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MIDDLE EAR FUNCTION SEMINAR

held on

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Major John L. Fletcher, MSC, USA, Editor

Psychology Division
US ARMY MEDICAL RESEARCH LABORATORY
Fort Knox, Kentucky

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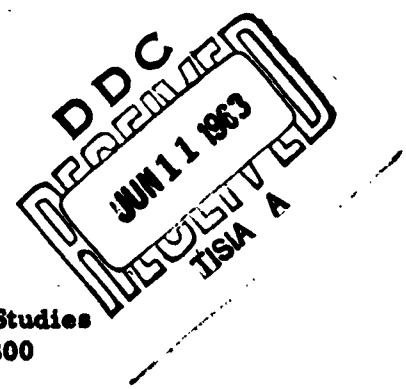

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INTRODUCTION

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Many aspects and facets of the reflex response of the stapedius and tensor tympani muscles and its relation to the acoustic properties of the ear have received research attention for the past hundred years. The practical implications of research on this subject have inspired a considerable increase in such research in the last decade. Research relevant to this problem has been conducted by investigators with widely varying scientific backgrounds, including physiologists, psychologists, electrical engineers, otolaryngologists, audiologists, and others. As a consequence of the variety of professions represented by those studying this problem, no one professional meeting provides an adequate medium for interchange of ideas among all those interested and publishing in this field. Scientific reports on this subject are to be found in a variety of journals. An obvious lack in this area is a coordinated and organized attack upon the problem by all those interested in it.

Dr. David Hilding, who had collaborated with the editor of this seminar on a study of middle ear muscle function, remarked while on a visit to the US Army Medical Research Laboratory on the lack of assembly under one roof of all those interested in middle ear function. This remark led to a discussion of why such an assembly had not been held and of the possible benefits to be derived from such a meeting. It was apparent that, because many disciplines were engaged in middle ear work, no one professional meeting would attract all of those interested in the problem. The logical end to our discussion was the resolution to hold a special meeting, apart from any one professional meeting, where a broad sampling of those investigating middle ear function would convene. Acting upon this conclusion, queries were directed to persons working in middle ear function to determine interest in such a meeting, an appropriate date for the event, and to arrange for a well rounded program. Response to the inquiry was overwhelmingly positive, and the dates of 7-8 May 1962 were selected.

The purpose of the seminar, consistent with the original idea, was to assemble as many as possible of those exploring middle ear function and to determine as precisely as possible the present level

of knowledge regarding this area. After delineating the known, an attempt could then be made to outline areas where further research was necessary. The success of the seminar can be judged from the papers and summary presented in the following sections.

The editor must accept blame for the faults that may be found in the organization and presentation of the seminar. Credit for the details of the arrangements should be shared among Colonel Bach, Commanding Officer of the US Army Medical Research Laboratory, Dr. Odell, Technical Director of Research, Colonel Carilia, Executive Officer, Major Huckabey, Adjutant, and Dr. Harker, Director, Psychology Division. Special mention and the undying gratitude of the editor are richly deserved by Mrs. LaVerne Downs, Miss Mary A. Rhodes, Miss Marilee Rosenfield, and Miss Cynthia Skaggs, who, together and separately, transcribed, edited, reproduced, and in other ways, managed to get this manuscript ready for publication. The efforts of Mr. Richard Wheeler and Mr. Walter Smith in managing the projection and recording of the seminar were also deeply appreciated, as are the contributions of so many others that space does not permit their listing.

FINE INNERVATION PATTERNS OF THE MIDDLE EAR MUSCLES

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The student of middle ear function today cannot help but be impressed with and challenged by the unique behavioral characteristics of the tensor tympani and stapedius muscles. Their contraction is initiated by auditory, tactile and pressure stimuli of homolateral or contralateral origin and they can be conditioned to respond to others. Their threshold is stable, and their latency is considerably shorter than that of other skeletal muscles, including those associated with vision. They develop an initial high tension within 100 msec followed by a decrease of 50% of maximum within 10-20 sec, a behavior which is paralleled by cochlear fibers and cochlear nuclei. Fatigue is apparently dependent upon auditory frequencies rather than intramuscular phenomena. The muscles do not exhibit summation of subthreshold tones, but respond to second threshold stimuli superimposed upon the first.

Although these parameters and others are subject to increasing experimentation, our knowledge of the function of the tympanic muscles is still incomplete. Galambos and Rupert (1959) and Simmons (1959) have clearly demonstrated the protective role of the stapedius, but the function of the tensor tympani is not clear. The tensor is apparently the stronger of the two but less important in auditory function. Klockhoff and Anderson (1960) consider its activity to be a component of the startle response.

While the literature of the last ten years testifies to a prominent interest in the function of the tympanic muscles, it appears that much physiological data have been accumulated without comparable knowledge of the neuromuscular substrate on which behavior depends. Although a variety of experimental animals have been used, the literature affords few accounts of the pattern of fine innervation of these muscles. Despite universal use of the cat in otologic investigations, no data are available for tympanic muscles in this species with regard to the caliber and spectra of nerve supply, the nature and distribution of nerve endings, and the size and number of motor units. This paper will report some of our findings on these points with the purpose

adding to our knowledge of the neurological basis of middle ear activity.

MATERIALS AND METHODS

Tensor tympani and stapedius muscles from 10 healthy adult cats were used in this study. Tissues used for nerve fiber analysis and neuromuscular histology were fixed in 10% neutral formalin by intra-arterial perfusion. Muscles for histochemical observations were removed directly from the anaesthetized animal or from freshly killed animals.

Four staining procedures were employed. For nerve fiber analysis, the Alzheimer-Mann-Haggqvist method (Rexed, 1948) and the silver protargol method of Bodian (1936) counterstained with analine blue were used. IHAB was utilized for muscle fiber counts and tracing intramuscular nerve branching. Bodian's silver method proved most successful for studying nerve endings. For histochemical studies, the Gomori (1952) modification of the thiocholine technique of Koelle was used.

Nerve fiber counts were accomplished by a modification of the strip method of Davenport and Barnes (1935). Nerve fiber calibers were measured from enlarged photographs after the method of Rexed (1944).

OBSERVATIONS AND RESULTS

Gross Nerve-Muscle Relations

Tensor tympani: The tympanic muscles of the cat lie within the lateral portion, or mesotympanum, of the middle ear cavity. Ventral exposure of this cavity brings the tensor tympani immediately into view. It is shaped like a fat teardrop and rests in a small fossa modified to its contour. Its longitudinal axis is oriented approximately 90° to the plane of the manubrium of the malleus and is, therefore, not directed in the plane of the pharyngotympanic tube. The perimysial capsule is continuous with the periosteum of its fossa and anteromedially it blends into the cartilage and connective tissue of the pharyngotympanic tube. The tendon issues from a broad base within the muscle and becomes narrowed near its insertion on the manubrium, at a point 1 mm ventral to the plane of the Eustachian tube. The muscle is basically circumpennate in arrangement.

The nerve to the tensor tympani originates from the mandibular nerve in close relation to the proximal border of the otic ganglion. After receiving fibers from this ganglion, it passes medially underneath the cartilaginous wall of the Eustachian tube. It runs posteriorly for a distance of 4-5 mm before entering the base of the muscle at nearly right angles to its longitudinal axis.

Stapedius: The stapedius muscle lies enclosed in a narrow bony cavity whose outline is marked by the Y-shaped anastomosis of the auricular branch of the vagus with the facial nerve. It is visible only after removal of an overlying cartilaginous styliform process that is used as a convenient support for the beginning portion of the chorda tympani. Its tendon emerges from a slender fissure to pass ventral to the facial nerve and insert on the posterior crus of the stapes. Its muscle fibers originate on the periosteum of the stapedial cavity and converge centripetally on an eccentrically placed tendon. It demonstrates a modified circumpennate pattern.

The nerves to the stapedium emerge from peripheral fasciculi of the facial nerve near the point where the latter is crossed by the stapedial tendon. Three or four branches emerge separately to follow a course parallel to the tendon before entering the muscle in the plane of the muscle fibers.

Neuromuscular Histology

Tensor tympani: In midsagittal section, the tensor tympani resembles the shape of a teardrop, with one flattened edge. The tendon forms the tip of the teardrop, and as it spreads out within the muscle it becomes enclosed by a cap of muscle fibers. It consists of collagenous fibers which fan out like the spokes of a wheel. Near their termination, these fibers separate to form spiral tendrils which embrace the muscle fibers. Muscle fibers are organized into well-defined fascicles which envelop the base and sides of the tendon. Deposits of adipose tissue are found between the radial components of the tendon as well as between muscle fibers. Primary and secondary nerve rami form a crescent-shaped arcade across the middle third of the muscle fibers at right angles to them.

In cross section, the muscle presents an oval outline enclosed in a well-defined epimysium. The base of the tendon, which is deeply placed, is surrounded by numerous muscle fasciculi containing muscle fibers 20-30 μ in diameter. Toward the periphery, smaller fasciculi

are evident, whose muscle fibers measure 5-10 μ in diameter. The main nerve forms three to four primary branches either immediately before or shortly after penetrating the perimysium. These branches follow the perimysial septae at right angles, and each one supplies a major zone of the muscle. Short secondary branches appear deeper within the muscle, which turn to follow the longitudinal axis of the fasciculi. Tertiary branches are subsequently formed which turn again to assume a course at oblique angles to the muscle fibers. Successive divisions are formed, each containing fewer nerve fibers until terminal branching is complete. Although terminal axons appear from all nerve trunks, the frequency of departure increases as smaller nerve branches are formed. Each terminal axon has a short course before ending on a single muscle fiber. Although extensive intramuscular nerve branching is evident, all branches are limited to a relatively narrow region in the middle third of the muscle fibers. An innervation band is thus formed, which in longitudinal section follows the contour of the muscle.

Stapedius: Relations of the stapedius muscle and the facial nerve are best seen in cross section. To prepare the specimen for section, the muscle is removed with short segments of the facial nerve and the auricular branch of the vagus. The muscle and the auricular branch are pulled toward the facial nerve, whose distal stump is straightened out so as to be in the longitudinal axis of the preparation. This orientation permits simultaneous section of the nerves and the muscle and enables one to follow the pattern of nerve supply. In such preparations, the muscle appears in triangular outline on the edge of the facial nerve. Its well-defined fasciculi contain muscle fibers 13-30 μ in diameter. Three to four nerves may be found originating from the facial nerve at different levels. Their fibers stream out peripherally toward their individual outlets and arch toward the muscle to enter it at different points. Although each nerve supplies a major zone of fasciculi, some overlapping may occur. Secondary nerve branches are soon formed which surround individual fasciculi within each zone. From these, tertiary and quarternary branches appear which form innervation rings around the muscle in limited planes. Terminal axons branch from all intramuscular nerve trunks, but with increasing frequency from the smaller ones. The innervation area follows the contour of the muscle and thus defines an innervation band that is restricted to the basal third of the muscle.

Caliber and Spectrum of Nerves

Although it has been well demonstrated that many cranial nerves exhibit a unimodal nerve fiver spectrum (Haggqvist, 1938, 1939; Rexed

1944; Fernand and Young, 1951), there is apparently only one report of the caliber of nerves supplying the tympanic muscles. Malmfors and Wersäll (1960 a, b) have shown that a unimodal pattern is characteristic of both the tensor tympani and stapedial nerves in the rabbit. This investigation has found much the same feature in the cat. Figure 1 shows the results of fiber analysis at different points along the nerve to the tensor tympani. It will be noted that regardless of level, fiber caliber is less than 6 μ and the peak of fiber population is within the 2 - 2.9 μ range. Figure 2 shows results for the stapedial nerves at intramuscular levels. Although the distribution is unimodal, the spectrum is broader than for the nerve to the tensor. The majority of fibers are within the 3 - 3.9 μ range except for the smallest intramuscular nerve branches.

Nerve Endings

The Bodian silver protargol method permits the identification of many well-developed motor end-plates in both tympanic muscles. Such endings are densely concentrated in the region of the innervation bands. Terminal arborizations and large sole-plate nuclei are readily evident. The terminal axon approaches the end-plate either at one pole or at the center to ramify in crab-like fashion among the sole-plate nuclei. Sole-plate formations vary from 14-40 μ in diameter in the tensor tympani and 14-30 μ in the stapedius. The motor end-plates are of the "terminaison en placque" type which appear to define three basic forms. The most numerous are the short compact forms which are found in closely packed groups on adjacent muscle fibers. A third type was occasionally found in which the terminal axon formed two branches, each of which supplied separate groups of sole-plate nuclei on the same muscle fiber. Terminal axons were never found to supply end-plate formations on more than one muscle fiber.

Although it is demonstrated that the middle ear muscles are generously endowed with motor innervation, the question of sensory innervation is less conclusive. Some investigators have reported tendinous and intramuscular sensory endings (Ruffini, 1893; Krebs, 1905; Steinitz, 1907), but photographic evidence is lacking. This investigation has revealed structures which, at first glance, give the impression of sensory formation. Near the edge of muscle fasciculi, connective tissue fibers wrap around peripheral muscle fibers in spiral fashion or form a reticular fusiform enclosure of muscle fibers. Such formations stain intensely with silver protein, but when exposed to IHAB they demonstrate collagenous composition. Small

intramuscular nerves have been found whose fibers form arcades at right angles to individual muscle fibers. Although these formations exhibit a slight resemblance to annulospiral endings of muscle spindles, they are traceable to end-plate formations in serial sections.

Malmfors and Wersall (1960 a, b) have used the Gross-Schultze and Bielschowsky-Landau methods to demonstrate slender nerve filaments ending blindly on the surface of muscle fibers. They describe such formations in both the tensor and the stapedius and assume them to be sensory in function. The Bodian method as employed in this investigation reveals similar structures, but faintly stained nuclei are found in close association at the end of such fibers. In view of the presence of sole-plate nuclei in the vicinity of endings of this type, the observation is made that such fine free endings are more probably motor fibers whose end-plate formations have capriciously escaped the deposit of sliver or are not visible in the plane of section.

The foregoing observations support the opinion that typical sensory endings are absent in the tympanic muscles. Further evidence favoring this view is afforded by histochemical techniques. There is ample evidence in the literature that sensory endings related to stretch or pressure exhibit cholinesterase activity. It has been demonstrated in muscle spindles (Coërs, 1954; Gerebetzoff, 1955), carotid sinus and aortic pressoreceptor fibers (Abraham, 1956) and in Pacinian corpuscles (Coupland and Holmes, 1957). The thiocholine method employed in this study shows that the tympanic muscles exhibit a considerable degree of cholinesterase activity. This activity is limited to the sites of motor end-plates which, therefore, demonstrate a band of cholinesterase activity that is coincident with the innervation band. Under high magnification the subneural apparatus of extrafusal end-plates is clearly stained, but neither spindle formations nor proprioceptor fibers are observed within the muscles or their tendons. If sensory endings are present, they do not clearly stain with traditional silver methods and they do not appear to exhibit cholinesterase activity.

Motor Units and Innervation Ratios

The definition of a "motor unit" is attributed to Sherrington (1929). He said, "the muscle with its nerve may be thought of as an additive assemblage of motor units, meaning by a motor unit, an individual nerve fiber with the bunch of muscle fibers it activates." The size of the motor unit is calculated by dividing the number of

fibers in a muscle by the number of motor fibers in its nerve. The resulting number represents the number of muscle fibers supplied by a single motor neuron. It may be expressed as a ratio of 1 (e.g., 1:20). The smaller the ratio, the greater is the possibility for refined delicate coordination. The number of muscle fibers per motor nerve may be as high as 2,000 in the gastrocnemius or as small as 25 in the platysma, but the majority of skeletal muscles exhibit ratios over 1:100. Muscles associated with organs of special sense apparently have smaller ratios. The extraocular muscles exhibit ratios of 1:10 or less in the sheep (Tergast, 1873) and 1:6 or less in man (Bors, 1925).

The estimation of motor unit size is not without experimental error. The variable direction of muscle fibers, particularly in pennate muscles, renders their enumeration difficult. This can be reduced by counting adjacent regions in sequential sections. Estimation of afferent fiber population of spinal nerves is accomplished by counting fibers after section of the posterior root ganglia. Using this technique, Sherrington (1894) estimated that one-third to one-half of the myelinated fibers of spinal nerves were afferent. Since that time, investigators have used these values in evaluating motor units for muscles innervated by both spinal and cranial nerves, although there are no data available for estimating afferent fiber populations of cranial nerves.

Limited data are available on the size of tympanic muscle motor units. Berlendis (1955) found a ratio of 1:27 for the tensor tympani of the rabbit and Berlendis and DeCaro (1955) observed a ratio of 1:30 for the stapedius. In the same species, Malmfors and Wersäll (1960, a, b) estimated a motor unit of 1:8 for the tensor and 1:14 to 1:20 in the stapedius. Torre (1953) studied a single specimen of the human tensor tympani in which he observed a ratio of 1:7.6.

Table 1 shows the results of this investigation on 5 specimens of the tensor tympani of the cat. Nerve fiber counts were taken at points prior to axon branching. The first column indicates the number of nerve fibers counted in each specimen. The second column indicates the number of motor fibers based on a motor fiber population of 50% to 66-2/3%. The last column of the table indicates the size of motor units if the values of Sherrington (1894) are used. It will be noticed that although the number of muscle fibers and nerve fibers in the individual specimens varies considerably, the size of the motor units is remarkably similar. A mean value of 1:7.1 to 1:5.3 is observed. In view of the apparent lack of sensory endings, it would seem that a

more accurate measure of nerve-muscle relations should make no allowances for afferent fiber populations. We have chosen to indicate this measurement as the innervation or nerve fiber:muscle fiber ratio. The results are shown in column four, in which a mean value of 1:3.5 is indicated. It is interpreted to mean that each motor neuron supplies three to four muscle fibers.

Table 2 shows the results of enumerations in six specimens of the stapedius nerve and muscle. The number of nerve fibers and muscle fibers is less than in the tensor, and the variation among individual specimens is smaller. Both motor unit and innervation ratios are remarkably similar. The mean value for the motor unit is 1:4.8 to 1:3.6 and the innervation ratio is 1:2.4.

The number of motor units in each muscle may be assessed by dividing the total number of muscle fibers by the number of muscle fibers in the motor unit. Using the data in columns three and five of the tables, the tensor tympani has a mean number of 564-754 motor units compared to 360-480 units in the stapedius. If one uses the values from the fourth column in each case, the tensor tympani contains 1140 motor innervated groups, while the stapedius has approximately 720.

SUMMARY

In summary, it can be said that the fine innervation patterns of the tympanic muscles in the cat provides a rich substrate for refined motor performance. They are plentifully supplied with motor endplates whose spatial arrangement provides for remarkable efficiency. Their innervation ratios are among the smallest recorded. In view of the lack of detectable intramuscular sensory endings, it would appear that limitation of movement is accomplished by other than proprioceptive means. Since the behavior of these muscles closely parallels the nature of sensory input, it is probable that their coordination is determined by factors of cochlear or central nervous system origin.

REFERENCES

Abraham, A. Über die Struktur und die Endigungen der Aorticusfasern im Aortenbogen des Menschen mit Berücksichtigung der cholinesterase-Aktivität der Pressorezeptoren. Ztschr. f. mikr. anat. Forsch., 1956, 62, 194-228.

Berlendis, P. A. L'unitá motoria del muscolo tensore del timpano. Boll. soc. med.-chir. Pavia, 1955, 69, 37-39.

Berlendis, P. A. and De Caro, L. G. L'unitá motoria del muscolo stapedio. Boll. soc. med.-chir. Pavia, 1955, 69, 33-36.

Bodian, D. A new method for staining nerve fibers and nerve endings in mounted paraffin sections. Anat. Rec., 1936, 65, 89-97.

Bors, E. Über Zahlenverhältnis zwischen Nerven und Muskelfasern. Anat. Anz., 1925, 60, 415.

Coërs, C. and Durand, J. La repartition des appareils cholinesterasiques en cupule dans divers muscles striés. Arch. Biol., Paris, 1957, 68, 209-215.

Coupland, R. E. and Holmes, R. L. The use of cholinesterase techniques for the demonstration of peripheral nervous structures. Quar. J. Micr. Sci., 1957, 98, 327-330.

Davenport, H. A. and Barnes, J. R. The strip method for counting nerve fibers or other microscopic units. Stain Technology, 1935, 10, 139.

Fernand, V. S. V. and Young, J. Z. The size of the nerve fibers of muscle nerves. Proc. Roy. Soc. Series B, 1951, 139, 38-57.

Galambos, R. and Rupert, A. Action of the middle ear muscles in normal cats. J. Acoust. Soc. Amer., 1959, 31, 349-355.

Gerebetzoff, M. A. Les quatre localizations de l'acétylcholinestérase dans les muscles striés des mammiferes et des oiseaus. C. R. Soc. Bull., 1955, 149, 823-826.

Gomori, G. Microscopic Histochemistry. Principles and Practices, Chicago, Ill.: University of Chicago Press, 1952.

Häggqvist, G. Zur Kenntnis einer doppelten cerebrospinalen Innervation der Skeletmuskeln. Ztschr. f. mikr. anat. Forsch., 1938, 43, 491-508.

Häggqvist, G. Die zwei Formen von zerebrospinaler Skelettmuskelinnervation. Anat. Anz., 1939, Suppl. 87, 74-80.

Klockhoff. I. and Anderson, H. Reflex activity in the tensor tympani muscle recorded in man. Acta oto-laryngol., 1960, 51, 184-188.

Krebs, P. Nervenendigungen im Musculus stapedius. Arch. mikr. Anat., 1905, 65, 704-727.

Malmfors, T. and Wersäll, J. Innervation of the middle ear muscles in the rabbit with special reference to nerve calibres and motor units I. Acta Morphol. Neerlando-Scand., 1960a, 3, 157-162.

Malmfors, T. and Wersäll, J. Innervation of the middle ear muscles in the rabbit with special reference to nerve calibres and motor units II. Acta Morphol. Neerlando-Scand., 1960b, 3, 163-169.

Rexed, B. Contributions to the knowledge of postnatal development of the peripheral nervous system in man. Acta Psychiat. et Neurol., 1944, Suppl. 33, 1-205.

Rexed, B. A modified myelin staining method for paraffin sections. J. Neuropath. and Exp. Neurol., 1948, 7, 462-466.

Ruffini, A. Sur un reticule nerveux spécial et sur quelques corpuscles de Pacini qui se trouvent en connexion avec les organes musculo-tendineaux du chat. Arch. Ital. de Biologie, Bd., 1893, 18, 101-106.

Sherrington, C. A. On the anatomical constitution of nerves of skeletal muscles. J. Physiol., 1894, 17, 211-258.

Sherrington, C. A. Some functional problems attaching to convergence. Proc. Roy. Soc., London, Series B, 1929, 105, 332-362.

Simmons, F. B. Middle ear muscle activity at moderate sound levels. Ann. Otol. Rhinol. and Laryngol., 1959, 68, 1126-1143.

Steinitz, W. Beiträge zur anatomia musculus stapedius. Arch. f. Ohrenheilk., 1907, 70, 45-50.

Tergast, T. Ueber das Verhältniss von Nerven und Muskeln. Arch. f. mikr. Anat., 1873, 9, 36-46.

Torre, M. Nombres et dimensions des unites motrices dans les muscles squelettique relies a des organes de sens. Arch. suisses de Neurol. et de Psychiat., 1953, 72, 362.

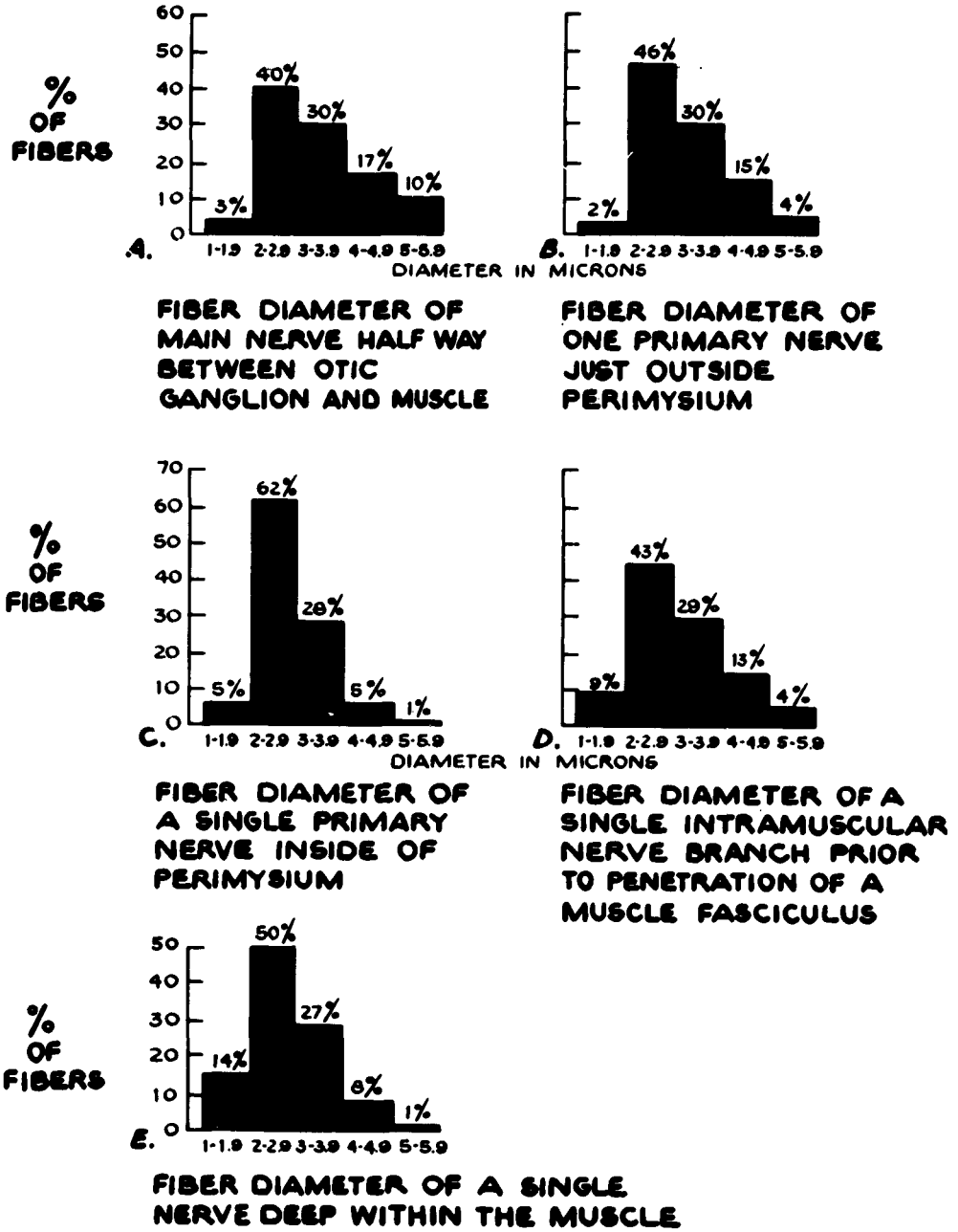

Fig. 1. Diagram of the caliber spectrum at different levels of the nerve to the tensor tympani muscle of the cat.

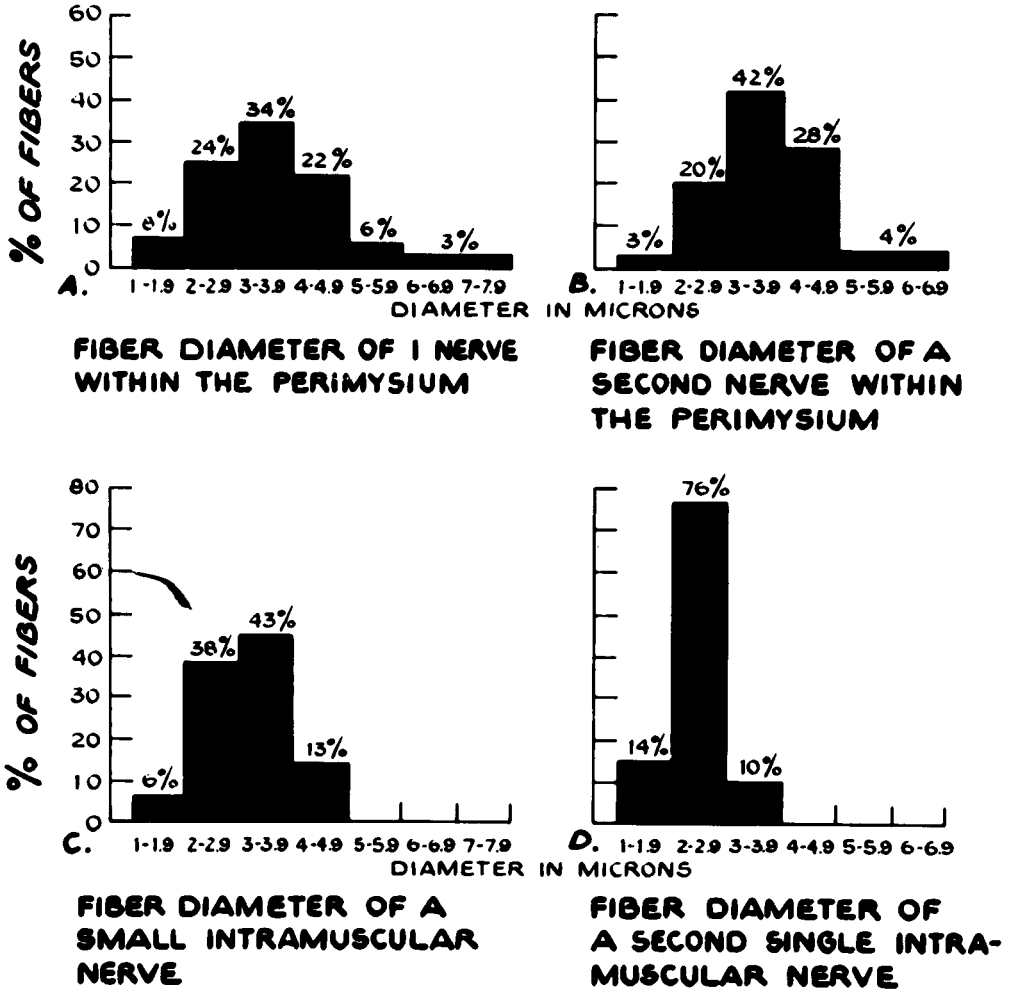

Fig. 2. Diagram of the caliber spectrum of the nerves to the stapedius muscle of the cat.

