developments in Audiology*. Jerger J, Ed. New York: Academic Press, 1963, pp. 193-239.
11. Politzer A: *Diseases of the Ear*, 5th Ed. London; Bailliére and Cox, 1909.
12. Smith H: Audiometric effects of voluntary contraction of the tensor tympanic muscle. *Arch Otolaryngol* 369-72, 1943.
13. Reger S: Effect of middle ear muscle action on certain psychophysical measurements. *Ann Otol Rhin Laryngol* 69: 1179-98, 1960.

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Requests for reprints of this article should be addressed to:

Erwin H. Rock, M.D., F.A.C.S., 970 No. Broadway, Ste. 110, Yonkers, NY 10701, U.S.A.