TABLE 1

INNERVATION RATIOS AND MOTOR UNIT SIZE OF THE TENSOR TYMPANI MUSCLE OF THE CAT

Specimen Number	Nerve Fiber Count		Muscle Fiber Count	N/M Ratio (Innervation Ratio)	Motor Unit Size
		667*			1:7.6+
1	1339**		5070	1:3.8	
		883***			1:5.7++
		636			1:6.3
2	1273		3967	1:3.1	
		853			1:5.7
		488			1:8
3	975		3920	1:4	
		653			1:6
		610			1:6.8
4	1221		4149	1:3.4	
		814			1:5.1
		403			1:7.0
5	806		2881	1:3.5	
		538			1:5.3
		562			1:7.1
Mean	1123		3997	1:3.5	
		752			1:5.3

*50% of total fiber count (if it is assumed that 50% of nerve fibers are efferent).

**Total fiber count.

***66-2/3% of total fiber count (if it is assumed that 66-2/3% of nerve fibers are efferent).

+Motor unit size if assumed that 50% of nerve fibers are efferent.

++Motor unit size if assumed that 66-2/3% of nerve fibers are efferent.

TABLE 2

INNERVATION RATIOS AND MOTOR UNIT SIZE OF THE STAPEDIUS MUSCLE OF THE CAT

Specimen Number	Nerve Fiber Count		Muscle Fiber Count	N/M Ratio (Innervation Ratio)	Motor Unit Size
1	663**	332*	1848	1:2.8	1:5.5+
		444***			1:4.1++
2	630	315	1711	1:2.7	1:5.5
		422			1:4.1
3	477	238	1324	1:2.8	1:5.6
		318			1:4.1
4	911	456	2089	1:2.2	1:6.4
		607			1:3.5
5	862	431	1715	1:2	1:4
		574			1:3
6	749	375	1694	1:2.3	1:4.5
		566			1:3
Mean	715	358	1730	1:2.4	1:4.8
		476			1:3.6

* 50% of total fiber count (if it is assumed that 50% of nerve fibers are efferent).

** Total fiber count.

*** 66-2/3% of total fiber count (if it is assumed that 66-2/3% of nerve fibers are efferent).

\+ Motor unit size if assumed that 50% of nerve fibers are efferent.

++ Motor unit size if assumed that 66-2/3% of nerve fibers are efferent.

STUDIES ON THE STRUCTURE AND FUNCTION OF THE MIDDLE EAR IN BATS

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The process whereby animals appreciate and locate objects within their environment by the production of sounds and the subsequent perception and analysis of echoes is called echolocation. In the past twenty-five years this has become a well established phenomenon, known to be utilized by many different vertebrates. Echolocation is highly developed among Chiroptera, especially the small, essentially blind Microchiroptera. The orientation sounds emitted by these bats are controlled by laryngeal mechanisms and a rather constant pulse duration, frequency pattern and intensity is characteristic of each species. The frequency range ordinarily falls between 150 and 12 kc/sec. The intensity of these sounds, when measured at distances of 3 to 10 cm from the bat's mouth, averages less than one dyne/cm^2 in the so-called "whispering" bats to as high as several thousand dynes/cm^2 in "loud" bats. Pulse repetition rates may be as high as 200 per second and the pulse duration ranges from less than 1 msec to about 80 msec. On the other hand, Megachiroptera are large visual animals and, except for one genus, do not emit orientation sounds. The one exception, Rousettus, can orient either visually or acoustically, the latter being accomplished by the production of clicks made with the tongue; the predominate components of these sounds are below 20 kc/sec.

The above information, the various theories of echolocation and the details with respect to species variation are found in the recent works of Griffin (1958), Novick (1958a; 1958b), Möhres (1953), Pye (1961) and many others. These investigators have given us considerable information about the orientation sounds emitted by bats. Since the studies of Wever and Vernon (1961a; 1961b) suggest that the auditory sensitivity of a given species is greatest over the frequency range of the emitted pulse, it is interesting to compare the morphology of the ear among the different forms and to ask what role the various parts play in ultrasonic perception. With respect to middle ear physiology and hearing mechanisms there are several questions to consider. (1) How

is the middle ear of bats adapted to permit effective transfer of ultrasonic vibrations across the ossicular chain? (2) How is the ear protected from the continuous bombardment of intense sounds to which it is subjected, especially the sounds of the emitted pulses? (3) How can bats hear faint echoes within milliseconds after the emission of an intense pulse, or perhaps even during the time when the same pulse or a subsequent one is being emitted?

The answers to these questions are not clear, and not until a detailed series of morphological and physiological investigations are carried out is there any hope of passing beyond current speculative stages. Preliminary studies in this direction have revealed certain structural features which may have some bearing on hearing mechanisms in bats. It is these features which I would like to consider and discuss.

The role of the bat middle ear in ultrasonic perception can be evaluated by comparing its structure with that of other small animals, especially the phylogenetically related insectivores (Cryptotis, Scalopus and Erinaceus). Such a comparison reveals the following basic differences. (1) The annulus fibrosus of the bat tympanic membrane contains a "cavernous plexus" of blood vessels which is not known to occur in other mammals. (2) The anterior process of the bat malleus is fused to the tympanic bone by an osseous ankylosis; in the insectivores this union is a looser fibrous ankylosis. (3) The architecture of the malleoincudal joint suggests that the joint is capable of being more closely coupled and locked in bats than in insectivores. (4) Bat ossicles are very delicate structures and possess discrete ligaments which anchor and suspend them; a comparable arrangement is not apparent in insectivores. (5) The combined mass of the middle ear muscles is from two to over 50 times larger than in other mammals, relative to the size of the structures on which they act.

Before considering these points another comparison can be made; this time with respect to middle ear structure in non-echolocating fruit bats and echolocating Microchiroptera; a similar comparison of structure can be made of bats which predominantly utilize frequencies above 50 kc/sec as compared with those which emit frequencies below 50 kc/sec. These comparisons reveal the following: (1) The tympanic membrane area is smallest in echolocating bats whose pulses are predominately above 50 kc/sec. The area is largest in non-echolocating Megachiroptera and intermediate in size in bats whose pulses are below 50 kc/sec. (2) The same size relationship holds for the ossicles; those bats which utilize the higher frequencies tend to

have smaller ossicular mass. Furthermore, these size relationships show no definite correlation with the size of the animals. (3) Dissection of one Rousettus, and examination of the skulls of other Megachiroptera suggest that the middle ear muscle mass, relative to the size of the structures on which they act, is greater in echolocating Microchiroptera than in non-echolocating fruit bats. The relative muscle mass also appears to be larger in Microchiroptera than in the primitive, echolocating Rousettus.

The above comparisons of middle ear structures among various insectivores and bats strongly suggest that the size of the tympanic membrane and ossicles and the nature of the malleoincudal articulation are important factors in the transmission of ultrasonic vibrations across the middle ear. On theoretical grounds these findings are reasonable. The significance of the cavernous tissue is not known; it is possible that it influences the transmission of sound across the middle ear by varying the tension of the tympanic membrane. On the other hand, these vascular channels may be a protective mechanism which keeps the thin membrane from drying out. The function of the middle ear muscles is more difficult to analyze. Besides being relatively larger in echolocating bats than in non-echolocating bats and other mammals, these muscles also vary in the richness of their nerve supply. Particularly striking is the number of nerve fibers in the stapedius muscle of the Molossidae. With respect to these findings, we can now turn to the problem of how the bat's ear is protected from the intense sounds emitted during echolocation.

Hartridge (1945) has suggested that the bat's ear needs protection from the intense outgoing signal if the ear is to be in a full state of sensitivity when the faint echoes return; as he suggests, the large middle ear muscles could provide this protection if their contractions were synchronized with the cries. The magnitude of these muscles, which implies high rates of activity, is generally said to support this theory; further support of Hartridge's theory is based on evidence that reflex activity occurs in response to tones with frequencies falling within the range of the ultrasonic pulses (Galambos, 1942; Wever and Vernon, 1961a).

Griffin (1958) has raised two "serious" questions with respect to this hypothesis. The first question is whether the muscles are able to relax in the brief time available between the pulse and the echo. He has regarded this as a speculative possibility, but not a very likely one. It must be pointed out, however, that if the muscles of the larynx are capable of controlling the emission of pulses with

rates as high as 200 per second, then there is no reason to believe that the middle ear muscles could not act at a similar rate; perhaps the physiology of bat muscle is different from that of other mammalian muscle to which Griffin has made reference. It is interesting in this respect to note that Revel (1962) has shown that the sarcoplasmic reticulum in the cricothyroid muscle of the bat larynx is modified along lines comparable to that of other vertebrate muscles known to be extremely fast acting. If similar ultrastructure could be demonstrated in bat middle ear muscles it would lend further support to Hartridge's hypothesis.

On the other hand, Hartridge's theory seems to imply that the muscles must be relaxed when the echo returns to the ear. In many cases, however, the returning echo must overlap with either the same cry or a subsequent one. In a recent paper (Henson, 1961) I have suggested that perhaps the muscles "tune" the ear to a useful range of frequencies, implying that contraction of the muscles may not hinder perception of ultrasounds, but may actually aid it by attenuating sounds not useful in echolocation. It would seem to be a definite advantage to reduce perception of lower frequencies, especially the sounds produced by beating wings and also those created by air rushing past the external ear. If the muscles could act in this manner, then a rapid contracting and relaxing mechanism would not be particularly useful; more useful would be muscles that could exert a tonic influence, much like the postural muscles of our bodies. This could account for the large size of the muscle just as well as the theory suggesting high rates of reflex activity. It is interesting in this respect to note that Erulkar, Shelanski, Whitsel and Geesey (1962) have reported the presence of a dual muscle fiber system in the tensor tympani of the cat, both "fast" and "slow" fibers being present. I have noted the presence of what may be a similar situation in the stapedius muscle of Eumops. Thus, perhaps some fibers exert a tonic influence while others are concerned with reflex activity.

The second question raised by Griffin against Hartridge's hypothesis was whether the muscles are actually protective against high frequencies. It has already been pointed out that reflex activity of the muscles can be stimulated by presenting the ear with sounds falling within the frequency range of bat pulses. The amount of attenuation, however, does not appear to be very significant for the upper frequencies of these pulses (Wever and Vernon, 1961a). Thus, the idea that these muscles provide adequate protection against the intense cries does not seem to be well-substantiated. Since these muscles show a very rich innervation and are probably capable of being delicately

controlled, and since low frequencies are much more effectively attenuated than high ones, the idea that these muscles act as a tuning mechanism, as well as being protective in function, is particularly attractive. Functioning of the ear in this manner could also help explain the resistance of echolocation to jamming. This, however, is only speculation. Furthermore, this speculation does not help to answer the question of how the ear is protected from the intense ultrasonic cries.

Since a temporary disconnection of the receiver (ear) from the outgoing signal by means of middle ear muscle contractions is not entirely acceptable other possible mechanisms should be examined. In this respect there are several physiological and anatomical points which suggest that bats may not hear their own pulses at a distance of several centimeters in front of the bat. In the first place many bats beam their pulses in a forward direction, especially those which emit sounds through their nose. Also the ears of bats are highly directional and certain species have nasal and facial appendages which could possibly shield sounds from the external ears, at least to some extent.

Since many mammals can detect their own vocalizations by bone conduction it is necessary to determine if this occurs in bats, and to what degree. There are several interesting anatomical features which suggest that evolutionary changes in the ear region have been directed toward acoustic isolation of the cochlea. First is the accumulation of fat deposits between the larynx and the cochlea. Second, the cochlea is not firmly joined to the adjacent skeletal elements, and third, the space between adjacent elements is filled with fat or venous channels. The presence of a "loose" cochlea is seemingly a feature characteristic of many lower mammalian forms, especially shrews, and the idea that it is merely an expression of a primitive condition can not be disregarded. In primitive mammals the petrosal, squamosal and mastoid elements tend to be separate; in higher mammals these elements are united to form the composite temporal bone. When the elements are separate they are usually strongly united with one another by dense fibrous connective tissue. I do not know of any mammals other than bats where this fibrous tissue is replaced by fat. Furthermore, preliminary studies on non-echolocating fruit bats (Pteropus) and on the primitive echolocating Rousettus do not show a state of cochlear isolation similar to that seen in the echolocating Microchiroptera.

If the cochlea of echolocating bats is acoustically isolated, as anatomical evidence seems to suggest, then this could prove to be a unique mechanism whereby the receiver (cochlea) is permanently

disconnected from the outgoing signal (at least to some degree), rather than being temporarily disconnected by middle ear muscle mechanisms. This could give a reasonable answer as to how the bat ear is protected from the intense cries emitted during echolocation; it could also help to explain how bats hear faint echoes so soon after emitting an intense cry, or at the same time that the cry or a subsequent one is being emitted.

SUMMARY

Reference has been made to anatomical features in the middle ear of bats which may have some bearing on the theories of echolocation and on certain auditory phenomena. It is suggested that the small size of the middle ear structures and the nature of the ossicular articulations play an important role in ultrasonic perception. It is also suggested that the middle ear muscles tune the ear to an upper range of frequencies by attenuating low frequency background noises. The presence of what appears to be an acoustically isolated cochlea may be important in providing protection against the intense ultrasonic pulses and may also explain how bats can hear faint echoes within milliseconds after the emission of an intense pulse. Studies are currently being undertaken in the hope of determining the physiological significance of the anatomical features cited in this study.

REFERENCES

Erulkar, S. D., Shelanski, M. L., Whitsel, B. L., and Geesey, C. N. A dual fiber system in the tensor tympani of the cat. Fed. Proc., 1962, 21(2), 357.

Galambos, R. Cochlear potentials elicited from bats by supersonic sounds. J. Acoust. Soc. Amer., 1942, 14, 41-49.

Griffin, Donald R. Listening in the Dark. Yale University Press, 1958.

Hartridge, H. Acoustic control in the flight of bats. Nature, London, 1945, 156, 490-494.

Henson, O'Dell W. Some morphological and functional aspects of certain structures of the middle ear in bats and insectivores. Univ. Kansas Sci. Bull., 1961, 42(3), 151-255.

Möhres, F. P. Uber die Ultraschallorientierung der Hufeisennasen (Chiroptera-Rhinolophinae). Zeitschr. f. vergleichende Physiologie, 1953, 34, 547-588.

Novick, A. Orientation in paleotropical bats. II. Microchiroptera. J. Exp. Zool., 1958a, 137, 443-462.

Novick, A. Orientation in paleotropical bats. I. Microchiroptera. J. Exp. Zool., 1958b, 138, 81-154.

Pye, J. D. Echolocation by bats. Endeavour, 1961, 20, 101-111.

Revel, J. P. The sarcoplasmic reticulum of the bat cricothyroid muscle. J. Cell. Biol., 1962, 12(3), 571-588.

Wever, E. G. and Vernon, J. A. The protective mechanism of the bat's ear. Ann. Otol. Rhin. and Laryngol., 1961a, 70, 1-13.

Wever, E. G. and Vernon, J. A. Hearing in the bat, Myotis lucifugus, as shown by the cochlear potentials. J. Aud. Res., 1961b, 2, 158-175.

INNERVATION OF THE TENSOR TYMPANI BY THE TYMPANIC NERVE*

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Colonel Bach, Dr. Odell, Major Fletcher and members of this seminar: Although we have worked on the middle ear muscles for some time a special interest was kindled by certain aspects of present day middle ear operations performed for the relief of deafness due to otosclerosis. Routinely, in the course of many forms of this operation, the stapedius muscle is cut thus removing a structure that is generally conceded to serve as a mechanism for protecting the inner ear from loud sounds. The reason the stapedius is cut is that the stapes is removed so that it can be replaced by a prosthesis of some sort which is inserted into the excavated oval window where the diseased footplate has been removed.

At a recent meeting I heard a surgeon say that he removed the stapes but left the stapedius muscle attached. This raised the interesting question concerning to what the remaining stapedius is attached. His answer was that the muscle remained attached to the lenticular process of the incus. This I did not believe was possible so I decided to go back and look over some of the human temporal bone sections we have to make a study of this. I thought that perhaps a number of photomicrographs that showed the attachment of the stapedius would be helpful. On re-examination of the anatomical situation, however, I found nothing that had not been observed and reported before. The stapedius is firmly attached to the neck of the stapes. Very close examination does show that a few connective tissue fibers surrounding the tendon do surround the incudo-stapedial articulation and appear capable of remaining attached to the lenticular process of the incus, but any prosthesis that is put over the lenticular process would abolish these

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and it is not likely that they could withstand the pull of a muscle contraction anyway.

This re-examination, however, renewed an interest in the intricate action of these two middle ear muscles. Some of the material available for the above review of stapedius muscle attachment was from our fairly extensive series of serial sections from fetal temporal bones. These seemed ideal for a study of the nature of nerve supply to the two muscles. Such a study appeared particularly appropriate because recent investigations have shown the muscles to have a complicated form of action for which the existing textbook descriptions of their innervation do not seem adequate.

Wever and Vernon (1) did an extensive study of the control the middle ear muscles exert on sound transmission and demonstrated a considerable complexity in the muscle activity. They described random fluctuations of muscle tonus and occasionally, under certain degrees of stimulation, sometimes an increase and sometimes a decrease in the tension of the muscles. They also demonstrated that this increase or decrease of tension could produce an improvement in transmission. Of particular interest was their observation that the tensor tympani muscle exhibits a regular alteration in the form of action according to stimulus intensity. They felt that the contractions which sometimes produce an increase in response were not contractions of the muscle as a whole but possibly only of its medial portion. They also described that as the stimulus intensity is raised the contractions grow stronger during a certain phase of response and, as the threshold for producing a loss in response is reached, the whole muscle undergoes a vigorous action. Their final statement says, "these results indicate that the neural patterns governing the reflex action of the two muscles are highly complex in nature, and that the pathways and connections differ according to the frequency of stimulation."

The neural innervations of these muscles, as generally described, however, are not as complex as the above experiments would indicate. The textbooks merely state that the stapedius muscle is innervated by a branch of the facial nerve and that the tensor tympani is innervated by the mandibular branch of the trigeminal nerve through the otic ganglion. The latest studies on the innervation of the tensor tympani and stapedius (Guerrier, Y. and Bolönyi, F. (2); Malmfors, T. and Wersäll, J. (3) and Portmann (4)) have resorted to dissection of the muscles from the animal followed by histological study; a method, of course, which destroys evidence of the source of innervating fibers. The investigations were carried out in order to gather information on the types of

nerve endings, but results have been conflicting. In order for a resting tonus to be maintained there should be some sort of kinesthetic mechanism, but there are observations that suggest the existence of sensory endings within the muscles (Malmfors, T. and Wersäll, J. (3)) and others that find no evidence for such (Blevins (5)).

Most of our present day information seems to stem from Politzer (6) in 1861, who first demonstrated through intracranial stimulation the separate innervation of these two muscles. He demonstrated that stimulation of the trigeminal nerve activated the tensor tympani and that stimulation of the facial nerve activated the stapedius but he did not explore all the other possibilities. The purpose of the investigation reviewed here was to trace the innervation of these muscles in human histological material in order to see if perhaps the pattern of innervation is not more complex than has been stated.

MATERIAL

We have in our hostological collection a number of serially sectioned temporal bones from human fetuses ranging from 10 weeks (34 mm) to the newborn. In general, the best preserved material is that from a fetus which is advanced enough to be well developed and yet still have a cartilaginous otic capsule which allows the fixative to penetrate easily. For this study we selected two 15-week (105 mm) fetuses that were extremely well preserved and which we were able to section serially in their entirety. The ears of these fetuses were treated in the usual manner, embedded in celloidin and sectioned at 20 microns. Routinely, for these smaller bits of tissue, every fifth section is mounted to make a series, and for this particular study every fifth section of one series was stained with hemotoxylin and eosin and mounted. In addition, another series was stained in Bodian's silver stain and another in Mallory's trichrome stain. With the various series stained differently it was possible to use the advantages of the various stains in following the small nerve fibers from section to section.

The sectioning of these fetuses was not always in the same plane and the one which presented the information to the best advantage has been used for presentation in this study.

Following the observations which were made on the 15-week fetuses, the temporal bone from a four month old full term infant was examined grossly to confirm the observations. This temporal bone was preserved in ammoniated alcohol for subsequent removal of the muscles. In the removal of the muscles from this fresh temporal bone the

course of the intratympanic nerve supply to the middle ear muscles was examined.

RESULTS

The plane of section of the 15-week fetus reported here is such that it begins at the posterior portion of the temporal bone where the facial nerve makes a 180 degree bend as it courses across the middle ear through the stylomastoid foramen and then peripherally through the parotid gland. This allows the muscles to present themselves in the serial sections one at a time; first, the stapedius and then the tensor tympani.

The procedure followed in this instance was to make a graphic reconstruction of the anatomical structures as they appeared in histologic study of the serial sections. Each section as it comes from the microtome knife is, after staining, placed upon a slide and studied with the microscope. This is, then, as though one were looking with a microscope down upon the structure and the graphic reconstruction is presented so as to show the structures as they would appear if one looked at the side of the whole unsectioned block.

The figure presents the graphic reconstruction of this temporal bone. On the left hand side of the figure are section numbers. It was not always possible to keep the orientation of the sections the same so that the continuous diagrammatic representation of a nerve fiber is not absolutely in proper perspective across the section. The one other aspect which is totally missing from this kind of presentation is that of depth. The sections being cut at 20 microns and numbered, however, allow the vertical scale to be quite accurate in its presentation.

The nerve fibers were all traced out and placed upon this drawing. The structures which are presented consist of Reichert's and Meckel's cartilage, the carotid artery, the two intratympanic muscles and the nerves which course through this region. All of the other structures shown in the photomicrographs and appearing under the microscope have been omitted from the diagram for the sake of clarity.

The first and obvious structure to follow is that of the facial nerve which, as seen in section 65, is cut across its bend. Also in this section appears the region of what is the beginning of the stylomastoid process which extends down into the hyoid region in the form of Reichert's cartilage. From this region also arise two other nerves; one is the chorda tympani and the other is Arnold's nerve. These are

traced in their entire length throughout the serial sections but will not be described since they are not important for the present observations. The facial nerve is seen to proceed to the left and to divide into its various branches within the parotid gland which extends throughout most of this series. On the right hand side the facial nerve continues and is followed in its uninterrupted course through to the geniculate ganglion and hence on into the brain stem.

The first muscle structure which is encountered is the stapedius, and as reported previously in the literature, only one nerve supply to this muscle could be found. That comes from the facial nerve as indicated. Throughout the entire length of the muscle there is no other indication of nerve supply from any other source or from any other branch of the facial nerve.

As the sections continue the tensor tympani muscle is encountered at its point of insertion into the manubrium of the malleus. The orientation of this particular series is especially advantageous because the sections cut the muscles across the longitudinal axis so as to show the nerve fibers penetrating the muscle along its length.

The first penetration of a nerve fiber comes in section 176. As shown in the reconstruction, this, as well as the next few groups of fibers, although extending down to the otic ganglion through the lesser superficial petrosal to connect eventually with the mandibular branch, arise mainly from the tympanic plexus which comes from the inferior ganglion of the glossopharyngeal nerve. This was traced in considerable detail and, although these small twigs of nerve fibers rest within the mesenchyme of the middle ear and not in grooves of the cartilaginous promontory, they can easily be followed, and the connection with this ganglion is clearly indicated. It takes a considerable number of observations back and forth on these sections but there is little doubt that the connections as presented in the diagram exist.

Section 245 is beyond the vestibule and the only portion of the cochlea, aside from the nerve fibers, that is visible is the basal hook. The tensor tympani muscle with its enclosed tendon appears between Meckel's cartilage and the cartilage of the otic capsule. The geniculate ganglion is also present. A tiny branch going to the tensor tympani from the tympanic nerve is present and also the lesser superficial petrosal connecting with the tympanic nerve and the otic ganglion in the manner shown in the graphic reconstruction (Fig. 1). In this section the cut is made at about the center of the tensor tympani, and, as

shown in the graphic reconstruction, the innervation from this point all the way to the insertion has been connected with the tympanic nerve.

In section 300 the tensor tympani muscle has thinned down; there is now very little evidence of the tendon, the middle ear cavity has narrowed done to become the orifice to the Eustachian tube, and the lesser superficial petrosal is still present. Directly above the tensor tympani muscle is the first appearance of the otic ganglion. The lesser superficial petrosal is headed for the otic ganglion and the nerve fibers that are seen entering the tensor tympani are coming directly from the otic ganglion and make no connection with the lesser superficial petrosal.

Finally, we approach the very extreme point of origin of the tensor tympani muscle. Section 325 shows Meckel's cartilage and the apex of the cochlea which is now nearly mid-modiolar. The carotid artery with the divided carotid sinus nerve can be seen. The otic ganglion appears and the lesser superficial petrosal which we have followed throughout the last two figures now joins the ganglion. Still very small nerve fibers enter the muscle, these fibers having come directly from the otic ganglion as shown in the graphic reconstruction.

As stated in the paragraph describing the preparation of the temporal bones those of a four month old full term infant were placed in ammoniated alcohol immediately upon removal at autopsy, in order to prepare this temporal bone for the subsequent special staining of nerve endings within the middle ear muscles. During the process of removal of the tensor tympani and the stapedius muscle from this temporal bone, the course of the tympanic plexus nerves entering the canal for the tensor tympani was closely observed. These nerve fibers were seen to course typically over the promontory to go beneath the tensor tympani canal to form the lesser superficial petrosal which is headed for the otic ganglion. However, it was perfectly clear as the canal was opened, that there were small fibers which passed through the canal and entered the tensor tympani muscle. Earlier investigators (7) have reported the tympanic nerve as entering the semi-canal for the tensor tympani but not as innervating this muscle. It appears from this observation and those of the other fetuses that the tympanic plexus in passing over the promontory to terminate in the lesser superficial petrosal, gives off branches to the insertion end of the tensor tympani.

CONCLUSIONS

The innervation to the stapedius muscle appears as has always been stated: a branch from the facial nerve. In the present observations no other pattern of innervation could be observed to the stapedius;

the one branch coming from the facial to the stapedius in a region where they approximate each other seems to be the only source of nerve supply for this muscle.

It appears, however, from the observations described above that the innervation of the tensor tympani is extremely more complex than has been previously stated. The earlier statements described the tensor tympani muscle as being innervated by the mandibular branch of the trigeminal through the otic ganglion. There is apparently also a connection with the tympanic plexus which gives off a pattern of innervating nerve fibers to the insertion end of the muscle from a point of connection with the lesser superficial petrosal nerve connecting between it and the otic ganglion.

Because of the nature of the nerves involved in innervating the tensor tympani as described here, it is not possible to speculate with any confidence as to the role that might be played by these various nerve branches. There are several possibilities. They could be sympathetic fibers to the blood supply of the muscle, but examination under high power did not show these fibers terminating on blood vessels. The various branches could be sensory or they could be motor and it is not possible, with the present observations, to make a definite conclusion.

Further research should follow in order to ascertain the significance of this pattern of innervation. The end plates in both the insertion and origin end of the muscle should be examined and compared, not only as to the difference among them but to distinguish, possibly, between motor and sensory endings. Electrophysiological studies of nerve muscle relationships should reveal more concerning the sensory and motor connections to the muscle. At the present, however, the reported observations merely indicate that the innervation to the tensor tympani is considerably more complex than hitherto described, and suggests an explanation of the extremely complex physiological response that this muscle demonstrates.

REFERENCES

1. Wever, E. G. and Vernon, J. A. The control of sound transmission by the middle ear muscles. Ann. Otol. Rhinol. Laryngol., 1956, 65, 5-14.

2. Guerrier, Y. and Bolönyi, F. L'innervation du muscle Marteau. Ann. Otolaryngol., 1948, 65, 94-96.

3. Malmfors, T. and Wersäll, J. Innervation of the middle ear muscles in the rabbit with special reference to nerve calibers and motor units. Acta Morphologica Neerlando-Scandinavica, 1960, 3, 157-169.

4. Portmann, M. Le muscle du Marteau: son innervation. Rev. Laryngol. Rhinol. Otol., 1949, 70, 534-546.

5. Blevins, C. E. Studies on nerve muscle relations of the middle ear. Ph. D. Thesis, Univ. of California Library, 1962.

6. Politzer, A. Beitrage zur physiologie des gehororgans, sitzungsber. Akad. Wiss. Wien, math.-nat. Cl., Abt. 2, 1861, 43, 427-438.

7. Rosen, S. The tympanic plexus. Arch. Otolaryngol., 1950, 52, 15-18.

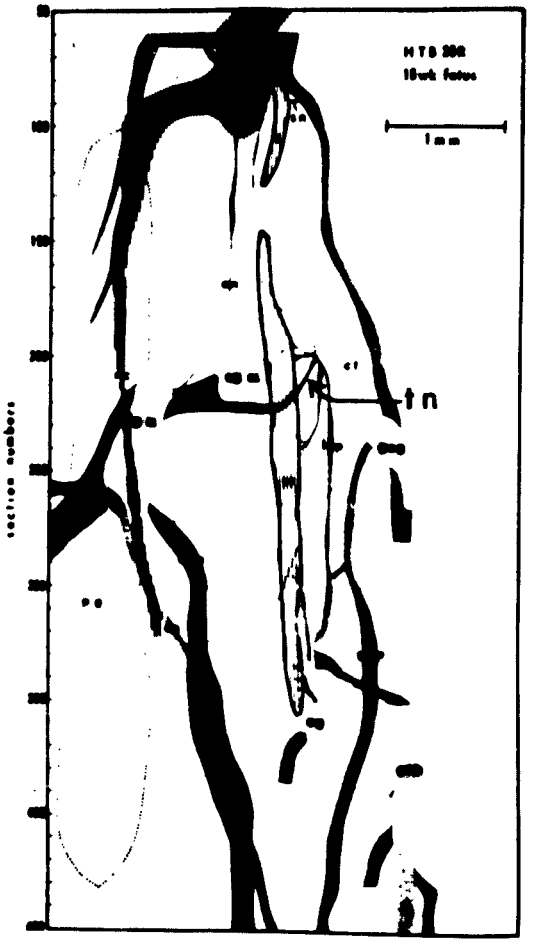

Fig. 1. Graphic reconstruction of the nerves of the fetal temporal bone. The tympanic nerve is seen to arise from the inferior ganglion of the glossopharyngeal and innervates the insertion end of the tensor tympani before becoming the lesser superficial petrosal. From the otic ganglion are fibers which innervate the origin end of the tensor tympani. Symbols are as follows: fn: facial nerve; sn: stapedius nerve; s: stapedius muscle; an: auricular (Arnold's) nerve; rc: Reichert's cartilage; ct: chorda tympani; igIX: inferior ganglion of glossopharyngeal; sgIX: superior ganglion of glossopharyngeal; tn: tympanic (Jacobson's) nerve; lsp: lesser superficial petrosal; geg: geniculate ganglion; tt: tensor tympani; gn: glossopharyngeal nerve; csn: carotid sinus nerve; pg: parotid gland; ca: carotid artery; gsp: greater superficial petrosal; og: otic ganglion; ln: lingual nerve; gag: gasserian ganglion of trigeminal (V).

AN ANALYSIS OF THE MIDDLE-EAR-MUSCLE ACOUSTIC REFLEX OF THE CAT*

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INTRODUCTION

It is common knowledge that the sensitivity and strength of the middle-ear-muscle acoustic reflex vary according to the stimulus frequency and intensity. The first portion of this paper discusses these and other stimulus parameters and describes a series of experiments which show this variation to be directly related to the presumed amplitude and place relations of the cochlear partition traveling wave, and a maintenance of these spatial relations throughout the central nervous system portions of the reflex arc.

It is also known that a wide variation in reflex responses exists which is not directly related to the stimulus frequency or intensity, but to other factors. The second portion of this paper describes a number of these additional factors, some attributable to alterations in the reflex loop, others partially independent of the loop.

METHODS

The responses of the middle ear muscles to various sound stimuli were observed in approximately 75 awake cats, using electrodes permanently implanted on the round window and in the muscles themselves. Both qualitative and quantitative estimates of reflex activity were obtained by a variety of techniques, most of which have been previously described and will be only briefly reviewed here (1, 2). Figure 1 serves as an illustration for these.

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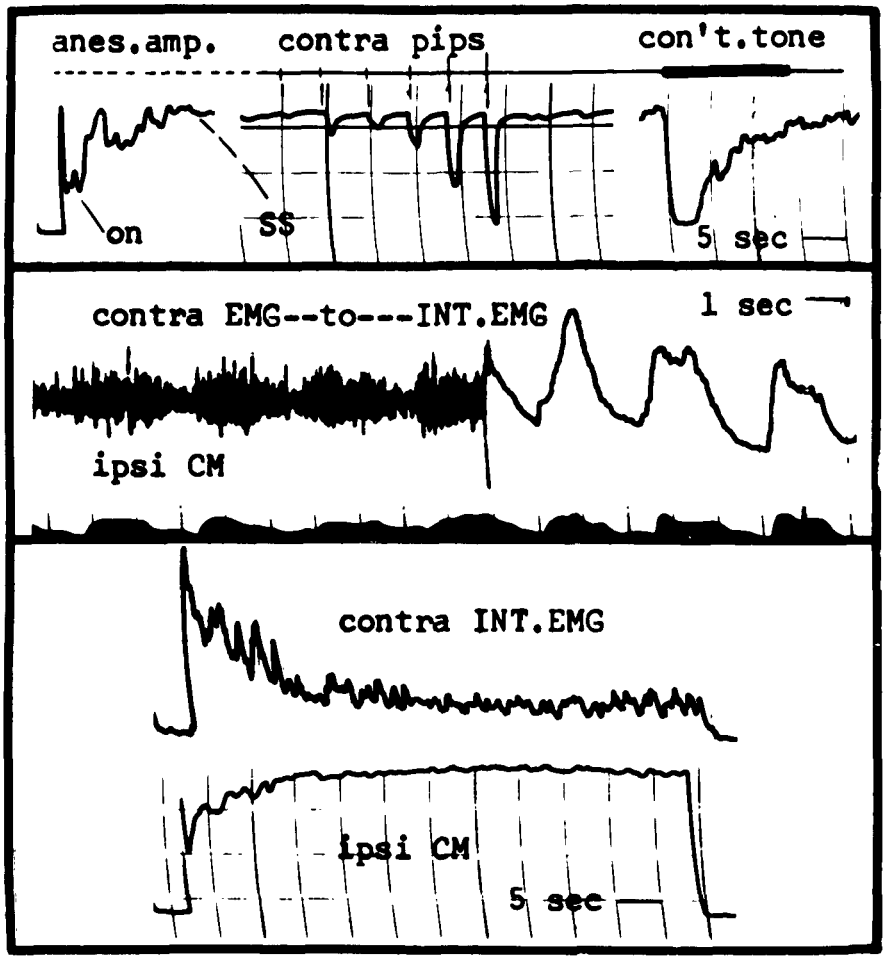

Fig. 1. Measurement of Middle Ear Muscle Responses in the Awake Cat: The top section is an inkwriter record of a rectified and logarithmically amplified cochlear microphonic response to a stimulus tone of constant intensity, turned on at the left-hand side of the first segment. The attenuating contractions shown in the 2nd and 3rd segments are responses induced by additional contralaterally presented tones. In the middle section, the top trace shows an EMG, then the integrated EMG response to contralateral stimulation by tone pulses whose cochlear microphonic (ipsilateral) responses appear in the lower trace. In the bottom section, responses--arranged as in the middle section--to a more intense continuous tone are shown, primarily to point out the close correspondence of the two measurement techniques. In this, and all inkwriter records shown, each line division on the record paper equals 7-1/2 db of cochlear microphonic.

1. The attenuating effects of muscle contraction on transmission of sound through the middle ear are reflected in the amplitude of the cochlear microphonic measured at the round window. The total attenuation due to muscle contraction can be measured by first presenting a stimulus to the awake cat (reflex active) and then repeating the stimulus after de-activating the reflex with Nembutal anesthesia. The difference between the cochlear microphonic amplitudes of the two measures has been shown to be the amount of reflex attenuation. The reflex attenuation of cochlear microphonic amplitude to a tone stimulus is shown by the inkwriter-recorded amplitudes in Figure 1 (top section). The reflex-activating stimulus was introduced at the left-hand side of the record and remained on throughout. Reflex activity was greatest immediately after onset, then gradually diminished to a steady, less-attenuating level of response. The amplitude of cochlear microphonic produced by the same stimulus with the animal anesthetized is shown by the dashed line.

2. The second and third portions of the top section of Figure 1 illustrate reflex responses initiated by contralateral stimulation, using the ipsilateral cochlear microphonic as a "carrier," amplitude modulated first by a series of tonal bleeps of increasing intensity (10 db steps), and then by a tone with a longer duration.

3. Mono- and bipolar-electrode EMG potentials were measured for ipsi- and contralateral reflex stimuli through implantations in either or both middle ear muscles. These potentials were observed by oscilloscope and by inkwriter recordings such as those shown in the remainder of Figure 1. In the middle section, EMG, and then a rectified, time-integrated stapedius response to contralaterally presented pulsed tone are shown in the upper trace. The lower trace is the cochlear microphonic response for the same tone in the stimulated ear. The bottom section of Figure 1 shows the same contralaterally induced response for a continuous tone--upper trace: integrated EMG, lower trace: ipsilateral microphonic.

In support of and in addition to the electrical measurements, ten cats of the series were trained to respond to tone at threshold levels. Four- or five-frequency audiograms were obtained both before and after electrode implantation. Such implantations did not alter threshold acuity.

RESULTS--PART I

It has been previously mentioned that the middle-ear-muscle reflex can be activated by any sound stimulus, provided its intensity

is great enough, and its frequency within the animal's acuity range, * but that a lower-frequency sound will produce a more sustained contraction than will a similarly intense sound an octave or more higher. This latter relationship is shown for one animal in the solid line of Figure 2. A less well known relation is indicated by the dashed line in this figure. Here, the relative sound intensity required to barely produce, but not sustain, a reflex contraction is shown to decrease as frequency increases. While the absolute values for both sets of measurements in Figure 2 vary widely from cat to cat, the direction of slope for each is constant, as is the relation between them. The possible reasons for these slopes and interrelations are the subject matter of the experimental observations which follow.

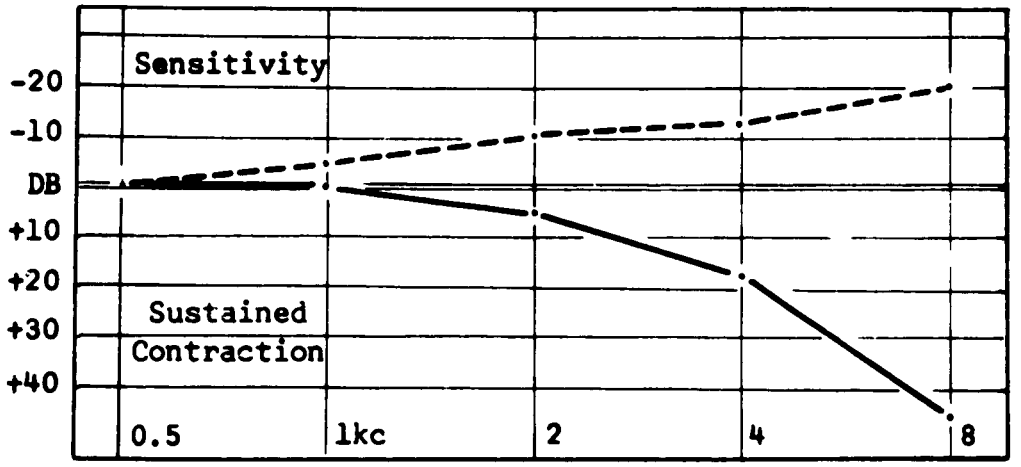

Fig. 2. Relative Sensitivity and Duration of the Acoustic Reflex Contraction: The solid line indicates the ability of the muscles to maintain a contraction to a continuous tone of abscissa frequency. The method illustrated in Figure 1, top section, 3rd segment was employed, i.e., relative intensity required to maintain equal attenuation of the carrier tone, measured 10 sec after introduction of the test tone to the contralateral ear. The dashed line--also a contralateral excitation--indicates the relative sound intensity required to produce a minimal unsustained EMG response.

For purposes of description and analysis, the reflex response can be divided into two levels of activity. The term "on" response

*Reflex responses for frequencies between 0.2 - 32 kc have been observed in this study. Most observations were limited to 0.5 - 8 kc for technical reasons. Stimulus intensities, unless otherwise mentioned, were well below 80 db SPL.

will be used to denote reflex response occurring immediately after a stimulus is introduced, and can be qualitatively equated to the dashed line in Figure 2. The term, steady state (SS), can be equated to the solid line, and will be used to denote a level of reflex response occurring later in time when the "on" response (about 0.3 sec and more) after transition to an equilibratory level of activity has begun or is complete. (See Figure 1, top section, first portion, for example.) While both "on" and SS are principally related to stimulus frequency and intensity, the sensitivity of the "on" response is also a function of two variables--stimulus rise-time and short-term duration--not directly affecting the SS level of the reflex. These will be described first.

Figure 3 shows the relation of "on" sensitivity to stimulus duration. For durations substantially less than 100 msec, sensitivity decreases proportionately until about 5 msec. Reflex responses for shorter durations of stimulus were obtainable from only 3 of the 10 ears represented in the mean of Figure 3. The measurements for this figure were obtained with regularly repeated bursts of wide-band noise having a rise-time of less than 0.1 msec.

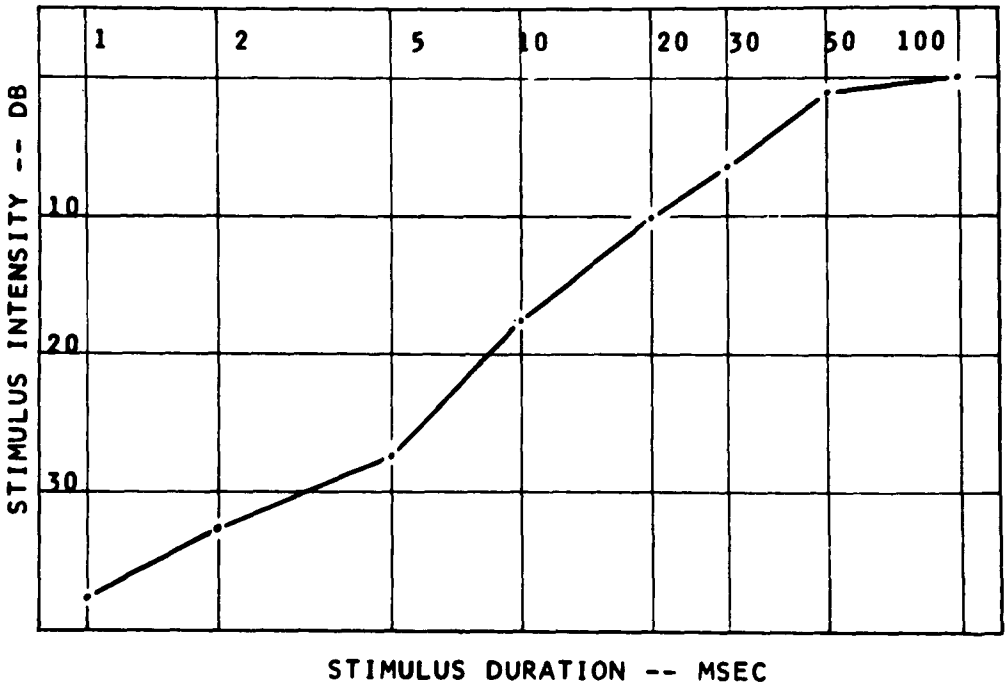

Fig. 3. "On" Response Sensitivity as a Function of Stimulus Duration: The ordinate values are mean EMG measured changes in the intensity of a wide band noise (rise-time less than 0.1 msec) for 10 ears required to compensate for the abscissa-indicated stimulus durations.

As anticipated, the more abrupt the onset of a stimulus, the more sensitive the "on" response. The difference in sensitivity between a rise-time of less than 0.1 msec and one of 100 msec was about equal to a 10-db change in stimulus intensity. This difference was equal (± 5 db) at all frequencies between 0.5 - 16 kc. In Figure 11, the comparative muscle responses for the two stimulus rise-times can be noted. Duration of each pulse was 2 sec. (It is also apparent that the effect of rise-time was not confined to the early portion of the reflex response. The prolonged difference may be attributed, at least in part, to temporal effects already mentioned.)

Intermediate stimulus rise-time rates were not studied in direct relation to change in "on" sensitivity, or the possible frequency-related gradient which was not apparent when comparing very long and very short rates. However, a qualitative comparison obtained by equating decrease in stimulus-response latency with increased "on" sensitivity can be seen in Figure 4. The heavier solid line is the mean latency of response for a 2 kc tone of constant intensity, presented ipsilaterally. Latency was measured from the onset of cochlear microphonic to the first evidence of its attenuation by muscle contraction. The range for three serial measurements is indicated by the short parallel lines. The upper dashed line was obtained with a 0.5 kc tone; the dashed line below, with a 5 kc tone. For relatively short rise-time rates, 0.5 kc adds, and 5 kc subtracts about 1 msec of response latency. For rates greater than about 25 msec, the variable latency, indicated for the 2 kc measurements, suggests that frequency of the stimulus assumes even less importance.

The response latencies measured herein were assumed to be those of the stapedius muscle. Those of the tensor tympani reflex, though similar in character, were always at least a few milliseconds later. The longer latency and the effect of varying the intensity of the 2 kc tone used to produce the reflex are shown in the two remaining plots of Figure 4. In one plot, the insertion of the tensor tympani was cut, leaving only the stapedius active. In the other, the stapedius insertion was sectioned.

It should be pointed out that the relation between the two sets of data shown in Figure 4 is an arbitrary one, resulting from abscissa placements. While there is no doubt that a relation does exist, the degree of variation among experimental animals and the interaction of stimuli have allowed only the generalizations just given. Even these, however, suggest that "on" sensitivity alterations resulting from envelope shape are small, and that the regularly observed greater "on"

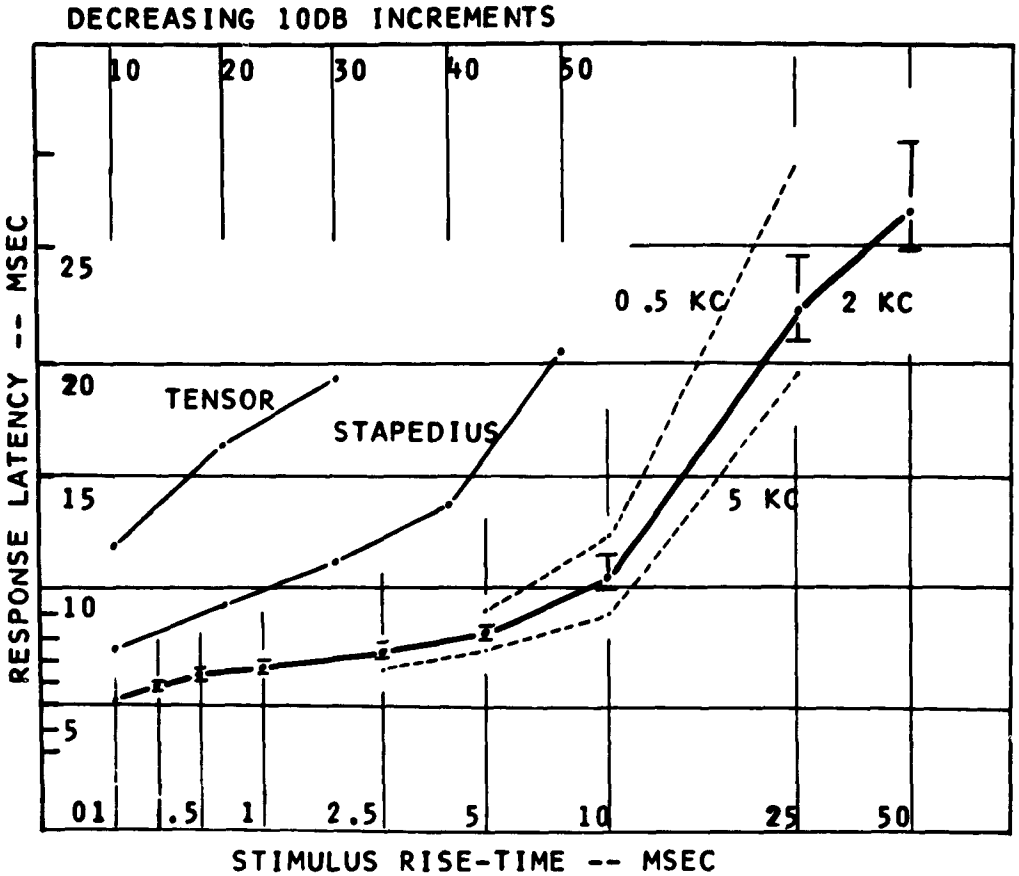

Fig. 4. Reflex Response Latency, Alterations with Rise-Time, Frequency, and Intensity Changes: Latency was measured from onset of cochlear microphonic to first evidence of its attenuation by muscle contraction.

than SS response sensitivity cannot be assumed due to sound transients or intra-cochlear wave forms which excite a larger area of the cochlear partition. A portion of the relative increasing "on" sensitivity with higher frequencies may be attributable to stimulus transients, but this would seem to be minor, particularly since intra-cochlear traveling waves can be reasonably supposed to cause frequency-related latency delays in a direction similar to those seen in Figure 4. Thus, while stimulus rise-time can be considered to change the absolute sensitivity by a factor of about 10 db, frequency, intensity, and duration remain the preponderant variables determining slope and sensitivity of the "on" response.

Frequency: "On" sensitivity is spatially oriented along the cochlear partition according to stimulus frequency. The simplest observation which indicates this is the observation that when two tones of different frequency are presented simultaneously, "on" sensitivity is not increased. In more detail: The intensity at which each would just produce an "on" response was determined for two tones, presented separately. These were attenuated, mixed, and introduced simultaneously. Their combined or individual intensities were then gradually increased until an "on" response occurred. None was seen at intensities less than that of one tone alone, if the mid-range-frequency separation was two octaves or more. The separation between the two tones could usually be reduced to one octave for frequencies centered around 1 kc, or to a half-octave when centered around 6 kc, before any indication of addition was observed. At stimulus intensities above "on"-response levels, inter-frequency separations greater than those mentioned were required to prevent increased SS response. For both "on" and SS, responses were such that the spatial relation for lower frequencies was more diffuse than that for higher frequencies.

The spatial (frequency) relation of "on" sensitivity could not be compared directly to that presumed for threshold auditory acuity for cats with normal hearing. Such a comparison was, of course, attempted in the animals whose thresholds were known and reflex responses were obtained within 40 db of several cats' sensation levels. However, differences between animals, and the influence of other variables to be described later on, made direct comparisons difficult, on a frequency-to-frequency basis.

Major variations were avoided by an autocorrelation study, in which threshold and reflex response were measured first in the normal animal, and then in the same animal with threshold acuity depressed (permanently) by sound-induced cochlear damage. Typical permanent changes in auditory threshold and reflex "on" sensitivity are shown in Figure 5. In evaluating such comparisons it must be remembered that a threshold index is being compared to a muscle response normally occurring at more intense sound levels. As a consequence, change in sensitivity of "on" at a given frequency was related not only to the threshold loss at that frequency, but also to the relative degree of cochlear damage sustained at higher frequencies--closer to the basal end of the cochlear partition, the direction of major traveling wave spread with increasing intensity. Within measurement limits of ± 10 db and a lowest frequency of 1 kc, "on" sensitivity decreased the same amount as threshold, if threshold losses at higher frequencies were equal to or greater than that of the compared frequency. If threshold

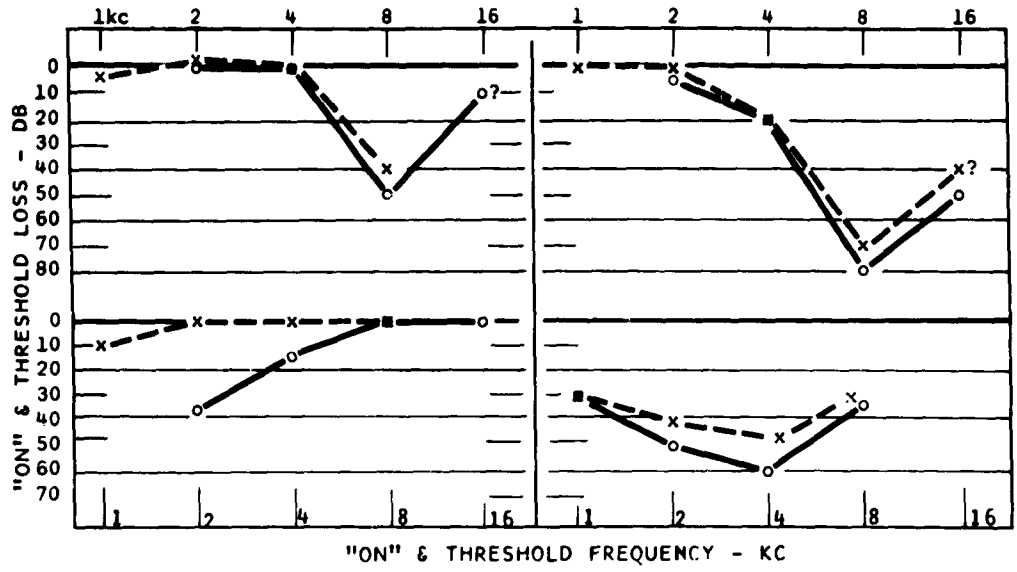

Fig. 5. Decreases in "On" Sensitivity (dashed lines) and Auditory Threshold (solid lines) following Sound Trauma.

losses were less severe for tones above the test frequency, "on" sensitivity was depressed less than the comparable threshold shift. These relations were not available in sufficient number either to study frequencies above 8 kc, or to calculate the bandwidth in slope per octave at each frequency required to affect "on" sensitivity at the next lowest frequency. It would seem, however, that, as with the addition studies, the "tuning" was much sharper at higher frequencies.

Intensity: As sound intensity increases above "on" response level, more of the cochlear partition is stimulated, and the maintained or SS level of reflex activity increases in proportion to this spread, toward the basal (high frequency) end of the cochlear. This is demonstrated in Figures 6 and 7. In both, the growth in SS reflex response to increasing intensities of a 1 kc tone is shown, first for the normal ear (top line), and then, for the same ear after sound-induced cochlear damage. Threshold acuity losses have been superimposed. Neither cat showed a post-trauma depression of "on" sensitivity at 1 kc. In the ear with the high-frequency loss, there is little growth in SS activity with increasing intensities of the 1 kc tone. In the other ear, SS activity is slightly reduced for the less intense stimuli, but as intensity increases, the pre-post-trauma differences are relatively smaller.

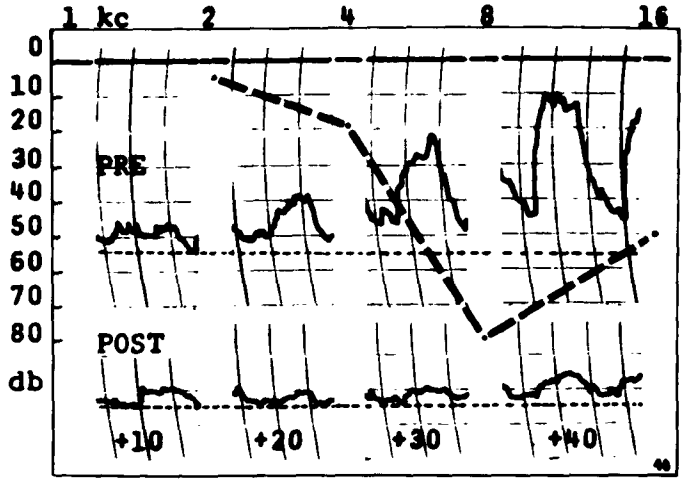

Fig. 6

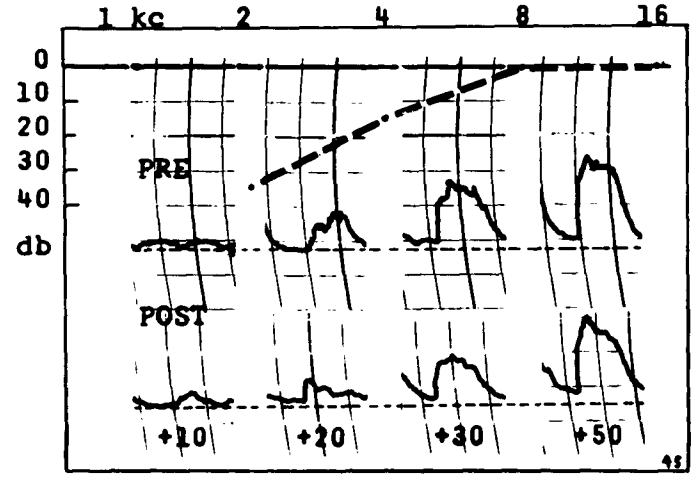

Fig. 7

Figs. 6 and 7. Growth in SS Level of Muscle Response Before (top) and After (bottom) Sound Trauma: The integrated EMG responses to the same increasing (+ db) intensities of a 1 kc tone are shown for each animal. Their auditory threshold changes with this damage are superimposed as dashed lines.

The reflex responses of Figure 7 illustrate one mechanism by which so-called "recruitment" in the muscle reflex can occur. A qualitatively similar type of reflex response could also be demonstrated for the ear of Figure 6 if, for instance, an 8 kc stimulus were substituted for the 1 kc tone used herein. Generally for such high-frequency losses, the rate of growth in SS, as compared to pre-exposure levels, was more rapid. Presumably, a more rapid "recruitment" might be a reflection of the more localized traumatic damage for high frequencies. "Recruitment" for sound intensities producing "on" responses was also observed, in the sense that the resolution of a tone just able to produce a response was much sharper after sound trauma.

Binaural Stimulation

The spatial relations demonstrated within the cochlea are maintained throughout the central nervous system (CNS) portions of the reflex arc. The nature of the spatial (frequency-intensity) separation was examined through the interplay of binaurally presented tones, as demonstrated in the experiments described below.

Bilateral summation of monaurally subliminal tones: The minimum intensity of a regularly repeated tone bleep (duration 200-300 msec) producing an "on" response was first determined separately for each ear. Both tones were then attenuated below their "on"-sensitivity level and introduced simultaneously. The intensity of one, or both was then adjusted to a level at which an "on" response was obtained. The bilaterally determined intensity was nearly always less than, and only rately equal to, that for a unilateral presentation. Figure 8 shows the measurements under such conditions for three cats, using a 1 kc stimulus. Zero on the xy axis indicates the relative intensity required to elicit an "on" response by a unilateral stimulus in either ear. The index of muscle response was an EMG from the tensor tympani. (Similar measurements were obtained from the more labile stapedius, but not with the same degree of stability.)

Bilateral tones of the same frequency produce the largest subliminal summations. As the delta-F fraction increases beyond about 0.1, the difference in sensitivity decreases until, at about one octave separation (1-2 kc), the monaural difference is only a few decibels. Usually, wider separations than an octave do not further reduce sensitivity. Bilateral presentation of the same frequency produces a larger sensitivity increase at low frequencies than high.

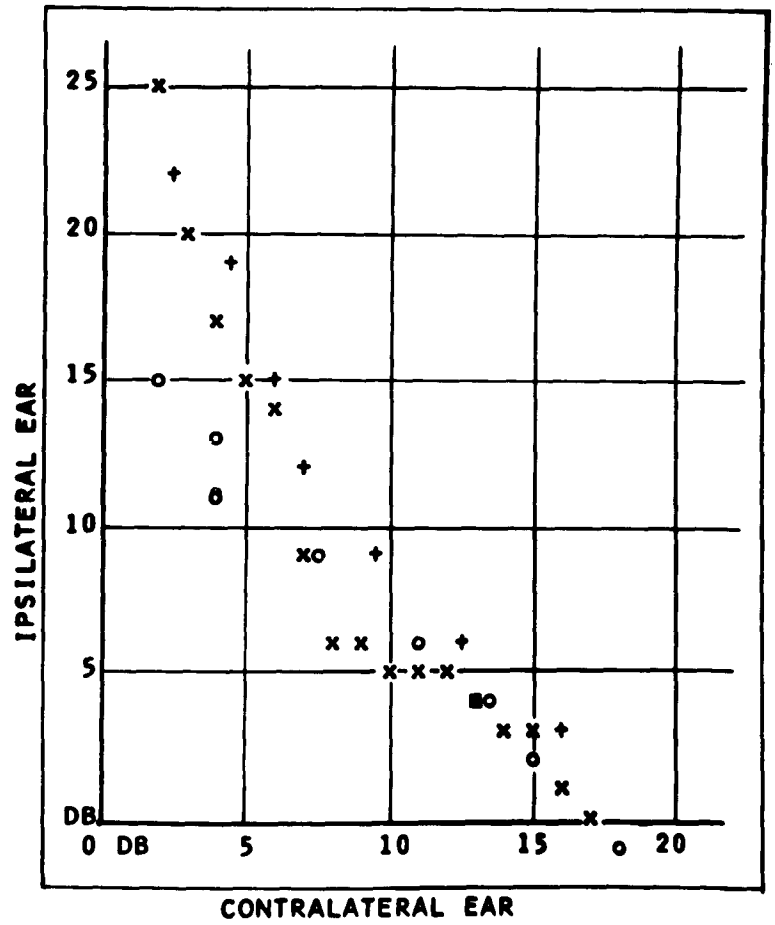

Fig. 8. Binaural Summation of "On" Response Sensitivity: "On" sensitivities for 3 cats binaurally stimulated by a 1 kc tone are shown in relation to their monaural sensitivities--0 db on xy axis.

Bilateral summation of supra-liminal tones: Bilateral stimuli introduced at greater intensities again reflect the spread of reflex activation over a progressively wider spatial area. In Figure 9, this growth is shown for an experiment in which increasing 10 db levels of a continuous 1 kc tone were introduced into one ear and tone pulses of varying frequency were presented through the contralateral ear. The length of each line indicates the decibel increase in "on" sensitivity at each of the pulse-frequencies as related to the least-intense 1 kc tone (about 45 db SPL) pairing. Note that the sensitivity to the 1 kc pulses

remains relatively unchanged, but the responses at higher frequencies become more sensitive, then begin to decrease again, as the intensity of the continuous tone rises through 60 db. (The reasons for these responses are no doubt complex and must involve both a binaural summation and direct contralateral pick-up of stimulus. The intra-cochlearly measured attenuation for mid-range air-conducted frequencies in the cat [measured by microphonic and neural responses] is at least 45-50 db. Failure of the 1 kc stimuli to add may be the result of cancellation of effects. At higher frequencies, particularly 4 and 8 kc, the increase in sensitivity was such as to produce response levels within 20-30 db of auditory threshold. The fact that sensitivity did not alter in 1:1 relation to 1 kc intensity increases suggests that across-the-head effects deserve only partial consideration.)

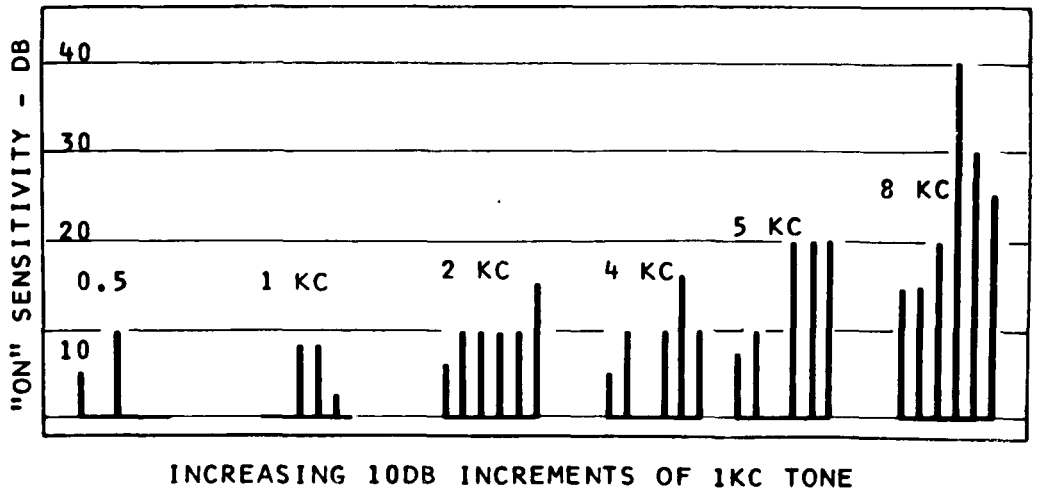

Fig. 9. Binaural Summation of Supra-Liminal Tones: Ordinate zero at each frequency is the relative "on" sensitivity for the least intense ipsilateral continuous tone (45 db SPL) and contralateral "on" response pairing. Each bar, or space, to the right indicates a 10 db increase in the 1 kc continuous tone. A gap means no change in sensitivity at that carrier level.

The limiting condition for binaural summation of SS activity occurs when unilateral intensities of more than about 100-115 db (at 1 kc) are presented. At these sound levels, monaural stimulation can be considered to "saturate" or occlude the neural units available for bilateral interaction. Some small degree of "on" response can be induced, however, in some animals, and, as suggested in Figure 9, more response can be induced by wider separation of frequencies.

It is interesting to note, both in regard to responses such as those of Figure 9, and in the earlier experiments, that inhibition (depression of reflex activity) was never seen under conditions where there was not a reasonable suspicion of end-organ fatigue or the presence of beat phenomena. Thus, it may be axiomatic that when either monaural or binaural interaction between stimulus pairs occurs, reflex responses always add. The binaural interactions not only suggest a maintained spatial (frequency) relation for the CNS portions of the reflex arc, but also serve once again to demonstrate one ear's influence on the responses of the other. The over-all result for reflex activity is a remarkable degree of bilateral symmetry for "on," SS, and even the minor second-to-second fluctuations of contraction.

Stimulus-Response Latency

Stimulus-response latencies were measured from the onset of the cochlear microphonic to the first oscilloscope-observed EMG spike. These were:

	Ipsilateral--				Contralateral--			
	Stapedius---		Tensor		Stapedius---		Tensor	
	min	max	min	max	min	max	min	max
msec	3.7	12	5.4	20	4.2-4.3	16	6.9	40

These values are the extremes observed, but they were confirmed in several different animals. The minimal latencies are somewhat shorter than those reported by others (3), and universally shorter by 1 1/2 - 2 msec than those observed for an actual contraction reflected as an attenuation of the cochlear microphonic. The stimulus parameters required for such short latencies are suggested in Figure 4, with the addition of one more variable mentioned later.

DISCUSSION--PART I

Within the limits imposed by absolute differences in response sensitivity, the spatial relations of the reflex arc have been shown to be similar to those for threshold discrimination, maintained in the central portions of the arc in the same orderly arrangements that have been shown for other auditory neural responses (4). The over-all reflex response throughout the arc parallels that of a loudness function, adding under some circumstances, and never showing suppression with multiple-input stimuli.*

*Possible binaural phase differences other than 0 and 180° have not been studied.

Reflex arc: The neural pathways by which both ipsilateral and contralateral reflex responses are transmitted, or mixed, are incompletely known. The spiral ganglion and cochlear nucleus are the first two neural units, while the motor nuclei of the 5th (tensor tympani) and 7th (stapedius) nerves constitute the final link in the chain.* While further anatomical studies are required, Figure 10 represents a diagrammatic attempt to "connect" the afferent and efferent segments of the loop through a consideration of known anatomy, analogs obtained from studies of spinal reflexes (10), and the observations reported here.

The figure suggests that there are several reflex loops. The shortest (spiral ganglion--cochlear nucleus--motor nucleus) is for an ipsilateral excitation and is required to satisfy the very short latencies observed, especially for the stapedius muscle. The relative number of fibers involved in the diagram suggests that this loop is not ordinarily the major one, but becomes increasingly significant only with intense stimuli. The more usually observed longer latencies, and the maximum latencies, suggest that at least one additional neural unit, and a more diffuse pathway, are involved in most ipsilateral stimulations.

The shortest contralateral pathway requires a 4-unit chain of neural units, again based on minimum latency. (Reliance on latency alone is not as secure in this instance since the minimum observed is still possible for a 3-unit pathway.) As in the ipsilateral loop, the majority of contralateral stimuli seem to be transmitted through a longer chain of neural units, requiring longer delays and showing longer reverberation times. The longer pathway--shown in Figure 10 as a 5-unit minimum--suggests a possible reason for the slight skew in the ipsi-contralateral additions of Figure 8. It also suggests that the ipsilaterally activated reflex may be slightly more sensitive than the contralateral one.**

A difference between the reflex arc of the stapedius and that of the tensor tympani is suggested by the observation that the tensor reflexes are usually characterized by less sensitivity and a poorer maintenance of contraction as well as a longer response latency. Observations of this general nature have been made by many others as

*Fibers from the region of the superior olive to the 5th nucleus (5), and fibers from the cochlear nucleus to the 7th nucleus (ipsilateral), and crossed connections from the accessory superior olive (6) have been described.

**For the cat, difference in sensitivity is not more than 5-10 db.

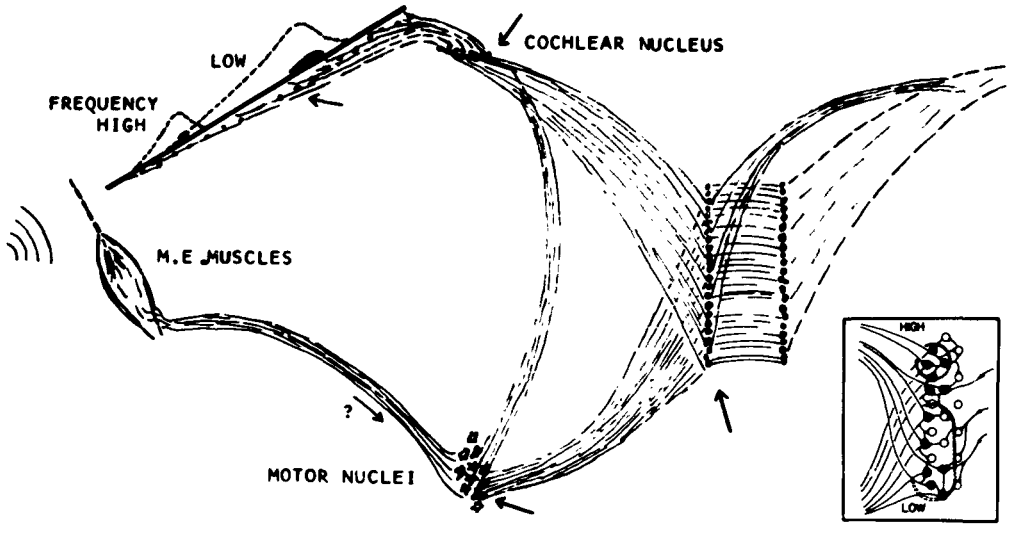

Fig. 10. Diagrammatic Model of the Middle Ear Muscle Reflex Arc: The insert on the left is intended to suggest that the reflex arc, at several of its synaptic junctions, may involve one or several neurons (and second-order feedback loops)--the minimal number being that shown in the larger diagram.

well. The difference does not seem to be due to afferent (cochlear) innervation since "on" sensitivity of the tensor tympani was equal to that of the stapedius in some animals when measured under carefully controlled conditions. Also, there does not appear to be any substantial difference in sensitivity shift of the two reflexes following varying degrees of sound trauma. It also seems unlikely that there is any fundamental difference in adaptation or fatigue of the efferent (motor) portion, since tensor tympani responses to some non-auditory stimuli, such as a puff of air, show vigorous contractions which are sustained for long intervals.

It seems most reasonable to suppose that the difference between the two arcs is due to their relative degrees of diffuseness, or coupling, through the interneuron population diagrammed as a near-midline structure in Figure 10. A separate population for each muscle is not indicated, nor required, since the differences between the stapedius and tensor tympani responses appear to be only quantitative. Under most circumstances the degree of auditory "coupling" for the stapedius

arc is closer and less diffuse, utilizing fewer interneurons than the tensor tympani arc.

"On" to SS: It has been suggested that a reflex contraction is maintained in direct proportion to the total area of the cochlear partition stimulated, and as indicated in Figure 10, this area is larger for low frequencies. The difference between this SS level of reflex response and the initial degree of contraction ("on" sensitivity) must be based on temporal factors, since it can be most reasonably assumed that there is little difference between the segment of cochlear partition initially activated and that portion activated after the stimulus has remained on for a period of time.

The decrement of reflex response incurred through transition from "on" to SS, could be attributed to fatigue, adaptation, or habituation. Of these, fatigue (a decrement due to the excitation itself) can be disregarded for most of the stimulus used--those 80 db SPL and below--and need not be considered a major factor. Adaptation (decrement in the rate of nerve fiber response to a continuing stimulus) adequately describes a majority of the reflex-response attributes, particularly when compared qualitiatively to those observed with gross electrodes at the level of the eighth nerve (7). The rate for individual fibers from the same region is much more rapid (8). The data do not, however, fit a concept of adaptation cited by Hood and others (9), wherein the underlying mechanism of the transition from "on" to steady state is an equilibrium of energy expended in the response to the energy available for replacement, if such an "energy" pool is limited to that available within the ongoing reflex-response arc. That such "energy" must also be introduced from additional sources is demanded by the sudden and often dramatic increases in reflex-SS activity after very slight alterations in the continuing stimulus, alterations which include reduction of intensity, superimposition of very brief transients, or even the introduction of a non-auditory stimulus such as an air puff. The implication of a difference between the two "energy sources" suggests that the decline in reflex activity with time is partially within the control of reflex-loop constants, thus due to adaptation; and partially external to the loop, more generalized within the CNS, and perhaps more properly defined as habituation.

Such a dual control over reflex activity is implicit in Sherrington's "final common path" concept (10), extensively studied within the spinal cord, and equally applicable to the acoustic reflex. Békésy has made admirable use of the sensory analog (skin) of such arcs to show that differences in the neural mechanisms are very likely only quantitative (11).

It is from the more extensive studies on the spinal cord that the best explanation for the observed increased sensitivity of "on" response at higher frequencies is derived. The principle involved has been termed convergence, and is illustrated in the insert in Figure 10. At high frequencies, the relative number of fibers activated is small, and, by the same token, concentrated in a small area, converging on a limited number of ensuing neural units maximally, and thus increasing the likelihood of stimulus transmission, even though the total number be small. At low frequencies, a larger population is involved, but innervation density is relatively small, thus decreasing the probability of stimulus transmission for a weak stimulus, but ensuring transmission in increasing degrees as stimulus intensity increases.

RESULTS--PART II

In Part I an attempt was made to limit observations of reflex responses to stimulus parameters whose interrelationships may be considered static. This second part includes descriptions of an ill-defined aggregate of reflex variables which may be considered to operate dynamically within the static framework, changing sensitivity on a basis of what might be termed past experience or the effects of previously presented stimuli.

Sensitization: (increased response resulting from previous excitation) The most regularly observed sensitization effects were those associated with presentations of trains of regularly repeated tones, all of which had the same physical characteristics. In about 80% of such presentations, the reflex response was greater with the second--occasionally third--tone of the series. Also, the response latency for the second stimulus was decreased. This effect can be seen in the reflex responses of Figure 11, to both the more abrupt and the longer rise-time stimulus series. The frequency or intensity (below damaging levels) within the stimulus envelope does not alter the basic first-second pulse-pair relation, nor do variations of stimulus duration measured between .025 and 2 sec or corresponding stimulus repetition rates tested between 2 per sec and 1 per 10 sec.

A second, possibly related, sensitization effect has already been described in detail elsewhere and will only be briefly reviewed here (12). Absolute sensitivity of the "on" response can be temporarily increased by introduction of a brief, loud stimulus into either ear. The nature of such a sensitizing sound is not critical, but with all other factors being equal, more intense sound (but not longer duration) produces a larger and more prolonged sensitivity shift. This effect is

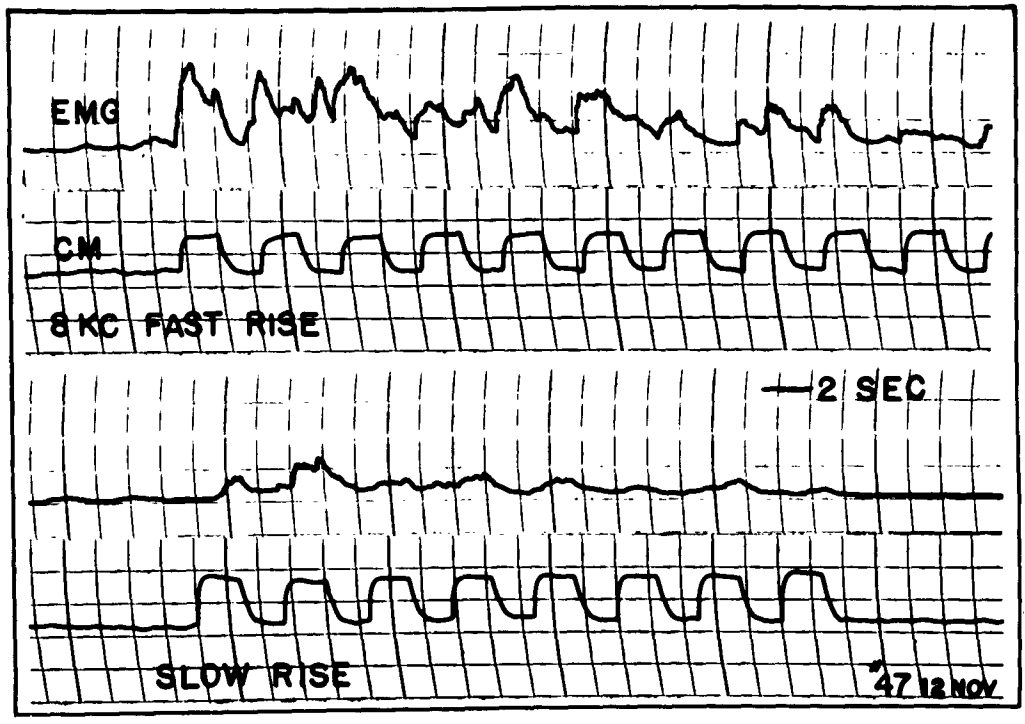

Fig. 11. Changes in Reflex Response: Stimulus Rise-Time, Sensitization, Habituation: The stimulus was 2 sec constant intensity 8 kc tone.

shown in Figure 12. Here, a continuous tone was presented to the ipsilateral ear until a SS level of reflex response was obtained. Tone pulses were then presented to the contralateral ear and a threshold sensitivity for "on" was obtained (70 db of attenuation). Then a more intense tone at the same frequency was presented (30 db of attenuation), followed by a repeat of the 70 db tone. Following this, the same procedure was repeated with a still more intense tone bleep--about 105 db SPL--(0 db of attenuation) as the sensitizing stimulus. Note and compare the sensitivity shifts produced in each case. Changes such as these have been shown not to be due exclusively to startle or dishabituation.

Fatigue: If an intense tone of a SPL similar to that of the previous experiment is left on for several seconds, a considerably different reflex change is observed--a temporary depression of the "on" response. This

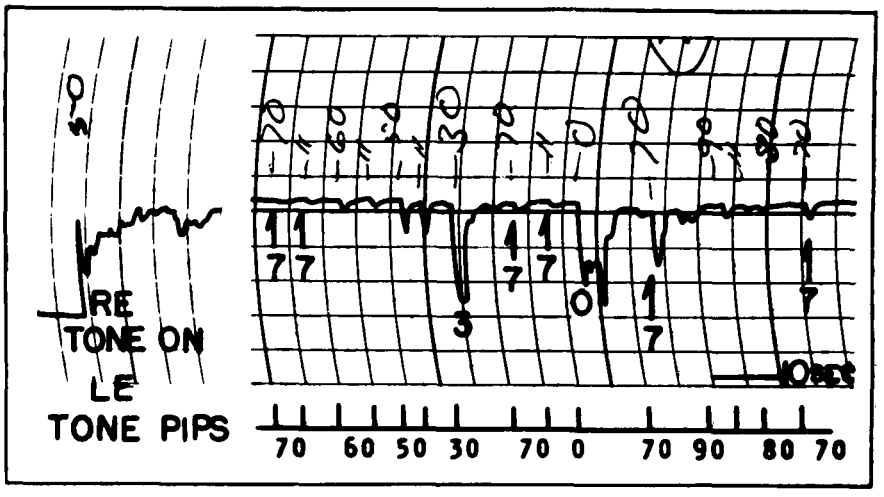

Fig. 12. Sensitization of the "On" Response, Contralaterally Induced: Stimulus intensities are in decibels--and bels--of attenuation for the contralateral (LE) tone pulses superimposed on a continuous RE tone.

depression is properly attributable to fatigue, since the degree of depression is directly related to duration and intensity of the loud-sound stimulus. Such depressions can also be presumed to reflect temporary sound trauma, inseparable from the parallel description, reflex fatigue.

Figure 13 (line 1) shows these changes as reflected in the cochlear microphonic attenuation of an ipsilateral stimulus. At "A" a low-level 1 kc tone was introduced and remained on for about 100 sec. This tone was then followed by a much more intense 1 kc stimulus (blank space) which remained on for about 30 sec. The "A"-level stimulus was then re-introduced. A comparison between the first "A" stimulus and the second one shows a considerably reduced "on" response. The first "A" stimulus was present for about 8 sec before contraction relaxation allowed the cochlear microphonic amplitude even to rise above the baseline noise level of the recording system. After the high-level tone, there is no appreciable delay. Note also that the SS level of reflex activity was relatively unaffected. Return of the reflex to its previous sensitivity required about 2 min, in this experiment.

Lines 2 and 3 of Figure 13 are the responses to repetitions of the same series of tones used in line 1, but after additional "fatigue" (permanent and temporary) had been introduced through exposure to a 1 kc tone for 2 hours at 135 db SPL. Three hours after this exposure the responses of line 2 were recorded. Here, reflex response to the

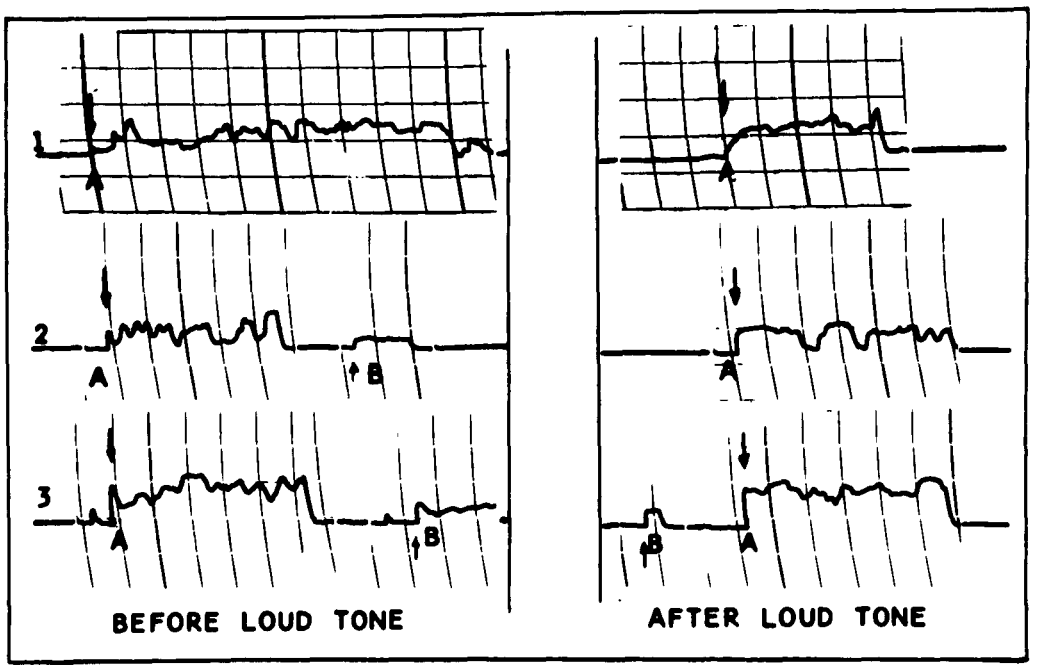

Fig. 13. Fatigue-Induced Changes in Reflex Sensitivity: Tones were introduced at points marked by arrows.

first "A" tone was depressed, as compared to the corresponding line 1 response, and, after the temporarily-fatiguing tone, the repeated "A" tone elicited no "on" response at all. The responses shown in line 3 were obtained 24 hours later. Reflex responses were still depressed, but not so severely as on the previous day.

The cochlear microphonic amplitudes marked "B" in lines 2 and 3 were amplitudes produced by a 1 kc tone 10 db less intense than the "A" stimulus. Because of SS middle ear muscle activity, this less intense tone did not produce a level of cochlear microphonic which was visible above the baseline noise level of the graphic record. Following severe trauma, even though there was an absolute depression of cochlear microphonic--measured under anesthesia--the response was measurable in the awake animal because of depression in SS muscle attenuation. Immediately post-trauma (line 2) no "on" or SS response was seen, but after 24 hrs, reflex activity was returning at "B" levels, and in the second "b" tone of line 3 "on" was again absent after the temporarily fatiguing more intense tone.

Portions of lines 2 and 3 are not, strictly speaking, classifiable as physiological temporal effects on reflex response, in that a factor of permanent damage was introduced, and thus might not seem appropriate to this discussion. They were included here to demonstrate one way in which the middle-ear-muscle reflex can be used as a tool to study auditory pathology, once a basic understanding of the normal response parameters has been achieved. This instance demonstrates an effect of superimposition of temporary trauma (fatigue) on a permanent or more severe sound trauma, during the latter's recovery period.

In another sense, these portions of Figure 13 are definitely appropriate, in that they serve to point out the perhaps obvious fact that the middle-ear-muscle-acoustic reflex is completely inactive when the loop is deprived of its efferent portion by cochlear deafness. The converse, that the reflex is most active at a given stimulus level when auditory acuity is the most sensitive, cannot be stated without the many qualifications which are currently under description.

Habituation: That ubiquitous plague of biological experimenters, or the gradually decreasing response to a particular stimulus--habituation--can be divided into two categories in the present experiments. One of these, habituation to the auditory stimulus, has already been brought up in the discussion of Part I, transition from "on" to SS. The effect of such habituation can also be seen in Figure 11, in the reflex responses which follow the sensitization described just before. While the rate of habituation was much slower for such regularly repeated pulse stimuli than for a continuous tone, and slower still for irregularly repeated stimuli, habituation continued to be more rapid for high-frequency stimuli. During an individual test session of 15-30 min such effects could be avoided by appropriate stimulus programming.

The second category, habituation to the test environment, was more insidious and difficult to estimate since some animals never gave any indication of adaptation to their test environment and continued to struggle and vocalize to an extent which made each test session more of a trial for the experimenter than for the cat. Others, after a few sessions, purred intermittently and lay more or less quietly, while a few occasionally slept, during prolonged testing; and rarely tones of a sensation level well above 100 db failed to stir their slumber. For the most part, the cumulative effects of environmental habituation were avoided for most cats by keeping sessions short and infrequent (average 1/wk), and by anesthetizing the animal immediately after the test.

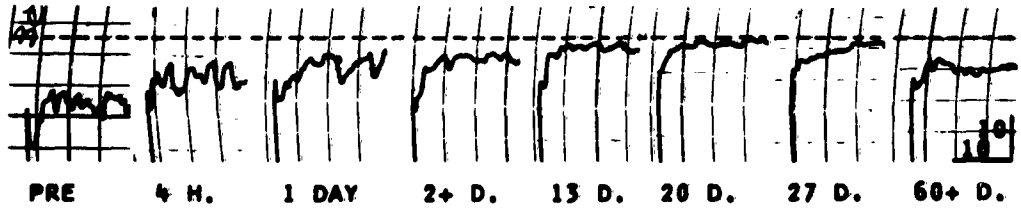

Fig. 14. Habituation of the Middle Ear Muscle Reflex: Reflex response (attenuation) of the cochlear microphonic response to a 90 db, 1 kc tone, as compared to muscle de-activated (anesthetized) amplitudes (dashed line) is shown for a series of tests over a 2 month period. The time sequence labeling refers to days after sound trauma exposure. (The animal's permanent acuity loss from this exposure was 25 db at 1 kc only, none at higher frequencies. Recovery to this level was complete after the 2nd post-exposure day.)

Figure 14 shows the reflex habituation to a 90 db SPL 1 kc tone for one animal over a 1 month period, during which time the cat was tested frequently and for prolonged sessions. The degree of attenuation of the cochlear microphonic with the reflex active is compared with the reference, anesthetized, muscle-de-activated amplitude. Note that both "on" and SS responses diminish, and that SS levels of activity, even to a 1 kc tone, relax to almost no contraction by the 20th day. Following the 27th day test, no tests were made until the 60th day, when a partial dishabituation can be seen. At no time was this animal somnolescent. Similar degrees of habituation were observed at even more intense sound levels, up to about 115 db SPL. That such habituation is to the test environment rather than to test tones is supported by the fact that the cat whose records appear in the figure had undergone 2 months of threshold and suprathreshold behavioral test sessions which used a 1 kc tone in a shuttle box prior to the reflex measurements, and continued to be tested behaviorally throughout the time period mentioned. Also, the reflex responses of the habituated animal could be temporarily and partially dishabituated by environmental changes such as turning out the lights in the test booth.

Arousal: Changes in reflex activity with the apparent degree of arousal or alertness of the animal parallel those withvaring degrees of habituation, in that sensitivity is characteristically increased with signs of alertness and decreased when the same animal becomes "inattentive"

or somnolescent. Indeed, the parallels are so close that most of such variations cannot be realistically described as different phenomena.

One change in reflex activity does seem to be specifically related to arousal, however. The characteristic minor irregular fluctuations in reflex response gradually diminish as an animal becomes less and less attentive, and finally disappear with apparent sleep. Figure 15 shows such changes in one cat who was alert when a tone was first presented, "A". The same tone was re-presented at "B", about 20 min later, after the animal had become somnolescent. The "B" tone remained on throughout the remainder of the figure, while at "C", "D", "E", and finally "F", "unexpected" tones were presented to the opposite ear. Following a rather loud tone at "F" the animal showed signs of arousal and no further sounds were introduced, but minor fluctuations in the ipsilateral response continued over about the next minute before the cat again became somnolescent. In attentive animals, habituated to the sound stimulus by continuous tone presentations with durations of up to 30 min, such minor amplitude fluctuations do not entirely disappear, though they are more pronounced in the first few minutes. In addition, these irregular irregularities, though accentuated by sound, are present in the absence of specific stimulation, as long as the animal shows signs of being relatively alert. *

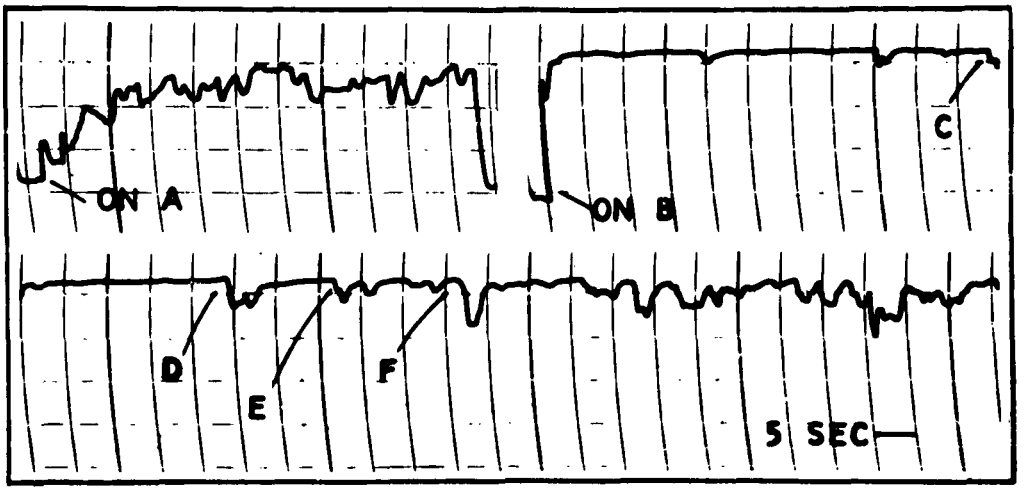

Fig. 15. Reflex Changes Associated with Alertness or Arousal: The tracing shown (described in the text) is the cochlear microphonic response to a 1 kc tone. The lower line is a continuation of the upper one.

*Measurements have not been made in ambient noise environments less than 40 db.

It should be stressed that these minor oscillations have no periodicity when observed visually either by oscilloscope or oscillograph, by EMG or by amplitude variations of cochlear microphonic. In general, rates of change from relaxation to contraction were between 2/sec and 1/5 sec, often this entire range in the same animal. Possible rates greater than 2/sec could not be reliably separated from the interference of other electrophysiological "noise."

Examples of such variable amplitude modulations of the acoustic input to the cochlea can be seen in several of the preceding figures. Though these variations were basically unpredictable, two patterns occur often enough to be of at least speculative interest. The first, seen only with stimulus intensities below about 90 db SPL, might be called a "sampling" response. One appears in Figure 1, line 1, where shortly after the tone is presented, reflex response relaxes, allowing more sound to reach the cochlea, then contracts again before reaching final SS level. Another but less well defined example can be seen in line 1 of Figure 13. The second pattern, characteristically seen at sound intensities above 90 db, was almost regularly cyclic. An example is shown in Figure 16. Following sound trauma, such oscillations have been observed at less intense stimulus levels. (See Figure 13, line 2, for example.)

In summary, the over-all effects of these "behavioral" variables--habituation, sensitization, etc.--occasionally changed reflex sensitivity ("on") by at least 50 db, and maintained contractions by 20-25 db of sound attenuation.

DISCUSSION--PART II

These observations demonstrate that the reflex response cannot be considered out of the context of the so-called static parameters, intensity, frequency, etc., is not enough. However, for the more dynamic variables just presented, with the possible exception of fatigue, a model cannot be constructed which does not encompass a large segment of the nervous system. Points within the static model where interaction can, and probably does, take place are indicated by arrows, the number of which suggest a great variety of possibilities.

In lieu of a model, and at some risk, the elusive characters of the probably interrelated phenomena of sensitization and habituation can be paraphrased: Within limits, the initial "on" sensitivity of the reflex for any sound stimulus depends on whether or not the animal can predict what that stimulus will be. If, for instance, with an alert,

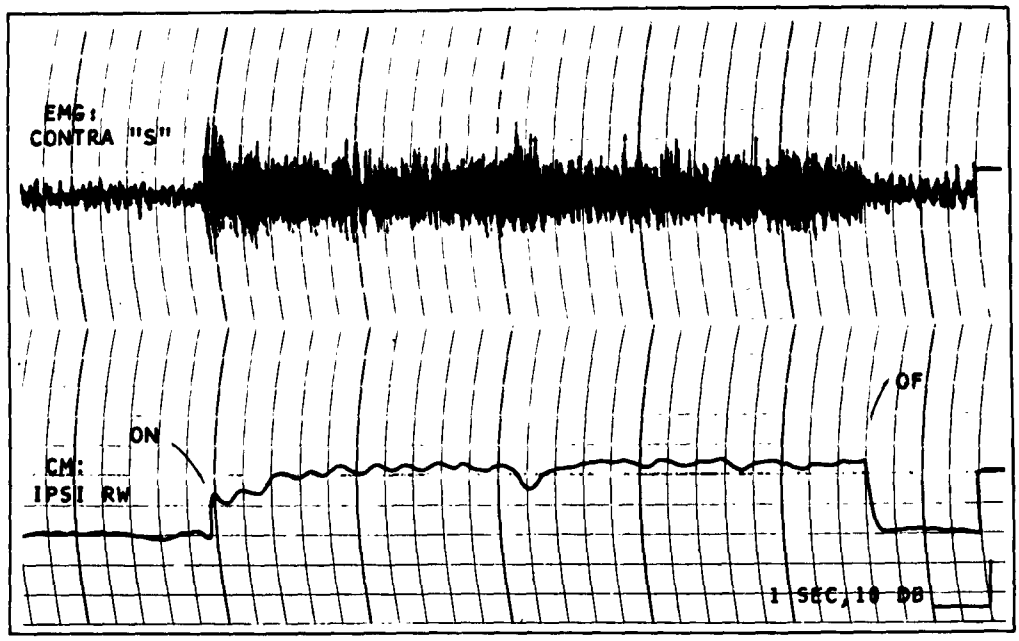

Fig. 16. Regular Oscillations of Reflex Origin as Seen in the Cochlear Microphonic Response to an Intense 1 kc Tone: Upper tracing is the contralateral stapedius EMG.

moderately habituated cat, the prior part of a given test sequence has consisted of sounds of the same frequency introduced with regular increments of intensity, the next sound that is presented will be greeted with an "on" response appropriate for the next loudest stimulus intensity of the series, even though the actual level of the tone presented is much less intense, or of a higher frequency, either of which would, in other circumstances, have produced a lesser "on" response. "Recognition" by the animal that such a "mistake" has been made requires time, and if test-tone duration is kept short, the resumption of a more appropriate degree of response requires more presentations.

The merit in such paraphrasing lies not in its novelty, since what it describes has been described in other circumstances many times before, but in its emphasis on, first, the need for consideration of what may be loosely called "psychophysical" parameters, and second, on problems in relating the reflex response sensitivity to auditory threshold acuity.

The threshold normal acuity was compared to muscle reflex sensitivity for ten animals of this group, and a qualified positive correlation found. However, it was decided not to present these data, for it is this observer's strong impression that such measurements are almost meaningless as a body of data separated from a consideration of the wide variability introducible through behavioral parameters. Such variability was the initial reason for using the autocorrelative methods of acoustic trauma to obtain more reliable information on one animal with two levels of auditory acuity.

Though such studies were mentioned only briefly in this second section, the possibilities are enormous for using the reflex, if all parameters are understood, as an index of ongoing alterations in auditory function studies, as has been quite gratifyingly apparent in concomitant observations on the recovery from acoustic trauma. It was during attempts to correlate pre- and post-exposure reflex responses to changes in auditory threshold acuity that the true significance of the minor oscillations in reflex activity was most forcefully suggested. In cats whose auditory acuity was severely depressed after a traumatic sound exposure, reflex-test tones presented at sound intensities well below the depressed acuity level, while showing no "on" or SS responses, continued to show the random minor fluctuations. (See Figure 13, line 2, 2nd "A"-tone response, for an approximating example.) In these instances, such oscillations could no longer be dismissed or attributed to possible "adjustments" in the feedback loop, even though, in normal ears, suprathreshold stimuli increased this activity.* An alternate explanation was sought, and found, on the basis of arousal.

The oscillations themselves, in an alert animal, can be attributed to reflections of cyclic sensitivity of a neuron pool, partially influenced by auditory input, and partially controlled by more generalized CNS activity. For conceptual purposes such a pool may be equated to that suggested to be active in determining the "on" and SS levels discussed in Part I, and called an "energy pool." (Though the two terms, neuron and energy, are not meant to be synonomous, either an increased sensitivity [energy] of already subservient neural units, or the addition of formerly neutral units [neuron pool] could account for effects observed, and probably both mechanisms are involved.) There is circumstantial

*The notion that such fluctuations were the result of artifact through movement, small changes in sound field, or poor electrical recording methods, had been abandoned much earlier when comparable animals with both middle ear muscles cut failed to exhibit them.

evidence for this equation in that the random oscillation activity becomes less and less apparent as sound stimulus levels increase above 90 db, the approximate level at which SS activity can no longer be increased by single-frequency binaural stimulation. At these intensities, neural elements formerly available only part of the time--thus showing random fluctuations--become fully recruited (saturated) for transmission of the auditory signal.

Such undulations in muscle activity have been previously reported for the tensor tympani, in decerebrate rabbits (13) and cats (3) at rates considerably higher (10-20/sec) than those seen in the normal animals studied here. Lorente de Nó felt that the 15-20/sec oscillations he observed represented a reflection of an inherent or natural frequency of the cochlear nuclei. The slower rates noted in the present experiments may represent the same phenomenon for the normal animal; and indeed, bipolar electrodes in the vicinity of the cochlear nuclei show the same low-frequency oscillations as do the muscle responses, under the same conditions and in many of the same animals. Such oscillations in the cochlear nucleus are, however, entwined with the reflex loop and cause-effect relationships. In the cochlear nuclei of the awake cat, the more rapid (10-20/sec) oscillations described for muscle responses were not seen, nor have EMG or cochlear microphonic oscillations at these rates been observed, even though repeatedly looked for. Especially for the tensor tympani a second explanation for the rapid oscillations may exist, indicated by a question-marked arrow in Figure 10. The question of presence or absence of efferent (sensory) structures within the muscle itself apparently is not resolved (15).

CONCLUSION

The middle ear muscle response to an acoustic stimulus is a kaleidoscopic composition, whose variegations include the parameters of stimulus, acuity and continuity of the loop, and the behavioral "attitude" of the organism, all reflected as a constantly changing pattern of cochlear microphonic and EMG.

The image reflected with each turn of the "kaleidoscope" may emphasize a slightly different view. In one, the emphasis can be placed on the word: reflex. It is indeed possible to show, with little difficulty, that all of the described variables affect any similarly complex reflex in analogous ways. In another view, the acoustic aspects might be emphasized. Again, there is no question that the input parameters are those of the auditory system, quantitatively different from those of other sensory systems and their reflex responses, and in the final analysis,

the muscle contractions are a reflection of ongoing activity within a neural structure, linked sometimes closely, sometimes loosely, to the auditory system through a common input and one additional relay, the motor neuron. In still another view, the focus may be the attenuation effects of the contracting middle ear muscles--a feedback loop, the first in a long series of such loops within the auditory system. The nature of the loop feedback may be visualized as positive or negative, depending upon both the input stimulus and the behavioral milieu through which it must pass.

The intent of this paper has been to present, and to some extent analyze, the middle-ear-muscle reflex in such a way as to unify the view of the muscle response as a reflex with that which considers its more specific auditory input. To this end, a spatially related model of stimulus-response parameters has been developed from a body of data, several of whose individual segments have been reported many times before.* The results provide a basis from which the wide variations in muscle activity, observed for both man and animal, can be understood. They also suggest a number of ways in which the reflex itself can be used as a tool to study ongoing activity in the auditory system.

Most of the data presented were observed at sound levels or durations well below those known to produce sound trauma. While a loudness function for muscle responses has been suggested, which indirectly supports the trauma-protection function of the middle ear muscles, the variety and complexity of change in responses, induced especially by the so-called behavioral variables, also demands that the search for additional auditory functions continue.

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*In fact, the bibliographic list is so extensive, including reports both on the middle ear muscles alone and on other reflexes, that the author was forced to avoid most specific references, the alternative being to lengthen the manuscript beyond publishable limits. The works of Kobrak and Lorente de Nó, because of the similarities of their conceptual approach to that of the author, require special acknowledgement.

BIBLIOGRAPHY

1. Simmons, F. B. Ann. Otol., Rhinol., Laryngol., 1959, 68, 1126-1144; and 1960, 69, 1063-1072.

2. Simmons, F. B., and Beatty, D. L. Trans. Amer. Otol. Soc., 1962, 50, 182-217.

3. Eliasson, S. and Gisselsson, L. EEG and Clin. Neurophysiol., 1955, 31, 399-406.

4. Rose, J. E. Neural Mechanisms of the Auditory and Vestibular Systems, Ed. G. L. Rasmussen (Charles C. Thomas, Springfield, Ill.), 1960.

5. de Nó, R. Lorente. Trans. Amer. Rhinol., Laryngol., Otol. Soc., 1933, 39, 26-42.

6. Rasmussen, G. L. Personal communication.

7. Derbyshire, A. J. and Davis, H. Am. J. Physiol., 1935, 113, 476-504.

8. Galambos, R. and Davis, J. J. Neurophysiol., 1942, 5, 39-47.

9. Hood, J. D. Brit. Med. Bull., 1956, 12, 125-130.

10. Sherrington, C. S. Integrative Action of the Nervous System, 2nd Ed. (Yale Univer. Press, New Haven), 1947.

11. von Békésy, G. Neural Mechanism of the Auditory and Vestibular Systems, Ed. G. L. Rasmussen (Charles C. Thomas, Springfield, Ill.), 1960.

12. Simmons, F. B. J. Acoust. Soc. Am., 1960, 32, 1589-1591.

13. de Nó, Lorente. Laryngoscope, 1935, 45, 1-23.

14. Malmfors, T. and Wersäll, J. Acta. Morph. Neerl. Scand., 1960. 3, 157-169.

15. Blevins, C. Studies on Neuro-Muscular Relations of the Middle Ear. Ph. D. Thesis, Univer. of Calif., San Francisco, 1961 (to be published).

16. Kobrak, H. Arch. Otolaryngol., 1938, 27, 59-66.

THE IMPEDANCE OF THE EAR--A VARIABLE QUANTITY

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INTRODUCTION

This paper reports on a series of measurements of the specific acoustic impedance of the ear of the guinea pig. During these measurements it was observed that the impedance of the ear was not a stable quantity at any given frequency, but sometimes varied within a short period of time by as much as a factor of 4 to 8 times. This rather annoying lack of repeatability of measurements, made with a technique in which we had confidence, lead us to further investigate the cause of the fluctuations.

Impedance is the complex quantity of the ratio of force to velocity and the phasic relation between the two: the quantity provides a fairly complete description of the function of a mechanical system. Specific acoustic impedance is the complex quantity of the ratio of effective sound pressure to effective volume velocity and their phase relation and describes the acoustic properties of the system. Units of specific acoustic impedance are dyne-sec/cm^3 or rayls. Both sound pressure and volume velocity were measured at the tympanic membrane and thus the impedance is that of the total ear as it appears to sound energy striking the eardrum. The impedance of the total ear is presented along with impedance measurements of some of the individual components that comprise the total system. Throughout this paper we shall indicate how alterations in the various portions of the acoustic structure of the ear may produce changes in the impedance of the total ear. Finally we shall present the results of experiments in which the impedance of the total ear was quite easily but dramatically altered and discuss the significance and implications.

ANATOMY

The anatomy of the guinea pig ear is reasonably similar to the human ear (Fig. 1). The tympanic membrane of 0.25 cm^2 area forms the lateral wall of the larger cavity of the middle ear space. This cavity is approximately 0.20 cm^3 volume and is joined by a smaller

cavity of 0.05 cm^3 through a small opening in the superior wall. The body of the malleus passes through the small opening so that the anterior end of the bone has its ligamentous attachment in the small cavity. The manubrium of the malleus is, of course, attached to the tympanic membrane; the joint between malleus and incus appears to be non-functional. The incudo-stapedial joint appears to be in part formed by a ligamentous attachment of the incus to the stapes and seemingly is a very loose joint. The tensor tympani muscle hides within a bony cave behind the cochlea on the medial wall of the large cavity and attaches to the manubrium of the malleus at the junction of the upper and middle thirds. The stapedius muscle arises from beneath a shelf of bone on the posterior wall of the large cavity and attaches to a protuberance on the posterior arch of the stapes.

IMPEDANCE OF THE TOTAL EAR

Impedance was measured with a technique which will not be described in detail in this paper. The technique is a direct measure of sound pressure on the tympanic membrane and an indirect measure of the volume velocity of the tympanic membrane and thereby avoids some of the difficulties encountered with other impedance measuring methods. Needless to say it has shortcomings, of which the necessity of entering the middle ear space with a probe tube microphone is the most restricting since it fairly well limits volunteer subjects to experimental animals.

The total impedance of a typical guinea pig ear is shown in Figure 2. Impedance values in rayls are plotted as a function of frequency; phase angle is given in degrees in the upper graph. Impedance decreases with increasing frequency at the rate of 6 db/octave from 3000 rayls at 100 cps to 150 rayls at 2000 cps. From 2000 cps to 900 cps impedance varies from 90 to 300 rayls. The specific shape of the curve is characteristic for each ear but in general in every curve there is a maximum value reached between 3000 and 7000 cps. Phase of the impedance changes from -90° (pure elastance) at 100 cps to -45° at 2000 cps. The impedance of the ear in this frequency range appears to be almost entirely elastance as suggested by the 6 db/octave slope and confirmed by the phase. From 2000 cps to 9000 cps the phase changes with small individual variations from -45° to +45°, usually remaining within 10° of zero for an octave or more. Therefore, in this frequency range where the impedance is more or less constant at about 150 rayls the phase angle indicates that this impedance is predominantly resistive or frictional in character.

The impedance of the total ear is reasonably approximated by a simple series circuit of elastance, resistance and mass, as is shown by the third graph of Figure 2.

Averaging of impedance values for a number of ears at each frequency obliterates individual variations. Figure 2 also presents the average impedance of 10 ears at each frequency and reveals that the impedance averages 150 rayls from 2000 to 10,000 cps. This curve is a close approximation to the impedance curve for any guinea pig ear in our series of 70 ears and is within ± 3 db of most "normal" animals.

IMPEDANCE OF THE VARIOUS STRUCTURES

The impedance of the cochlea was calculated as the difference between impedance measurements at the tympanic membrane before and after disarticulation of the incudo-stapedial joint. The value does not represent the actual value of the impedance of the cochlea but is rather the value of the cochlea as it appears to sound energy striking the tympanic membrane. Though the impedance of the stapes and attached stapedius muscle is included in the value for the cochlea they probably add very little. The impedance of the cavities of the middle ear was calculated from static measurements of their volumes by assuming them to be perfect cavities. The dynamically effective acoustic volume of these cavities was measured experimentally and was found to be in close agreement with static measurements of volume. The impedance of the tympanic membrane and ossicular chain was obtained by subtracting the impedance of the cavities from the value of the impedance of the ear after the cochlea was removed by disarticulation of the incudo-stapedial joint. This quantity represents the impedance of the tympanic membrane, malleus with attached tensor tympani muscle, and incus, as they appear to sound energy at the tympanic membrane.

THE MIDDLE EAR SPACE

The cavities of the middle ear form an interesting acoustic structure. A microphone placed in the small superior cavity revealed that sound pressures are not passed to this cavity equally well at all frequencies. At low frequencies sound pressures were equal in both cavities, but at higher frequencies no sound pressure could be measured at all in the small one. Thus the opening between the cavities, through which the body of the malleus passes, acts as an acoustic filter with a high frequency cut-off, i.e., like an acoustic mass.

Measurements made with microphones in both cavities revealed that the acoustic mass (inertance) of this opening and the acoustic spring (elastance) of the small cavity formed a resonant acoustic system, whose resonant frequency was between 3000 and 7000 cps. For simplicity of nomenclature we shall refer to this as the resonance of the small cavity. The impedance of a typical system of both cavities is shown in Figure 3. Both cavities function together as a system of coupled cavities. At low frequencies the impedance of the system is equivalent to the sum of the volumes of the two cavities (0.25 cc). Just below the frequency of resonance of the small cavity the impedance of the system of cavities falls to a minimum. As the resonance frequency is reached and passed the impedance of the cavity system increases quite rapidly from a minimum to a maximum value. At frequencies above resonance the impedance of the opening between the cavities increases rapidly and very quickly acoustically closes the small cavity from the large one. The impedance of the cavity system then, of course, becomes simply the impedance of the larger cavity alone (0.20 cc). Figure 3 presents impedance values for the total ear and for the system of combined cavities. It is quite apparent that the impedance of the cavities comprises virtually the entire impedance of the ear at frequencies below 1000 cps since the two impedance curves closely approximate each other over this range. Thus the impedance of the other structures of the ear are insignificant at frequencies below 1000 cps. This is an important fact to keep in mind in experiments in which the bulla is entered for any reason, such as in mensuration of cochlear potentials. Unless the bulla is heremetically sealed after entry, the ear will present a markedly altered acoustic system and parameters such as threshold of microphonics or sound pressure levels which produce alteration of microphonics will not be accurate.

The maximum peak in the total ear impedance curve which always appeared between 3000 and 7000 cps coincides with the maximum peak in the middle ear cavity system and thus the structure producing this consistent individual variation is found to be the coupled cavity system of the middle ear space. The frequency and magnitude of the peak is established by the opening between the cavities and the volume of the small cavity. In the experiment illustrated in Figure 4, impedance of the total ear was measured and then the opening between the cavities was blocked with grease. The impedance of the total ear increased slightly at lower frequencies because the volume of the middle ear space was reduced by removing the small cavity from the system and the maximum peak which was so prominent at 4200 cps disappeared. Instead there is a minimum around 5000 cps.

Measurements made with microphones in both cavities revealed that the acoustic mass (inertance) of this opening and the acoustic spring (elastance) of the small cavity formed a resonant acoustic system, whose resonant frequency was between 3000 and 7000 cps. For simplicity of nomenclature we shall refer to this as the resonance of the small cavity. The impedance of a typical system of both cavities is shown in Figure 3. Both cavities function together as a system of coupled cavities. At low frequencies the impedance of the system is equivalent to the sum of the volumes of the two cavities (0.25 cc). Just below the frequency of resonance of the small cavity the impedance of the system of cavities falls to a minimum. As the resonance frequency is reached and passed the impedance of the cavity system increases quite rapidly from a minimum to a maximum value. At frequencies above resonance the impedance of the opening between the cavities increases rapidly and very quickly acoustically closes the small cavity from the large one. The impedance of the cavity system then, of course, becomes simply the impedance of the larger cavity alone (0.20 cc). Figure 3 presents impedance values for the total ear and for the system of combined cavities. It is quite apparent that the impedance of the cavities comprises virtually the entire impedance of the ear at frequencies below 1000 cps since the two impedance curves closely approximate each other over this range. Thus the impedance of the other structures of the ear are insignificant at frequencies below 1000 cps. This is an important fact to keep in mind in experiments in which the bulla is entered for any reason, such as in mensuration of cochlear potentials. Unless the bulla is heremetically sealed after entry, the ear will present a markedly altered acoustic system and parameters such as threshold of microphonics or sound pressure levels which produce alteration of microphonics will not be accurate.

The maximum peak in the total ear impedance curve which always appeared between 3000 and 7000 cps coincides with the maximum peak in the middle ear cavity system and thus the structure producing this consistent individual variation is found to be the coupled cavity system of the middle ear space. The frequency and magnitude of the peak is established by the opening between the cavities and the volume of the small cavity. In the experiment illustrated in Figure 4, impedance of the total ear was measured and then the opening between the cavities was blocked with grease. The impedance of the total ear increased slightly at lower frequencies because the volume of the middle ear space was reduced by removing the small cavity from the system and the maximum peak which was so prominent at 4200 cps disappeared. Instead there is a minimum around 5000 cps.

The effect of alteration of volume of the large middle ear space such as might be produced by accumulations of fluid may be seen in Figure 5. In this experiment the volume of the large middle ear space was reduced by about one-third by adding mercury to the cavity. There is an increase of 6-12 db in the impedance values at frequencies below 3000 cps. Thus accumulation of fluid in the middle ear space results in an increase in impedance of the ear at low frequencies and has little effect at higher frequencies.

IMPEDANCE OF DRUM AND OSSICULAR CHAIN

The combined impedance of the tympanic membrane, malleus with attached tensor tympani muscle, and the incus appear as a simple resonant system composed of inertance and very little resistance (Fig. 6). Resonant frequency for the system is between 2000 and 2500 cps. The specific location of the reactances, whether in the tympanic membrane or in bones, supporting ligaments and joints of the ossicular chain, was not delineated. For simplicity of nomenclature we shall refer to the elastance as lying in the tympanic membrane and the inertance in the ossicular chain. The elastance of the tympanic membrane is equivalent to the elastance of a cavity of volume 0.8 cm^3, or is about one-fourth as stiff as the elastance of the middle ear space. Thus at low frequencies the impedance of the drum is small compared to the impedance of the middle ear space and has no appreciable effect on the total impedance of the ear. At high frequencies, however, the inertance of the drum and chain appears to dominate the reactive impedance of the total ear as the effect of the mass causes the phase angle of the impedance to become positive with increasing frequency. It is interesting to note that the measured inertance of the tympanic membrane and ossicular chain is equivalent to a mass of 0.6 mgm while the weight of the tympanic membrane and manubrium attached to it were found to weigh 1.6 mgm. Thus the effective mass of the entire system is much less than the mass of the drum and the manubrium alone. The explanation for this is, of course, that the mass is well distributed about the axis of rotation of the ossicular chain and does not load the system.

The impedance of the cochlea as it appears at the tympanic membrane is shown in Figure 7. The magnitude of the reactance and the sign of the reactance were not accurately determined and results were inconsistent. However, the reactive component of the cochlear impedance is small; the impedance of the cochlea is predominantly resistive or frictional in character, having a value of around 150 rayls. This is approximately the value of the impedance of the total ear from

2000 to 9000 cps and we noted previously that the impedance of the total ear was predominantly resistive in character through this span of frequency.

We have demonstrated that the total impedance of the ear is predominantly the elastance of the middle ear space up to 2000 cps, the resistance of the cochlea from 2000 cps to 9000 cps, and is probably the inertance of the ossicular chain above 9000 cps. In the frequency range where the impedance of the total ear is the least (2000-9000 cps) that impedance is predominantly cochlear and resistive. This is an optimum situation wherein most of the sound energy absorbed by the ear goes to the cochlea and the frequency response is reasonably flat because there is little reactance in the total ear.

EFFECT OF STATIC PRESSURE

Most dramatic changes can be made in the impedance of the total ear by producing a static differential of pressure between the middle ear space and the outside of the tympanic membrane. Reduced pressure in the middle ear undoubtedly draws the eardrum inward, decreasing the middle ear volume and shifting the "center position" about which sound pressure fluctuations will cause the drum to move. Increased pressure in the middle ear must produce similar effects in the opposite direction. Such static pressure differentials between middle and outer ear must occur quite frequently as a consequence of natural events (for example, barometric pressure fluctuations, swallowing, temperature changes). Figure 8 illustrates impedance measurements made with static pressure changes as indicated in the middle ear. Increased pressure produces the largest values of impedance of the total ear at all frequencies. Decreased pressure also appears to increase impedance at frequencies below 1000 cps. However, above 1000 cps decreased pressures decrease the impedance. Above 1000 cps the amount of increase due to increased pressure and the amount of increase due to increased pressure and the amount of decrease due to decreased pressure apparently depends upon what "normal" values are--the maximum values occur with increased pressure and minimum with decreased pressure and the "normal" ear lies somewhere between. Minimum values below 1000 cps occur when there is no pressure differential across the tympanic membrane. It was noted while producing these changes in impedance by varying the static pressure that when either an increase or decrease of pressure was released and pressure across the tympanic membrane was equalized the impedance did not return to the same value it had had at the beginning of the pressure

change, i.e., a hysteresis in the system was quite apparent. Impedance could be returned to the starting value only by producing a small pressure differential in the opposite direction.

Figure 9 presents the resistive and reactive components of the complex impedance of Figure 8. It is easily seen from this data that there is much non-linearity of the system. Either increase or decrease of pressure will produce an increase in the elastance but the rate at which elastance increases depends upon direction. Increased pressure increases elastance about four times faster than corresponding decreased pressure. Most remarkable changes are seen in the resistive portion of the complex impedance. At higher frequencies increased pressure has little effect on the resistive element, but small decreased pressures decrease the resistance to very small values. It will be recalled that we have demonstrated that at frequencies above 2000 cps the total impedance of the ear is primarily resistive and closely approximates the impedance of the cochlea. Since it seems very unlikely that the impedance of the cochlea is actually decreased we must conclude that there is a change in the manner in which the cochlea is connected to the tympanic membrane producing a change in the apparent impedance of the cochlea measured from that point.

Almost all of the resistive change is produced by a decreased pressure of 4 mm of mercury or less and decreasing the pressure still more adds very little additional change. It will be recalled that 4 mm mercury is approximately equivalent to 146 db relative to 0.0002 dynes/cm^2. Thus if we now suppose alternating pressures between plus 4 mm and minus 4 mm of mercury such as would occur in a pure tone of 146 db, it is easily seen from Figure 9 how nonlinear and assymetrical the response of the mechanical system of the ear is to such a driving force as both resistive and reactive elements vary with intensity.

Changes of impedance with increasing sound intensity are illustrated in Figures 10 and 11. There is a distinct increase in elastance from 100 to 130 db SPL, but the change is gradual and subtle and it is difficult to say at what sound intensity the changes begin. At most frequencies there is a definite change of resistance from 110 to 120 db SPL and changes may have occurred at lesser sound levels at some frequencies. The changes, both in the total impedance and the resistive and reactive components, are consistent with the preceding data on static pressure.

Figure 12 is a plot of two impedance curves on the same ear of a living guinea pig--nothing was changed in the experiment, but two hours elapsed between the two series of measurements. We may postulate that the temperature of the animal may have changed and changed the static pressure across the tympanic membrane (one degree will produce about 2.5 mm H_g change) or perhaps that the response of the tympanic muscles changed due to wearing off of the anesthetic. The temperature change appears to be the most likely. At low frequencies below 1000 cps, impedance was significantly increased and above 2000 cps significantly decreased, fitting the general pattern of impedance change produced by a decrease of pressure or inward shifting of the tympanic membrane. The minimum at 7000 cps appears to be the resonance of the total ear. This set of data is offered as evidence that the impedance of the ear is indeed a variable quantity.

We have previously indicated that decreased pressure in the middle ear space will decrease the resistive component of the total impedance of the ear in a frequency range where this total impedance was very nearly equal to the impedance of the cochlea. Figure 13 compares the effect on the resistive impedance of the total ear of static pressure in the middle ear and the effect of disarticulation of the cochlea from the ossicular chain by disrupting the incudo-stapedial joint.

Further confirmation of the "de-coupling" is given by the following experiment. During the impedance measurements of Figure 12, microphonics were measured at the round window of the cochlea of the same ear in which the impedance was measured. These results are shown in Figure 14. There is a parallel decrease in resistive impedance of the total ear and round window microphonics showing that this decrease of resistive impedance is accompanied by a decrease in the amount of sound energy getting to the cochlea.

We are of the opinion that static pressure changes in the middle ear, per se, do not alter the conductive mechanism to any great extent, but rather static displacement of the conductive mechanism resulting from the static pressure is of much greater significance. Based upon this opinion we would like to offer the following hypothesis of action of the tympanic muscles. (1) There is a complex relationship between tensor tympani and stapedius muscle in which the muscles act in opposition. Tensor tympani draws the manubrium and attached tympanic membrane inward to tighten the ossicular chain against the head of the stapes which is "anchored" by the action of the stapedius muscle.

(2) This combined action results in an increase of elastance which is asymmetrical. As sound pressure now drives the drum, an inward motion sees the elastance of the stapedius muscle and an outward mction sees the elastance of tensor tympani. (3) A further result of this combined action of the two muscles is to alter the incudo-stapedial joint in such a manner as to change the mode of rotation of the stapes in the oval window (as described by Békésy for loud sounds). This effectively "de-couples" the cochlea from the conductive mechanism.

The increase of elastance increases the impedance of the total ear at low frequencies and thereby decreases the amount of sound energy getting to the cochlea. "De-coupling" of the cochlea protects it but also has removed damping from the conductive apparatus and, consequently, it is now much more liable to injury from large sound pressures. However, since stiffness is added directly in the conductive apparatus, this provides the needed protection for these structures against large displacements. Referring again to Figures 12 and 14, it will be seen that impedance of the ear reached a minimum and resonance at 7000 cps on the second curve and that round window microphonics were unchanged from the first curve at that frequency. Apparently the reflex is unable to protect the ear at some narrow band of frequencies around the resonance of the ear where the increased displacement is more than enough to off-set the "de-coupling" of the cochlea. In the study of the effect of static pressure it was mentioned that the impedance did not return to the value from which it started when pressure was released and that it was necessary to introduce pressure in the opposing direction to return the impedance to its original value. This adds further significance to the opposing action of the two muscles which permits an active return of the system rather than a passive return as would occur with simple muscular relaxation.

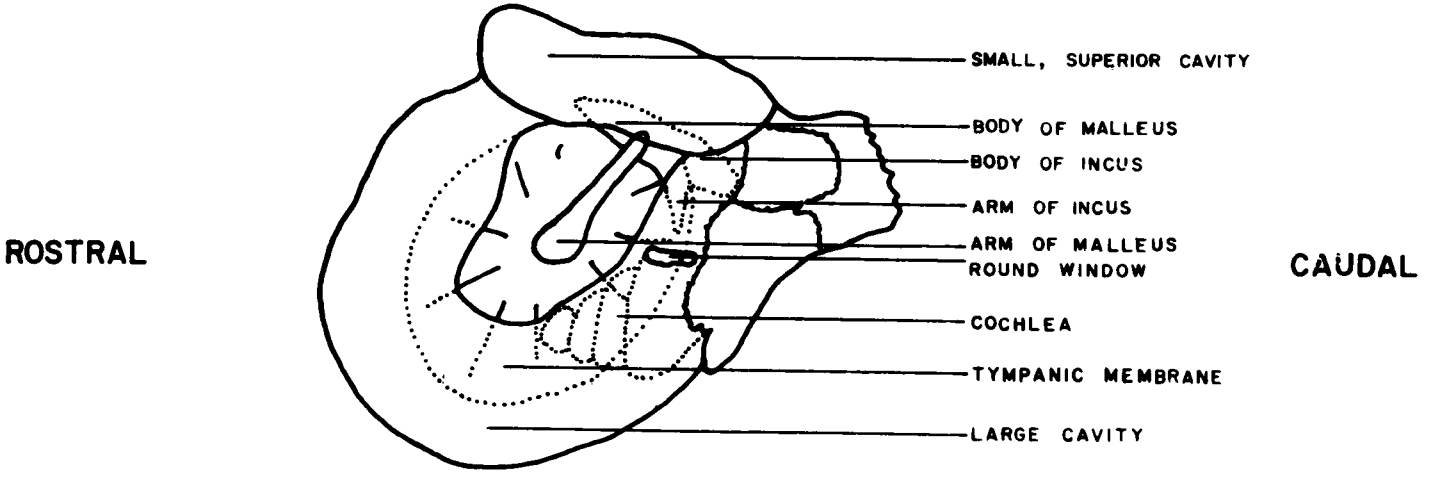

Fig. 1. Schematic drawing of the ear of a guinea pig showing the most important acoustical features.

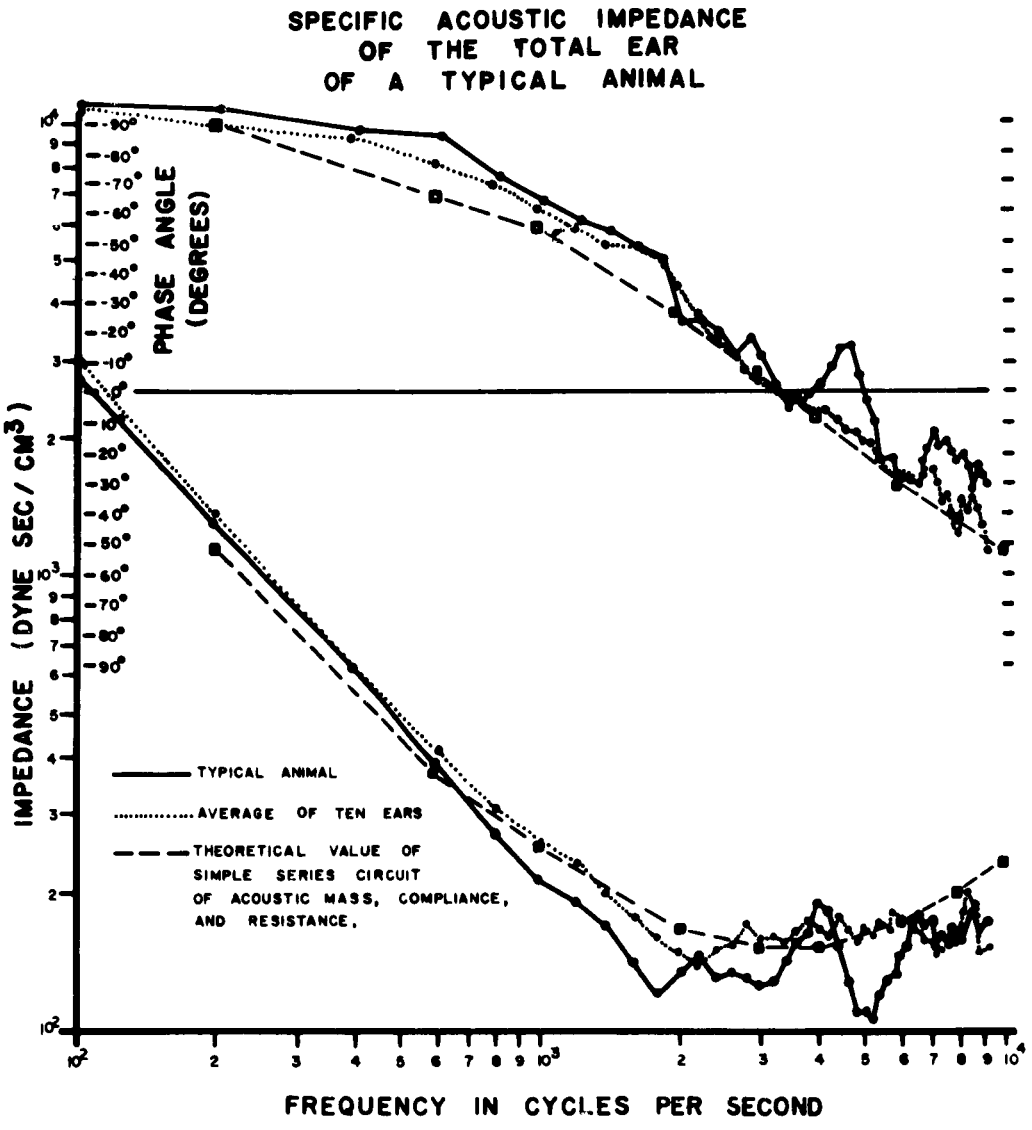

Fig. 2. Specific acoustic impedance of the guinea pig ear. Solid line is of a typical ear; dotted line is average value of ten ears at each frequency. The impedance of the ear is grossly approximated by series circuit composed of a mass of 0.6 mgm, compliance equivalent to 0.25 cc, and a resistance of 40 rayls.

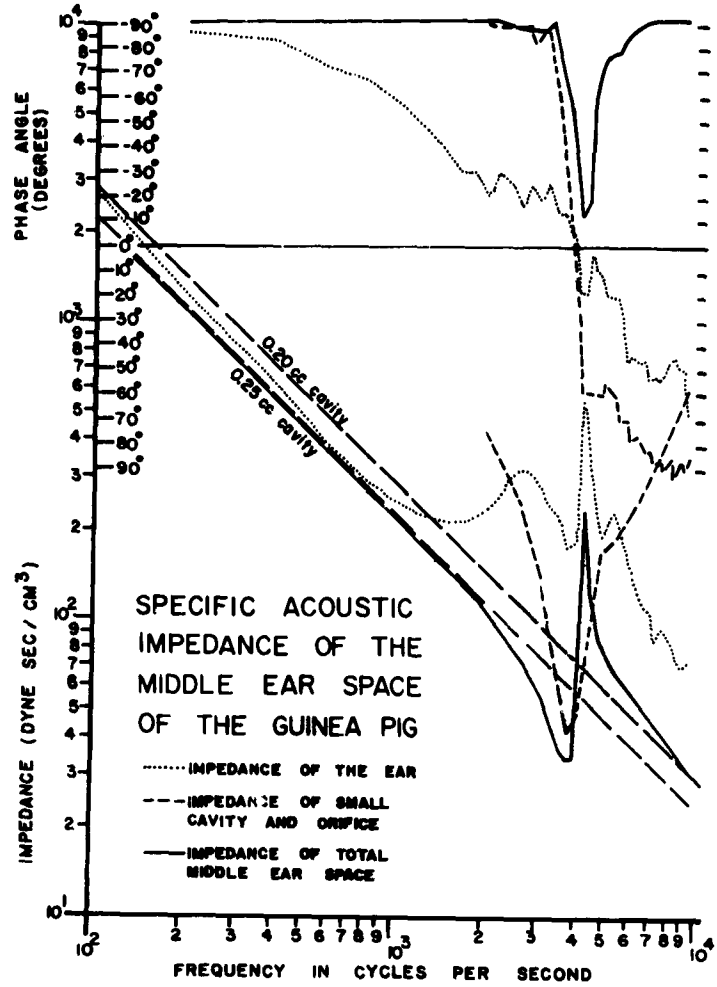

Fig. 3. Impedance of the middle ear space. Short dashed line is impedance of small cavity of 0.05 cc combined with impedance of orifice to this cavity. This impedance is combined in parallel with impedance of large cavity (represented by line marked 0.20 cc) to give impedance of total middle ear space (solid line). At low frequencies the two volumes act as one and impedance is equivalent to 0.25 cc; at high frequencies orifice to small cavity is acoustically closed and impedance is equivalent to large cavity alone. Resonance of small cavity with its orifice produces a minimum and maximum peak in total impedance of middle ear space, which is clearly reflected in impedance of the total ear.

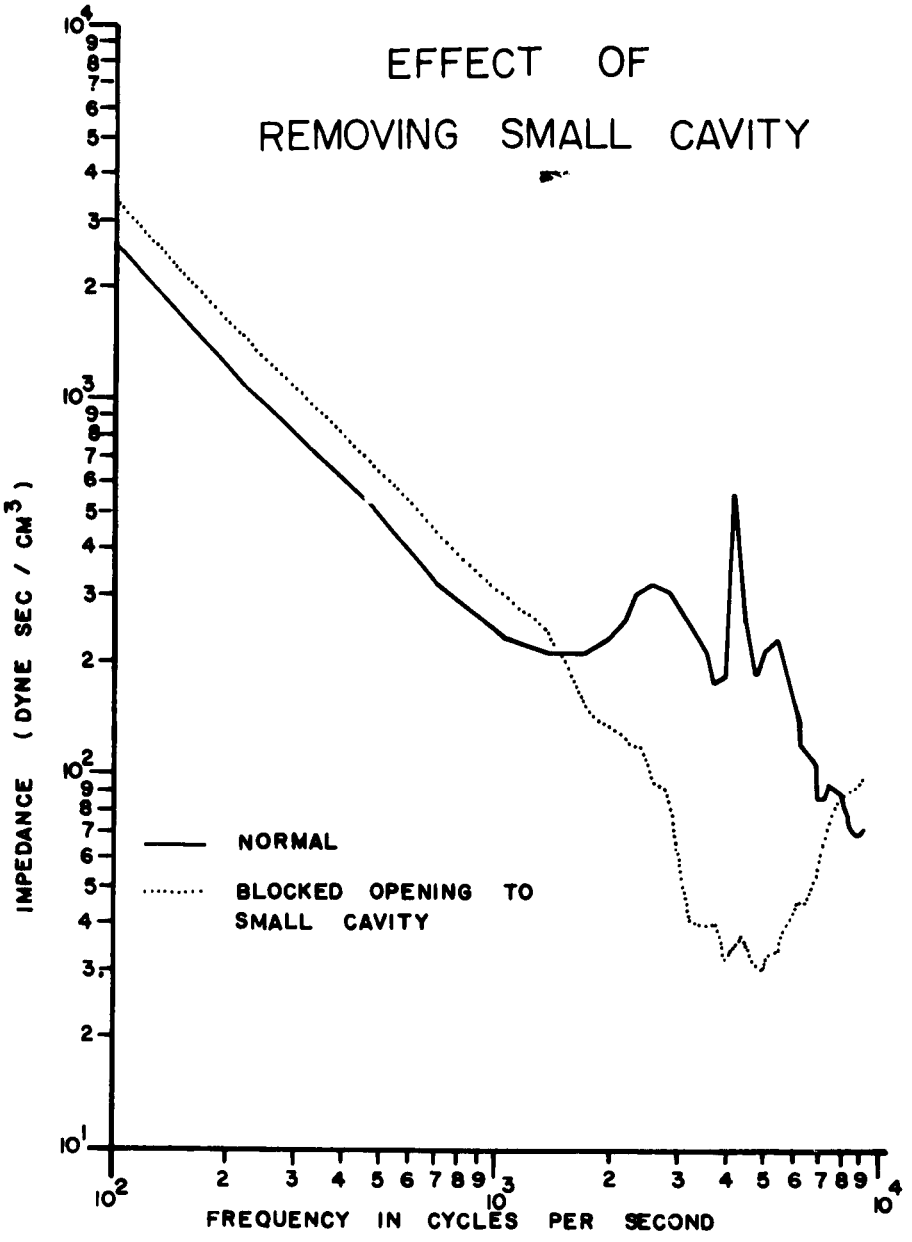

Fig. 4. When orifice to small cavity is closed impedance of the total ear reflects this with an increase at low frequencies, since total volume is reduced, and disappearance of maximum peak.

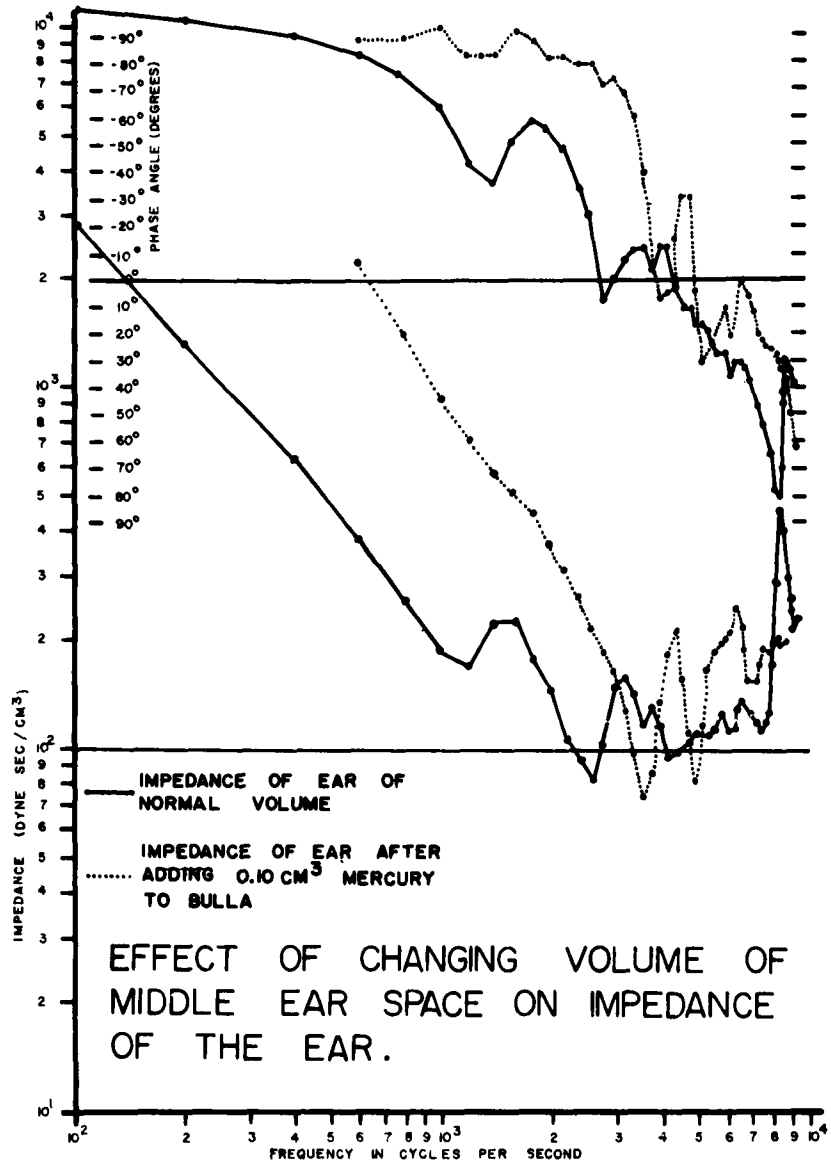

Fig. 5. Decreasing volume of large cavity of middle ear space produces a significant increase in impedance of total ear at low frequencies. Note in this animal maximum peak due to resonance of small cavity and its orifice apparently occurs around 8000 cps; this is highest frequency at which this occurred in all animals studied.

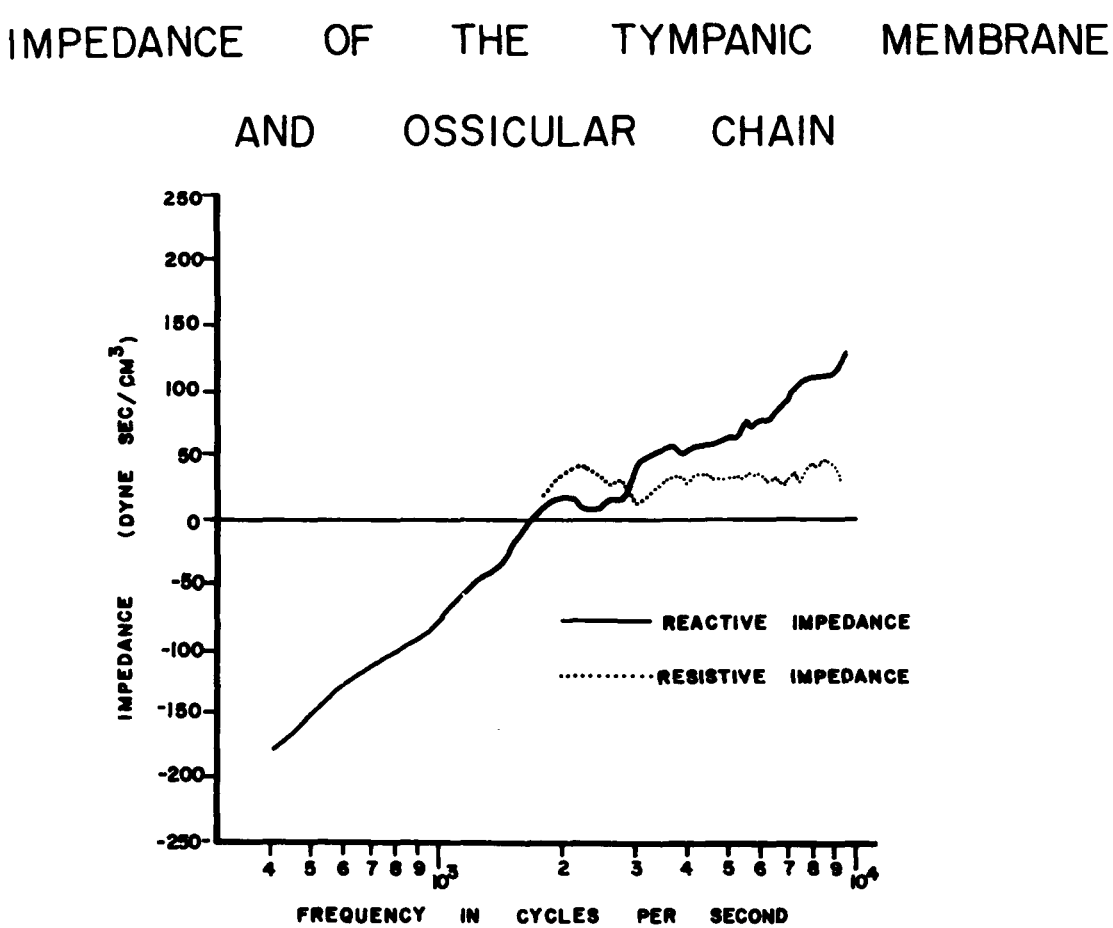

Fig. 6. Impedance of the composite system of tympanic membrane, malleus, incus and ligaments and joints associated with these structures.

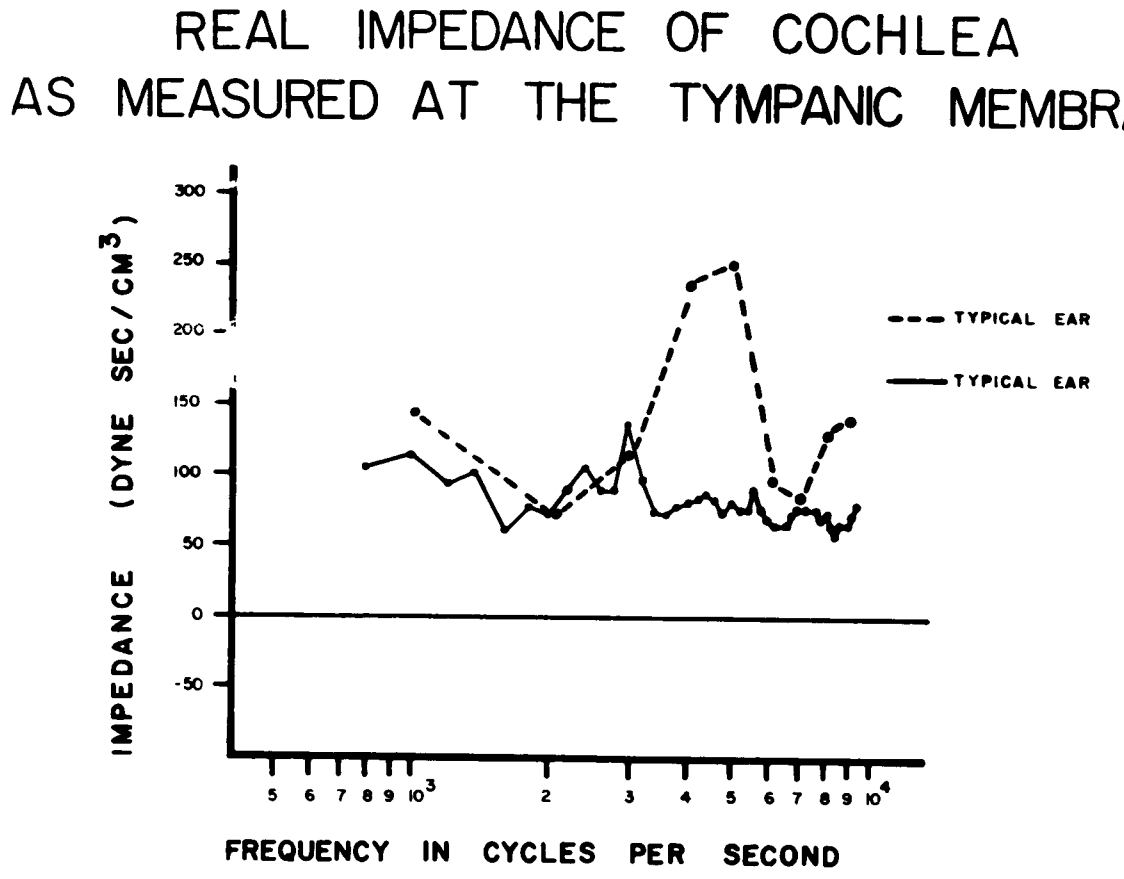

Fig. 7. Resistive impedance of the cochlea, stapes, and stapedius. The maximum peak around 4000 cps corresponds to maximum peak in total impedance produced by resonance in cavity system, therefore, peak in cochlea impedance probably is not correct but is produced by small errors in measurement around this point.

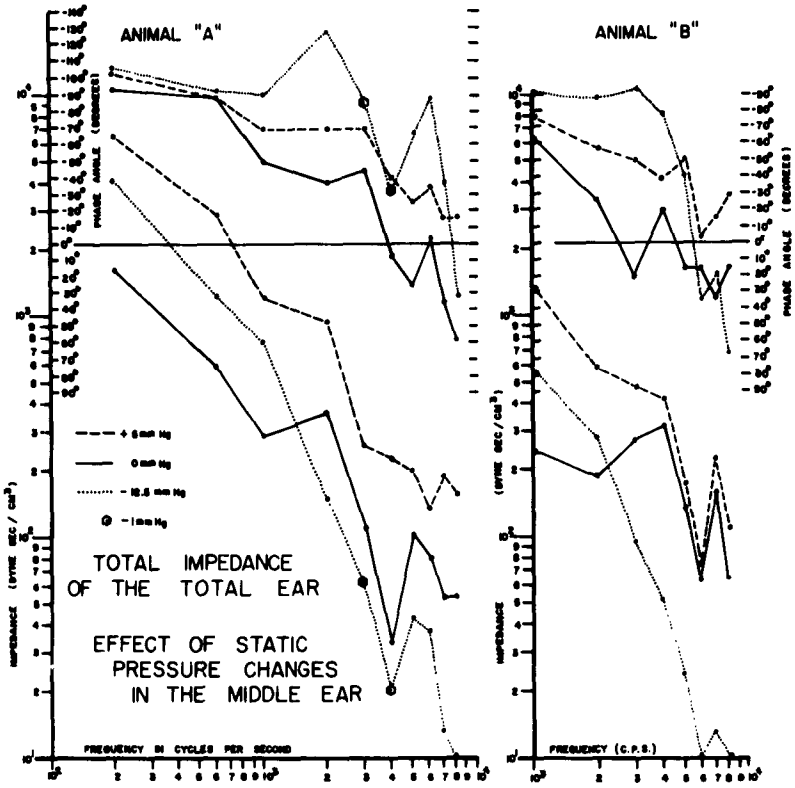

Fig. 8. Increase or decrease of pressure in middle ear space increases impedance of the ear below 2000 cps. Above that frequency increased pressure increases impedance, decreased pressure decreases impedance; "normal" ear apparently can be anywhere between these extremes, but is usually close to increased pressure values, i. e., increased pressure has little effect.

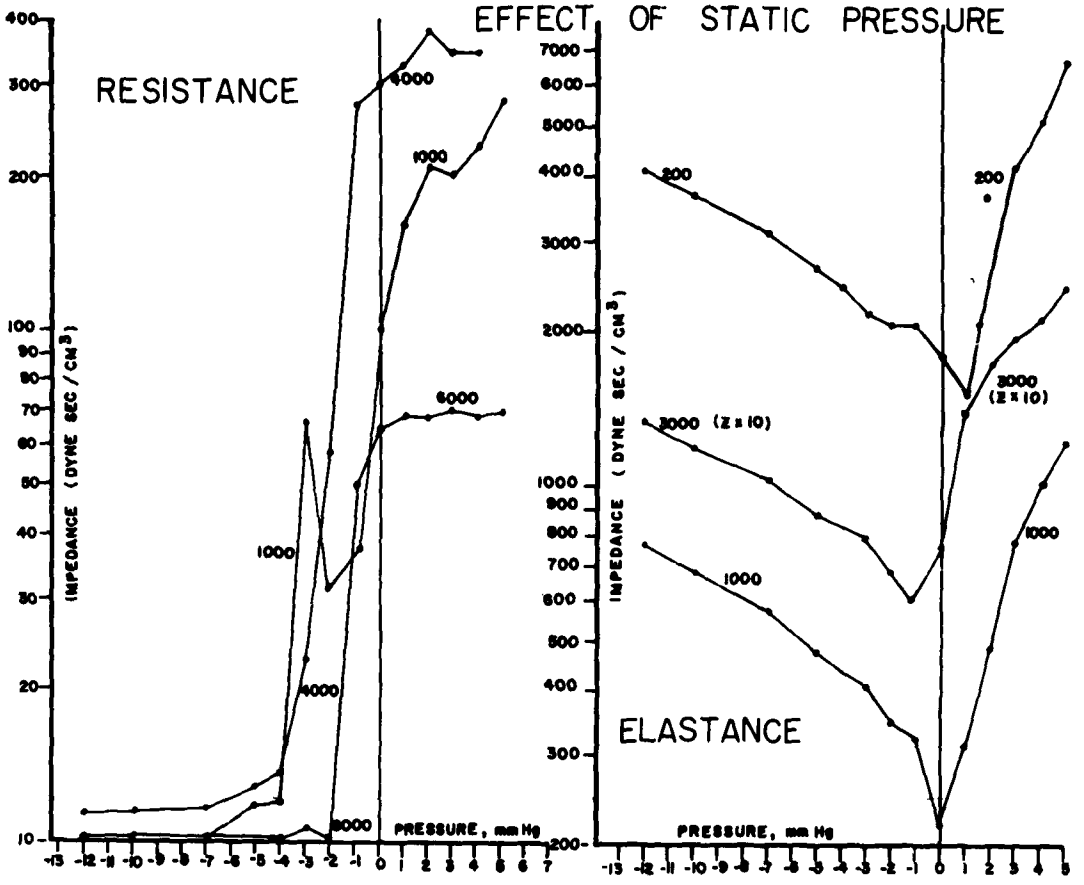

Fig. 9. Elastance of the ear is not symmetrical, being stiffer for outward motion of the drum than for inward. There is a dramatic decrease of apparent resistance of the total ear with decreased middle ear pressure at frequencies where this resistance was shown to be due to the cochlea.

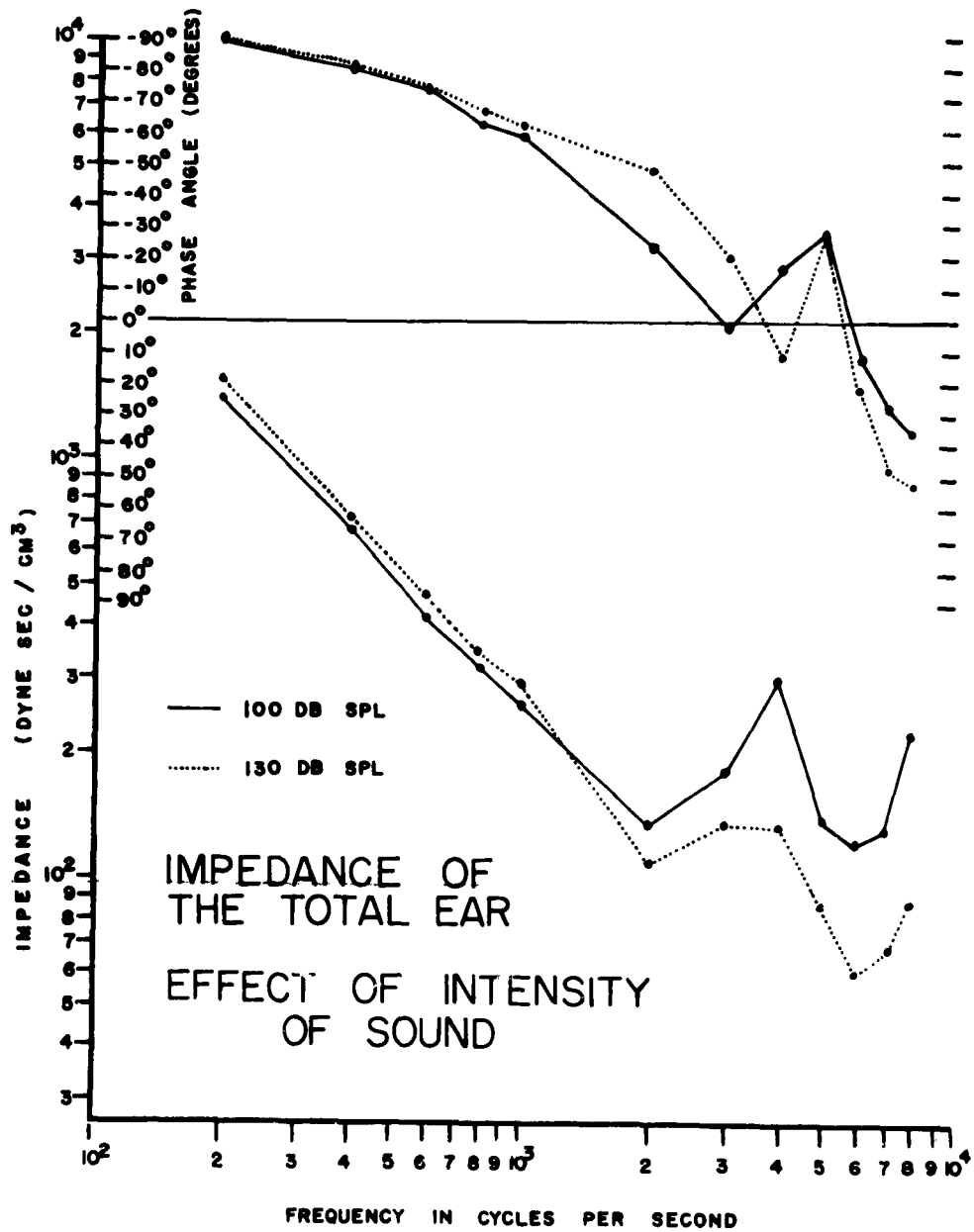

Fig. 10. Impedance of the ear is nonlinear with respect to sound intensity. These changes are consistent with what would be predicted from Figure 9.

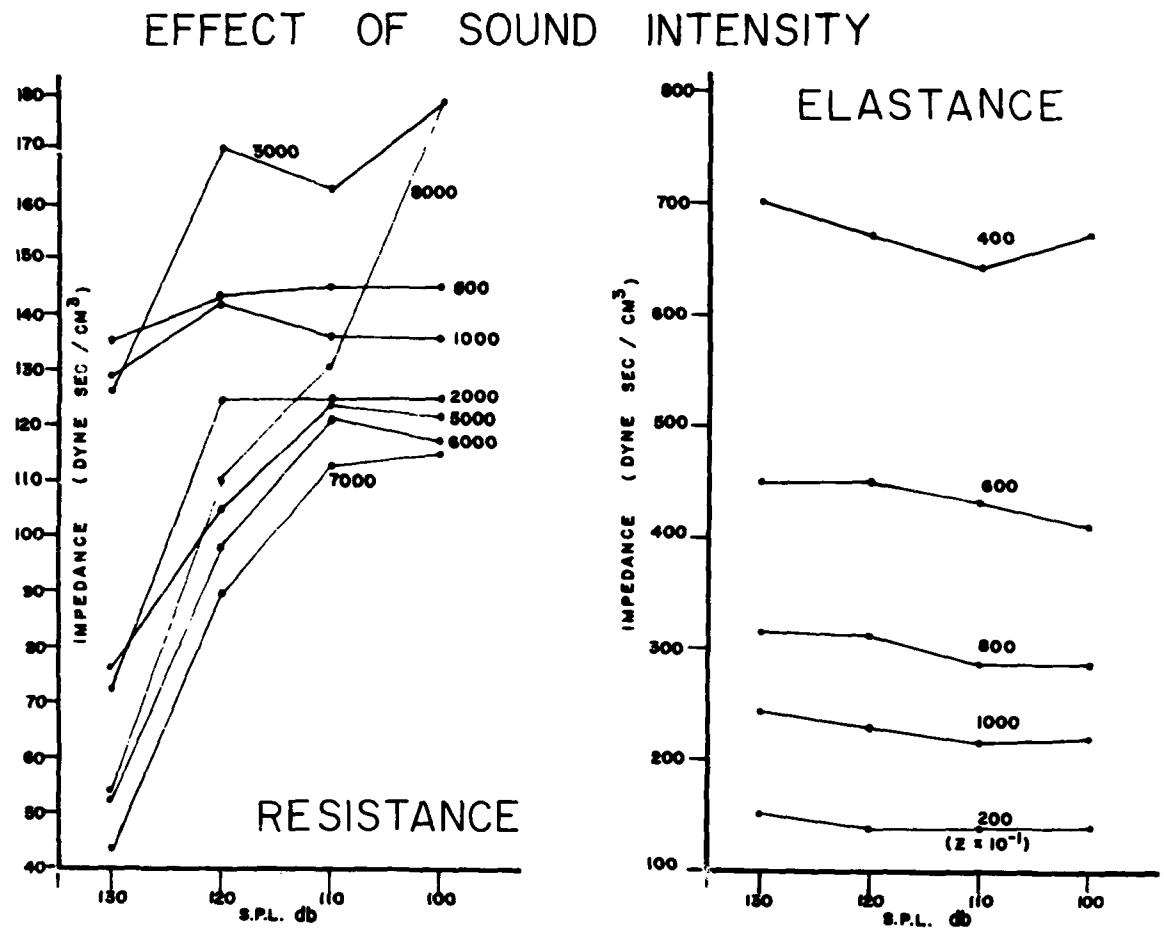

Fig. 11. Nonlinearity with intensity is present in both reactive and resistive impedance, but is predominantly in resistive portion.

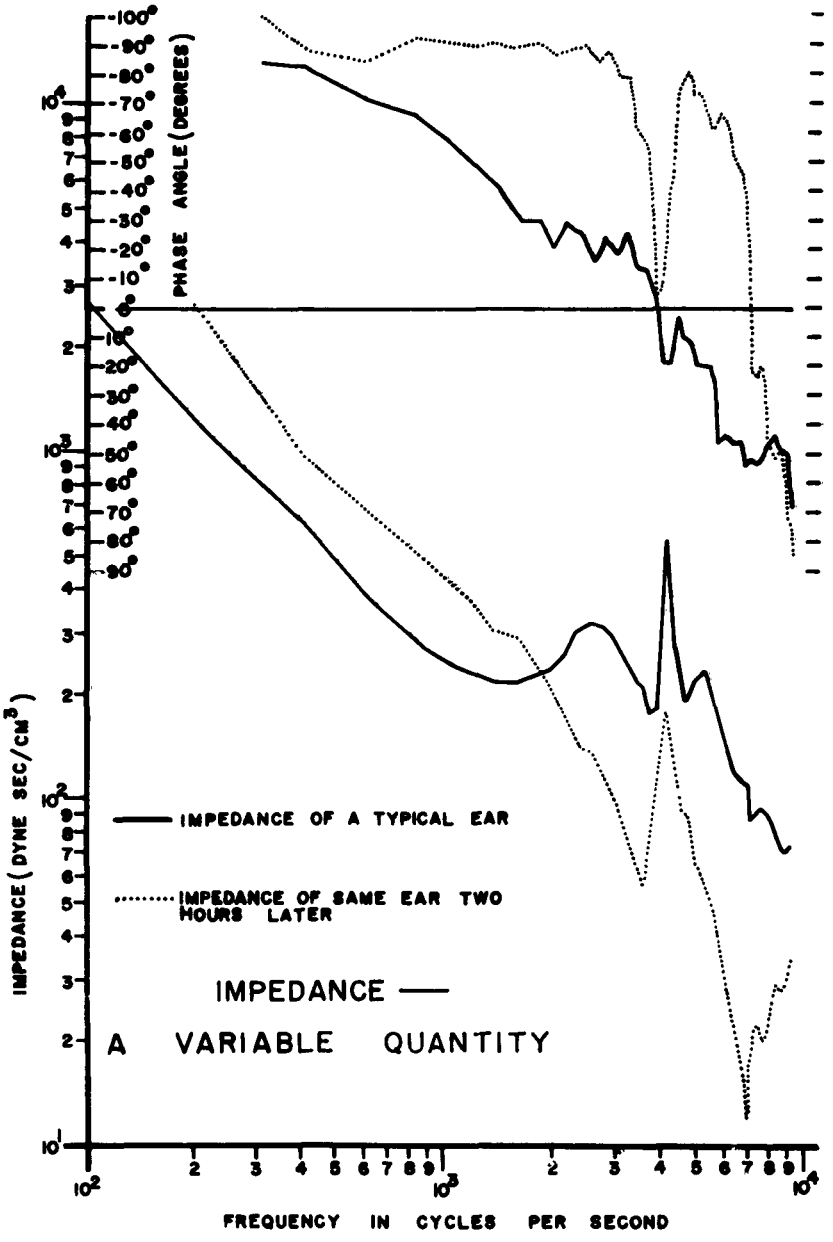

Fig. 12. The impedance of the ear is not a fixed and stable quantity. The decrease of impedance in the "typical ear" above 6000 cps is not a usual finding and probably represents the beginning of the impedance change shown by the other curve.

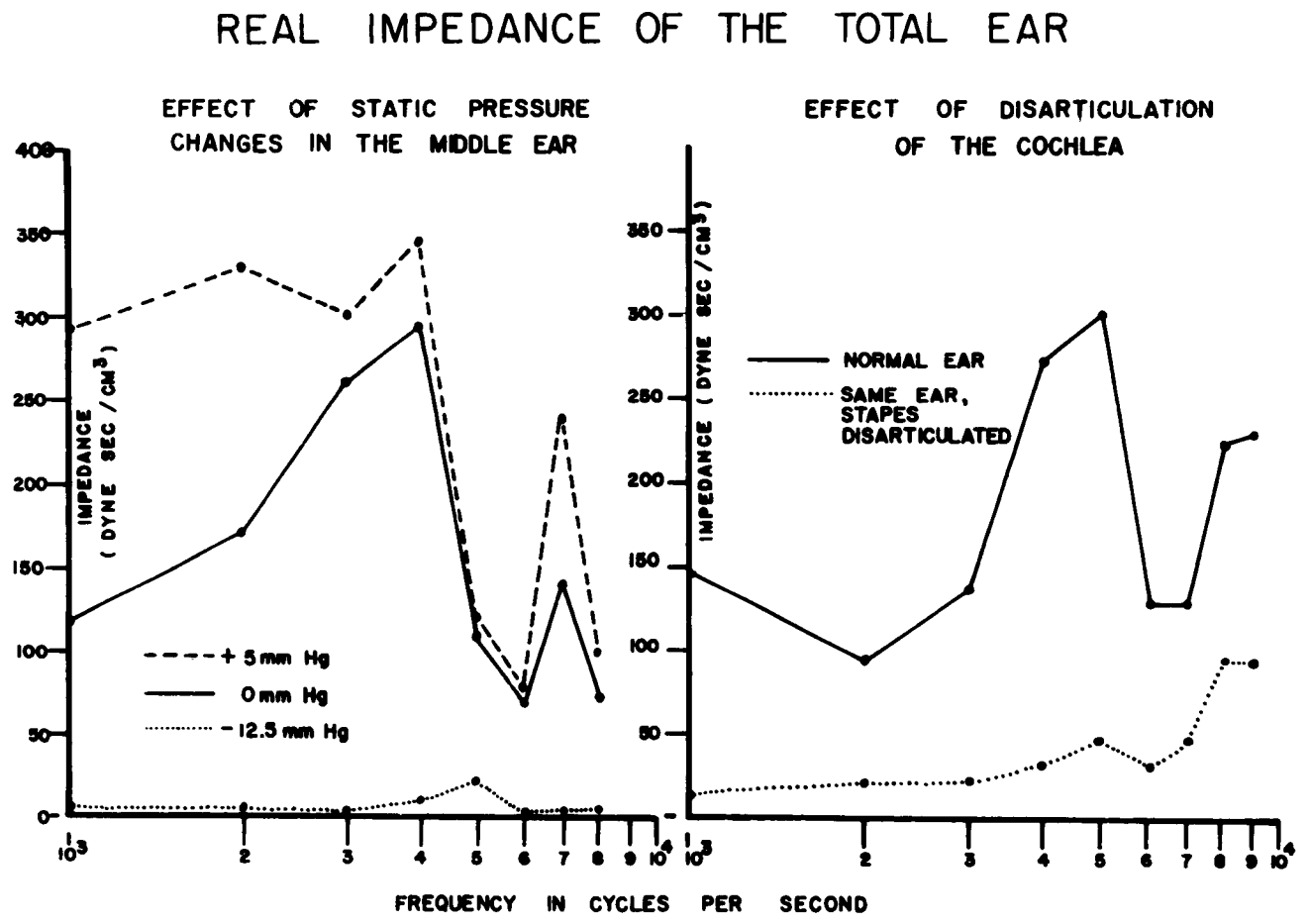

Fig. 13. Decreased pressure in middle ear space and disarticulation of the cochlea from the conductive mechanism produce similar changes in resistive impedance of the total ear.

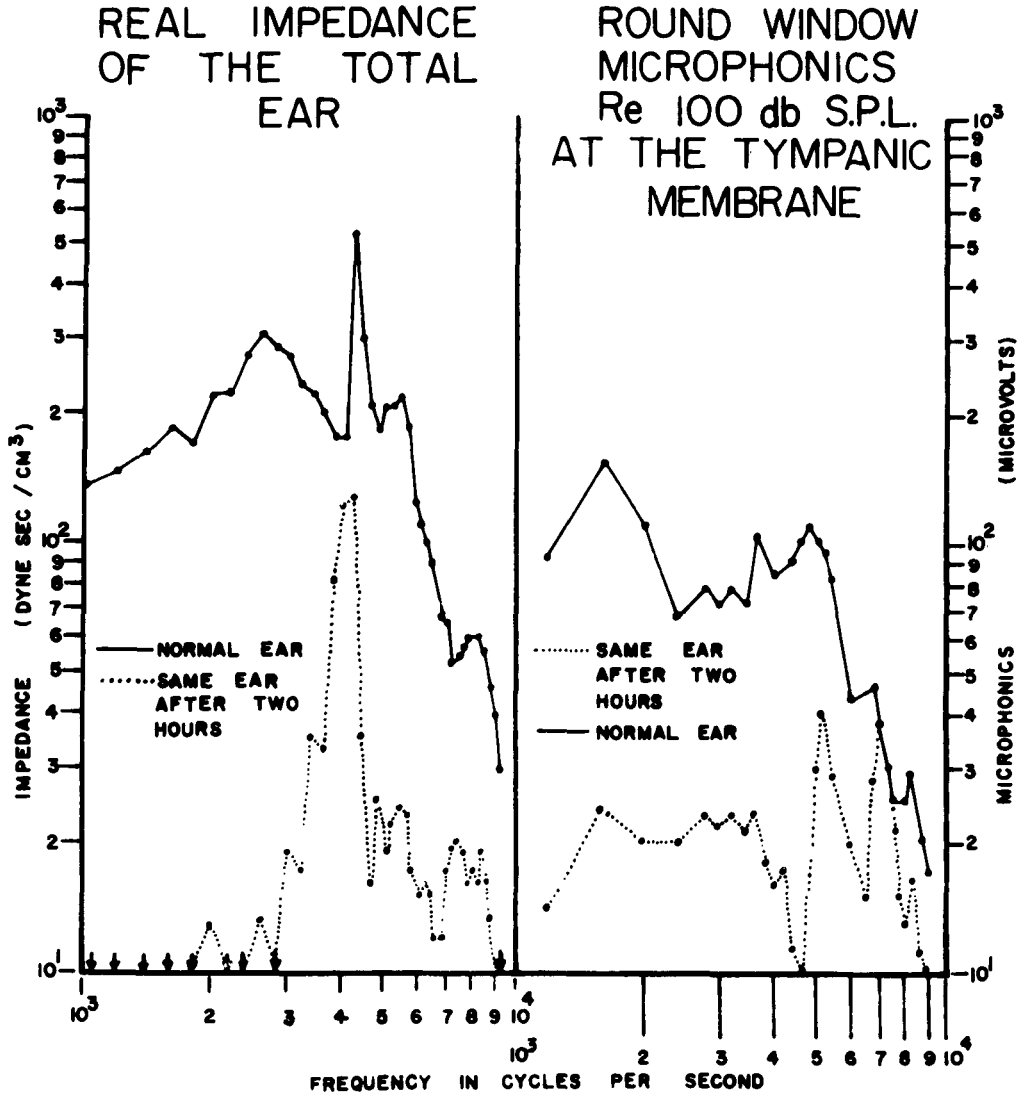

Fig. 14. Round window microphonics are reduced when the resistive impedance of the total ear is reduced.

ACOUSTICS OF THE MIDDLE EAR

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Most of the auditory research centers on psychophysics and neurophysiology. The great unknowns that stimulate the imagination seem to lie in particular in neurophysiology. One may wonder why some of us persist in investigating the sound transmitting parts of the ear, especially after von Békésy appears to have terminated his work on the subject. Yet, there is an unmistakable revival of interest in the mechanics of the ear. No doubt, practical considerations like otosclerosis operations, the use of middle ear muscles for ear protection, and the differential diagnosis of auditory disorders, which is helped by impedance measurements at the eardrum, have contributed to this development.

The state of the art in the investigation of the acoustic properties of the middle ear appears to me to be the following:

1. We know almost completely the middle ear characteristics of the guinea pig.

2. We know almost completely the acoustic characteristics of human cadaver ears.

3. The evidence is accumulating that the acoustic properties of live human ears are different from those of post mortem preparations.

4. An integration of various bits of information concerning the middle ear function of live humans into a coherent formal model is nearing completion.

The discovery that the acoustics of live ears is not the same as that of post mortem preparations has rendered the investigation of the middle ear function more difficult. We may have to wait a considerable time until a direct measurement of the transmission characteristic of the middle ear becomes possible. For the time being, we have to be satisfied with a model derived primarily from the anatomy, and from

measurements of the input impedance of the ear. Such a model may be considered valid as long as it agrees with all the experiments that are available. In order to be quantitative, the model must be basically mathematical. However, the functional structure of the middle ear can be described most easily by means of an electro-acoustic analog. When the analog is realized it can be used for computations of various kinds.

An electrical analog in which a capacitance replaces an acoustic compliance, an inductance an acoustic mass, and an electric resistance an acoustic resistance, appears as the most convenient means of describing the acoustic middle ear function. When the input and the transmission characteristics of an acoustic system are known, it is always possible to devise an electrical network with the same characteristics. The elements of such a model are not necessarily in a one to one correspondence with the elements of the original and their variation does not in general produce the same effects as the variation of the original elements. The usefulness of the model is limited, therefore, to one set of conditions.

In order to produce a middle ear analog that remains valid under most, if not all, possible conditions, the functional units have been matched one by one. A block diagram of the network was derived entirely on the basis of anatomy. The same is true for all the network elements of the blocks, for which sufficient anatomical data could be found. The missing links had to be derived from impedance measurements on normal and pathological ears.

DERIVATION OF THE ELECTRICAL ANALOG

I shall now discuss the systematic derivation of an electric middle ear analog. During this process the effect of various functional units of the middle ear should become apparent.

Figure 1 shows a schematic of the middle ear anatomy. It should not be difficult to see from the drawing that every volume displacement of the eardrum produces a compression of air in the middle ear cavities. This is true irrespective of the mode of vibration. As a consequence, all the current flowing through the electric analog must pass through the network representing the middle ear cavities. Von Békésy has observed that, at low frequencies, the eardrum and the malleus vibrate as a unit. From the anatomical aspect and from observations during otosclerosis operations it may be concluded that the coupling

between the malleus and the incus is practically rigid. Consequently, the eardrum, the malleus, and the incus appear to constitute another functional unit. This unit also includes the ligaments holding the two ossicles and the muscle tensor tympani. The eardrum is not a completely rigid piston, of course, and parts of it can be moved even after the ossicles have been immobilized. The eardrum motion that is not transmitted to the ossicles must be represented in the electric analog by a separate functional unit. The sum of the currents flowing through this unit and through the analog of the malleo-incudal complex must be equal to the current flowing through the analog network of the middle ear cavities. The incudal motion is transmitted to the stapes and the cochlea via the incudo-stapedial joint. Since the joint is not completely rigid, as can be inferred from observations during otosclerosis operations, not all the acoustic energy is transferred from the incus to the stapes. The result for the electric analog is that the stapes, the round window, and the cochlea may be represented by one functional unit, since they all vibrate with the same amplitude; however, the incudo-stapedial joint necessitates a separate unit. The sum of the currents through the two units must be equal to the current flowing through the unit that stands for the malleo-incudal complex.

Figure 2 shows the block diagram of the electric analog. It contains five units, three in series and two in shunt. The order between the units comprising the eardrum and the unit representing the middle ear cavities is reversed in order to make all the current flow through the latter but let only part of the current through the unit standing for the malleo-incudal complex. If the eardrum were a perfect piston and the incudo-stapedial joint were completely rigid, all the current would be transmitted to the cochlea. Consequently, the units simulating the middle ear cavities, the malleo-incudal complex and the cochlear complex are connected in series. The units simulating the coupling between the eardrum and the malleus, and between the incus and the stapes, where some energy leakage occurs, are in shunt. Thus, the block diagram satisfies all the functional requirements.

The next step is a determination of the circuits for the units of the block diagram.

THE MIDDLE EAR CAVITIES

The acoustically important parameters of the middle ear cavities are their volume and their configuration. The total volume of the middle ear cavities was estimated previously to be of the order of 2 cc and to account for the major part of the acoustic impedance at the eardrum.

Last year, Onchi showed that the latter conclusion is erroneous, and our investigation and several others are in accord. Onchi discusses one middle ear preparation with a volume of the middle ear cavities of approximately 3.5 cc. Our own investigations indicate that the average volume is greater still.

Since anatomical sections through temporal bones seemed to point toward a volume of pneumatic cells well in excess of 2 cc, several volume measurements were undertaken on bone preparations and on patients during otosclerosis operations. On bone preparations three methods were used. In the first, four temporal bones were sealed with wax and connected to a vacuum pump. After the air had been evacuated, they were filled with water from a calibrated container. The water level in the container indicated volumes of the middle ear cavities ranging from 2.43 to 10.5 cc. In a second experiment, the middle ear volume of the same bones was measured with an acoustic bridge at low sound frequencies. With one exception almost identical values were obtained. The exception concerned the bone with the largest volume. The bridge yielded a volume of 17.44 cc instead of the previously obtained 10.5 cc. Because of this discrepancy, a third method was applied in which the sound pressure produced at the entrance to the middle ear cavities was compared to that produced in a standard cavity by a high impedance source. The obtained values confirmed the results produced by the acoustic bridge. Apparently, in the first method, the water did not fill the largest volume completely.

It is often risky to apply results obtained on post mortem preparations to living organisms. Consequently, bridge measurements were undertaken on otosclerotic patients. During one phase of the mobilization operation the eardrum is reflected upwards, so that the middle ear cavities become directly accessible. Impedance measurements undertaken on six patients during this phase of the operation produced volumes ranging from 5.5 to 14 cc with a median of 9.3 cc and a mean of 8.7 cc, in excellent agreement with the data obtained on bone preparations.

Having established the total volume of the middle ear cavities, it became necessary to determine its distribution. In agreement with the data given in the literature, the volume of the tympanic cavity was estimated to amount to .5 cc and to be approximately equal to that of the antrum. This leaves, on the average, 7.7 cc for the pneumatic cells, since the contribution of the epitympanum is small. The epitympanic space is almost completely filled by the ossicles, and what remains of it serves as a narrow passage between the tympanic cavity

and the remaining middle ear cavities. As a consequence the general structure is that of two cavities coupled by means of a constriction. Such a structure exhibits a resonance in a certain frequency range followed by an antiresonance at higher frequencies. Onchi has demonstrated that the middle ear cavities behave in this manner, indeed. Unfortunately, Onchi published data obtained on one specimen only, so that they cannot be used as a numerical basis for the electrical analog.

The analog that has been accepted as a first approximation is shown in Figure 3. It contains two capacitances simulating the tympanic cavity and the rest of the middle ear cavities respectively. Their values have been determined directly from the anatomical dimensions. The two capacitances are coupled by an inductance and a resistance, both representing the narrow passage of the epitympanic space. A second resistance bridging the circuit accounts for sound absorption in the porous walls of the middle ear cavities. The inductance has been so chosen that the reactance of the analog shows approximately the same zero crossings as the impedance determined by Onchi. The resistances have been adjusted in accordance with subsequent experiments. The impedance curves of the analog (thin lines) are compared to those of Onchi in Figure 3. Both sets have the same general character, and the numerical difference is chiefly due to the fact that the electrical analog refers to larger cavities than do Onchi's data. Since the effect of the middle ear cavities on the acoustic properties of the middle ear system is small at most frequencies, the achieved results appeared satisfactory for the present phase of investigation.

ANALOG OF EARS WITHOUT INCUS

Ears in which the incus has been surgically removed, without otherwise disturbing the middle ear structures, are of great help in the analysis of the middle ear function. Their analog circuit is limited to three functional units comprising the middle ear cavities, the eardrum and the malleus. It follows from the anatomy that the malleus with its ligaments and with the rigidly coupled portion of the eardrum should be represented in the analog network as a series circuit of a capacitance, an inductance, and a resistance. The portion of the eardrum that is not rigidly coupled to the malleus should be represented by a transmission line. However, at the present level of knowledge the experimental results could be duplicated by means of the simplified circuit of Figure 4. The elements of the two units added to the unit of the middle ear cavities have been determined from impedance measurements at the eardrum. The obtained values appear anatomically reasonable. The inductance L_o corresponds to a mechanical mass of

12 mg, approximately one-half of the mass of the malleus. Since the malleus tends to rotate about its point of gravity, it is to be expected that its effective mass should be smaller than its total mass. The inductance L_d corresponds to a mass of 4.5 mg which is approximately one-third of the total mass of the eardrum.

Figure 5 compares the reactance obtained on the analog to that measured at the eardrum of two patients. Within the range of the empirical data the agreement is almost complete. The same is true for the resistance shown in Figure 6.

ANALOG OF OTOSCLEROTIC EARS

In otosclerotic ears the cochlea is disconnected from the remaining structures, due to the fixation of the stapes. The analog network can be obtained by adding to the network of ears without incus, a circuit representing the incudo-stapedial joint. It could be demonstrated that the addition of the incus has practically no effect as a consequence of the high impedance of the joint. The inductance L_o may be varied over a considerable range without producing any noticeable effect on the input impedance. Figure 7 shows the analog circuit of the incudo-stapedial joint connected to the previously established network. A series circuit consisting of a capacitance and a resistance is the simplest analog of an elastic coupling with friction. The value of the capacitance could be determined from the low frequency impedance of 17 otosclerotic ears, and the value of the resistance from the impedance of the same ears at medium frequencies. Figure 8 compares the impedance curves of the analog with the impedance data obtained on otosclerotic patients in two experimental series. There is excellent agreement between the reactance values. The analog resistance appears somewhat too low. In general the agreement is better with the second experimental series (crosses) performed with improved instrumentation. Figure 9 compares the analog impedance to the impedance of three typical otosclerotic ears. No significant differences between the impedance of the real ears and the analog impedance are apparent.

ANALOG OF NORMAL EARS

By adding to the analog of the otosclerotic ear a circuit representing the two cochlear windows, the input impedance to the cochlea and the column of perilymph that separates the cochlea proper from the oval window, the analog of the normal ear is obtained. There is little doubt that the two windows should be simulated by a capacitance. The

input impedance to the cochlea is a resistance, and the additional mass of perilymph between the oval window and the cochlea corresponds to an inductance. All the elements must be connected in series (Fig. 10). The inductance could be calculated from anatomical evidence. The input impedance of the cochlea has been calculated in 1948. Only the capacitance had to be determined from the low frequency impedance of normal ears.

Figure 11 shows the reactance curve of the analog and the reactance of normal ears obtained in several experimental series. It appears that the analog reactance agrees with the empirical data within the experimental error. The same is true for the resistance as shown in Figure 12.

In order to demonstrate that the electrical analog is not only a model for an average impedance at the eardrum, but that it agrees with the characteristics of a typical ear, the last two figures (13 and 14) compare the analog reactance and resistance with individual data of three subjects.

CONCLUSIONS

The described electrical analog of the middle ear appears to satisfy all the requirements that can be reasonably advanced for a model of this kind. It is in one to one correlation with the anatomical structures. The numerical values of its elements agree with those of the functional units of the ear. The analog input impedance matches closely the acoustic impedance measured at the eardrum. The effect of pathological changes in the middle ear can be duplicated by a corresponding adjustment of circuit parameters. As a consequence, the analog may be regarded as an adequate functional model of the middle ear. As such it may be used for various studies that cannot be done directly on the ear. For instance, it should make it possible to determine the transmission characteristic of the ear, or to investigate the interrelationship between the input impedance and the transmission characteristic in presence of various pathological as well as surgical changes. It could become helpful in the analysis of the function of the middle ear muscles.

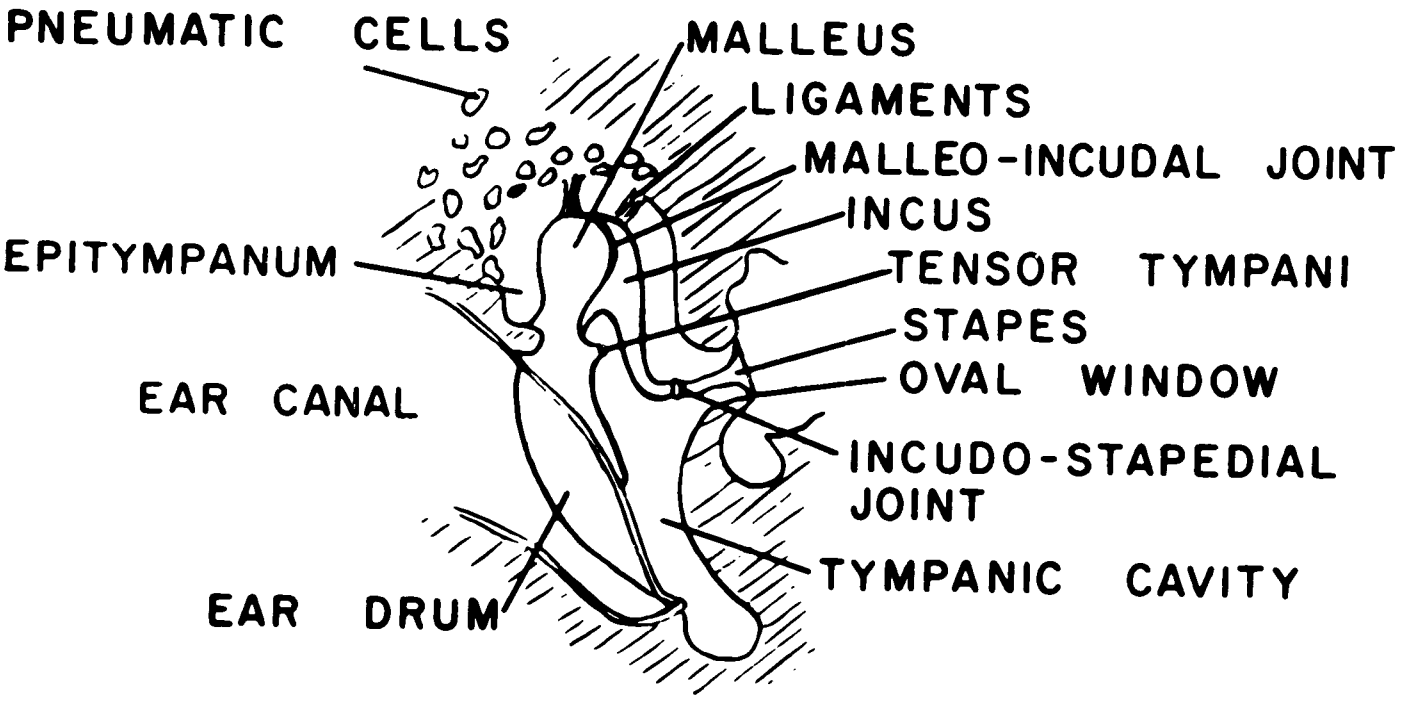

Fig. 1. Schematic of the middle-ear mechanism.

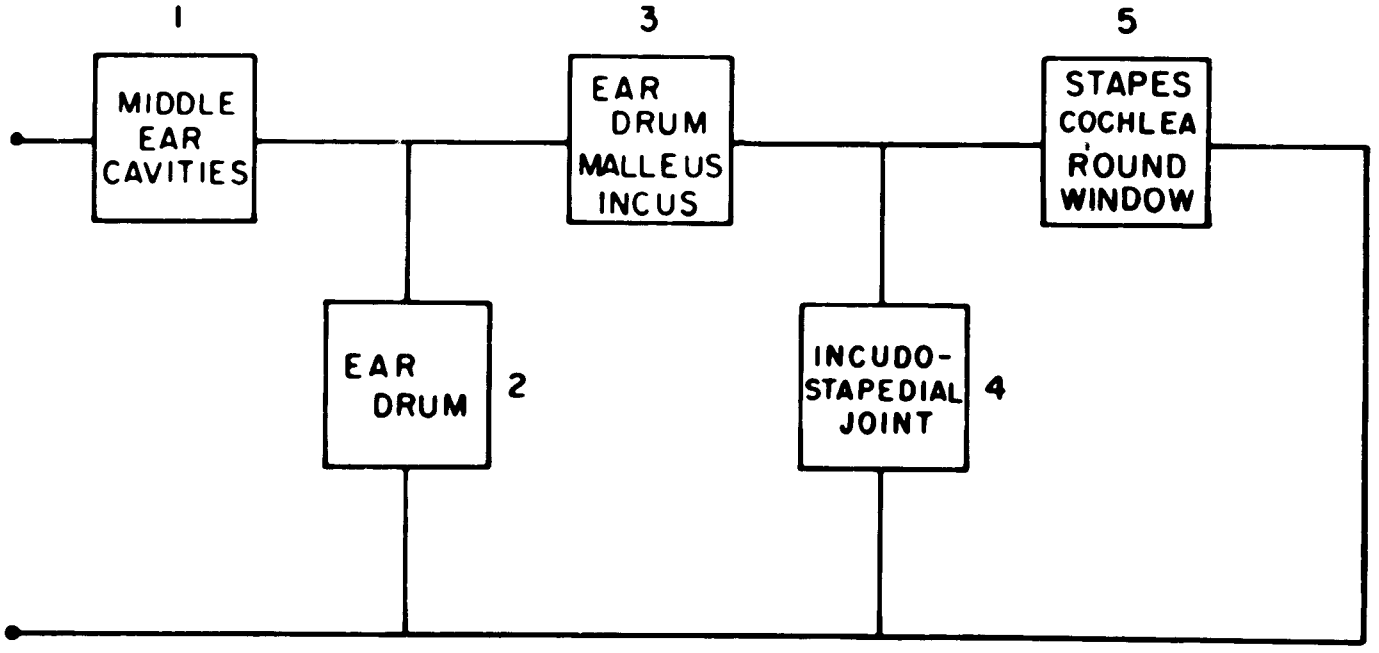

Fig. 2. Block diagram of the middle-ear mechanism.

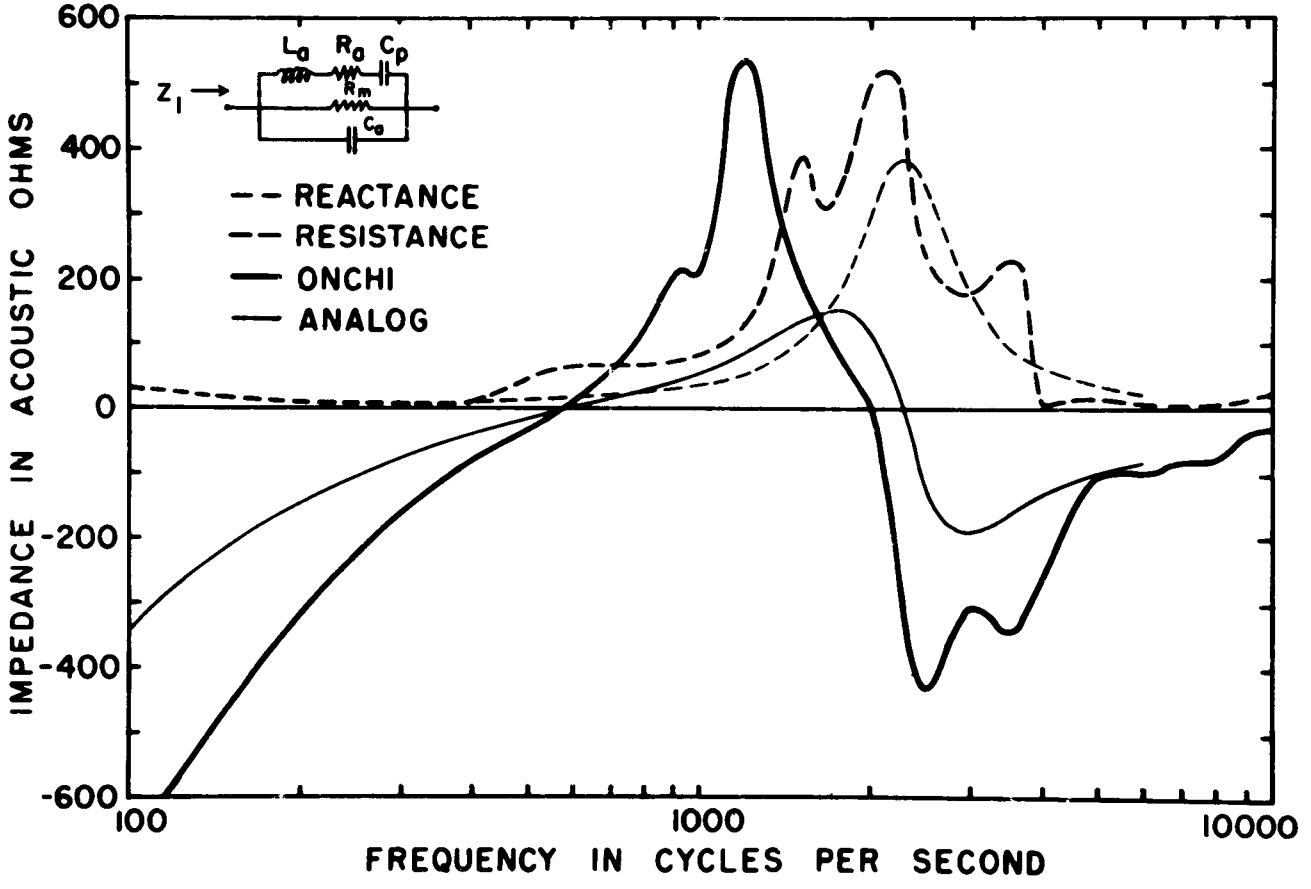

Fig. 3. Acoustic input impedance of the middle-ear cavities and their electric analog. Heavy lines show the impedance components obtained by Onchi on one temporal bone; thin lines indicate analog results based on average data.

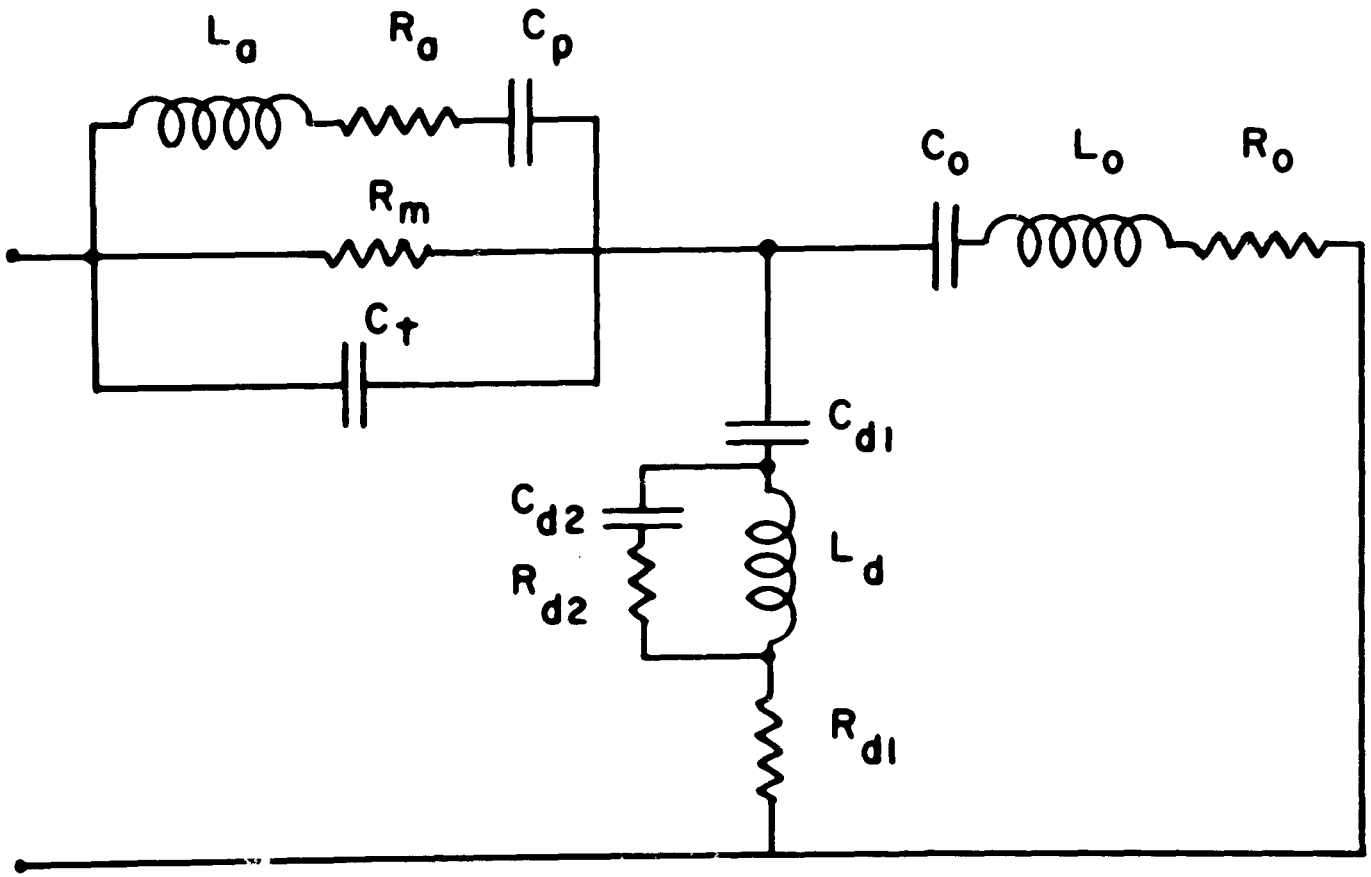

Fig. 4. Electric analog of the middle ear without incus. Elements denoted by subscripts a, p, m, and t belong to the middle-ear cavities, those with the subscript d to a portion of the eardrum, and those with the subscript o to the malleal complex.

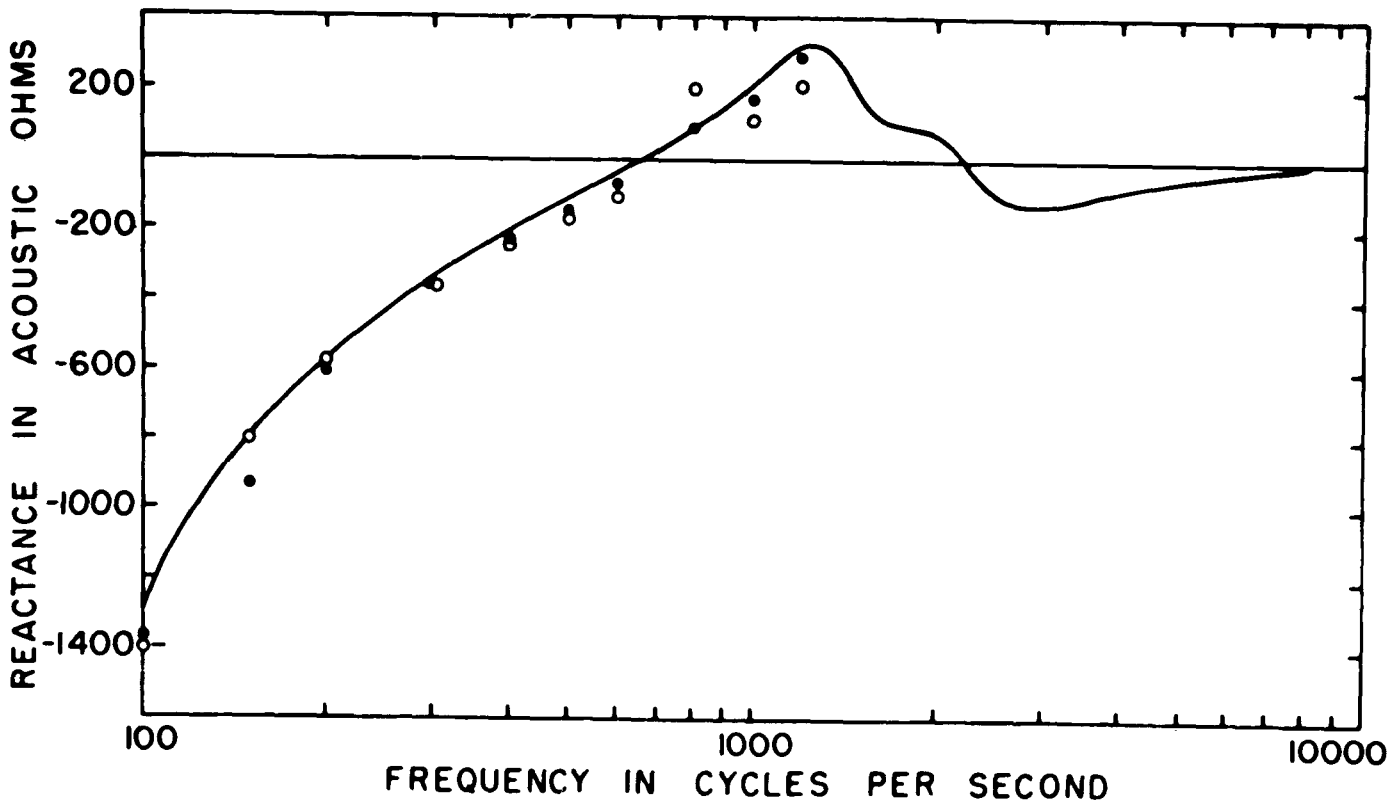

Fig. 5. Reactance at the eardrum of an ear without incus. Points show data obtained on two patients; curves indicate analog results.

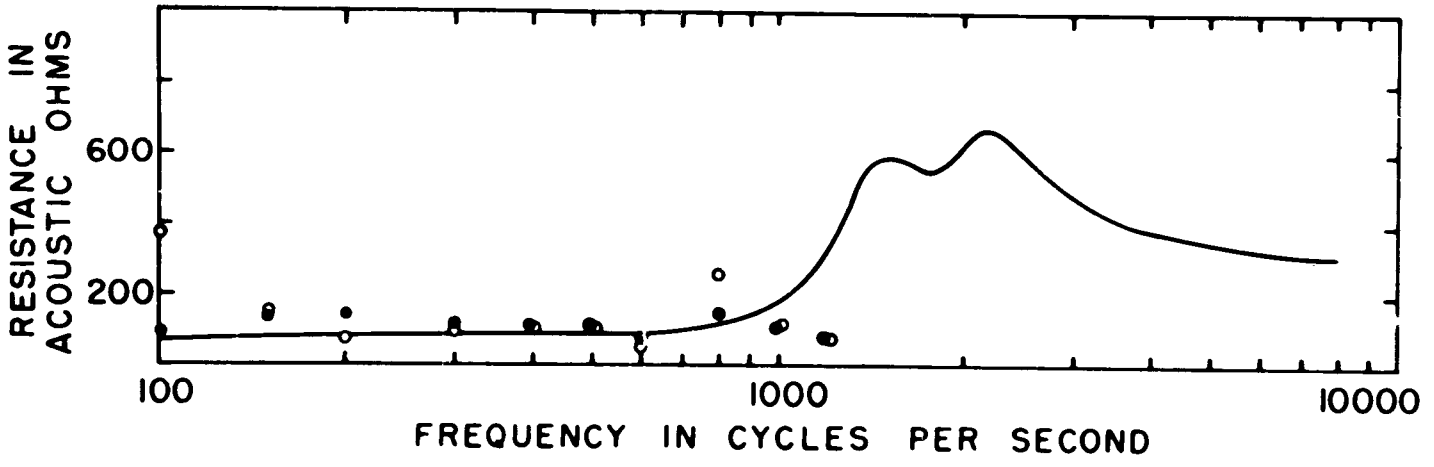

Fig. 6. Resistance at the eardrum of an ear without incus. Points show data obtained on two patients; curves indicate analog results.

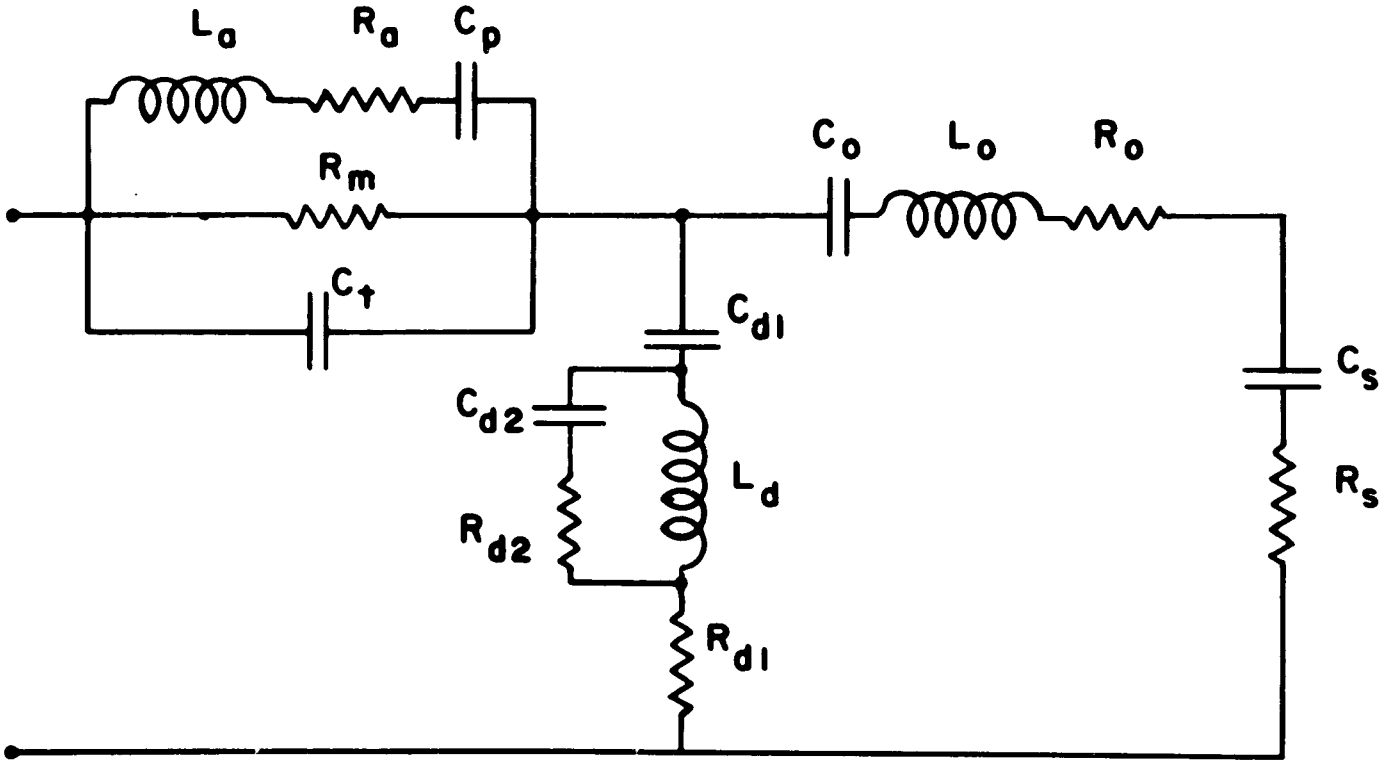

Fig. 7. Electric analog of the otosclerotic ear. Elements denoted by subscript s belong to the incudo-stapedial joint. The other elements are the same as in Figure 4.

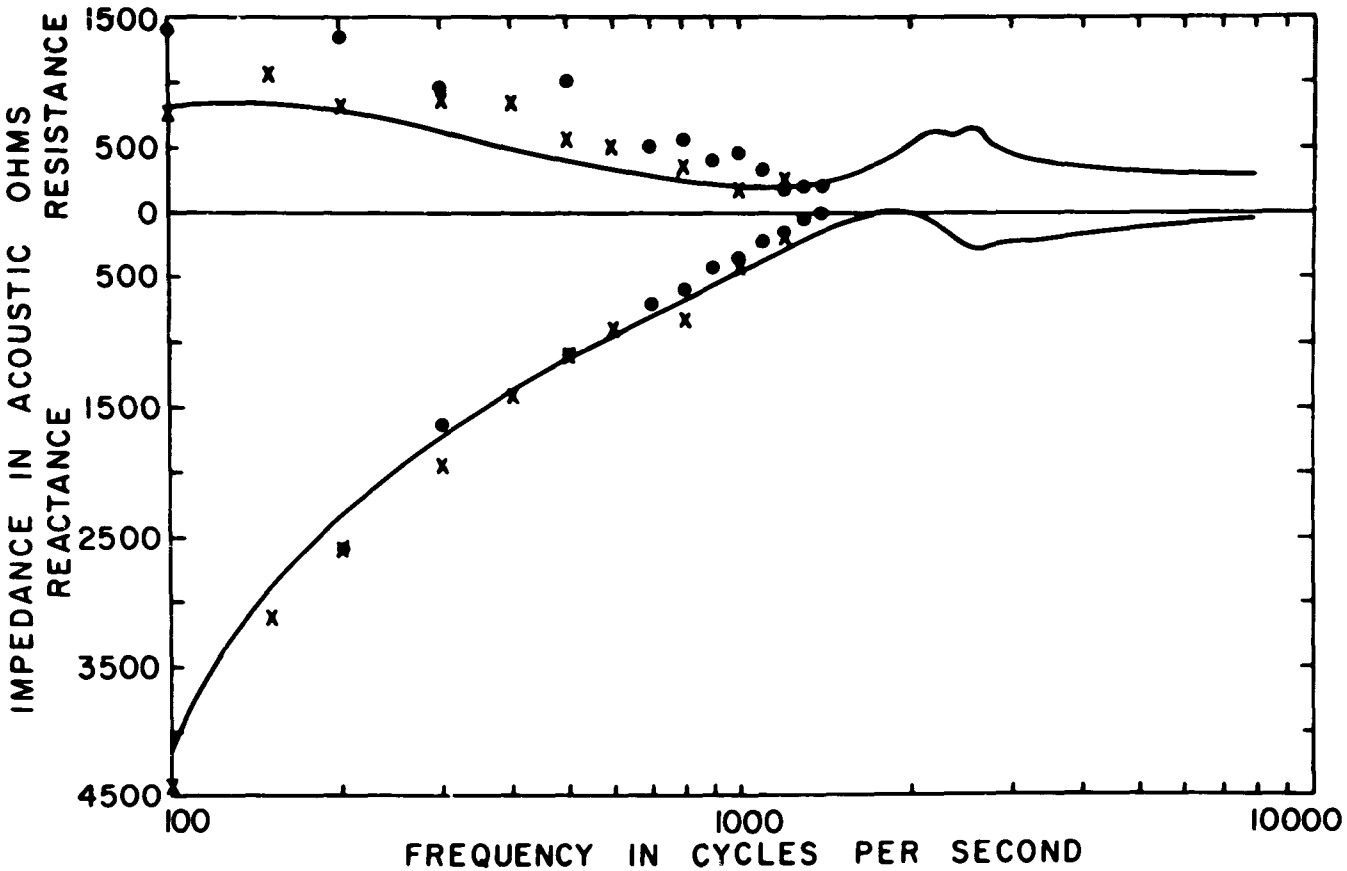

Fig. 8. Average reactance and resistance at the eardrum of otosclerotic ears. Closed circles indicate experimental results of an earlier series; crosses those of a more recent series where an improved technique was used; curves show analog results.

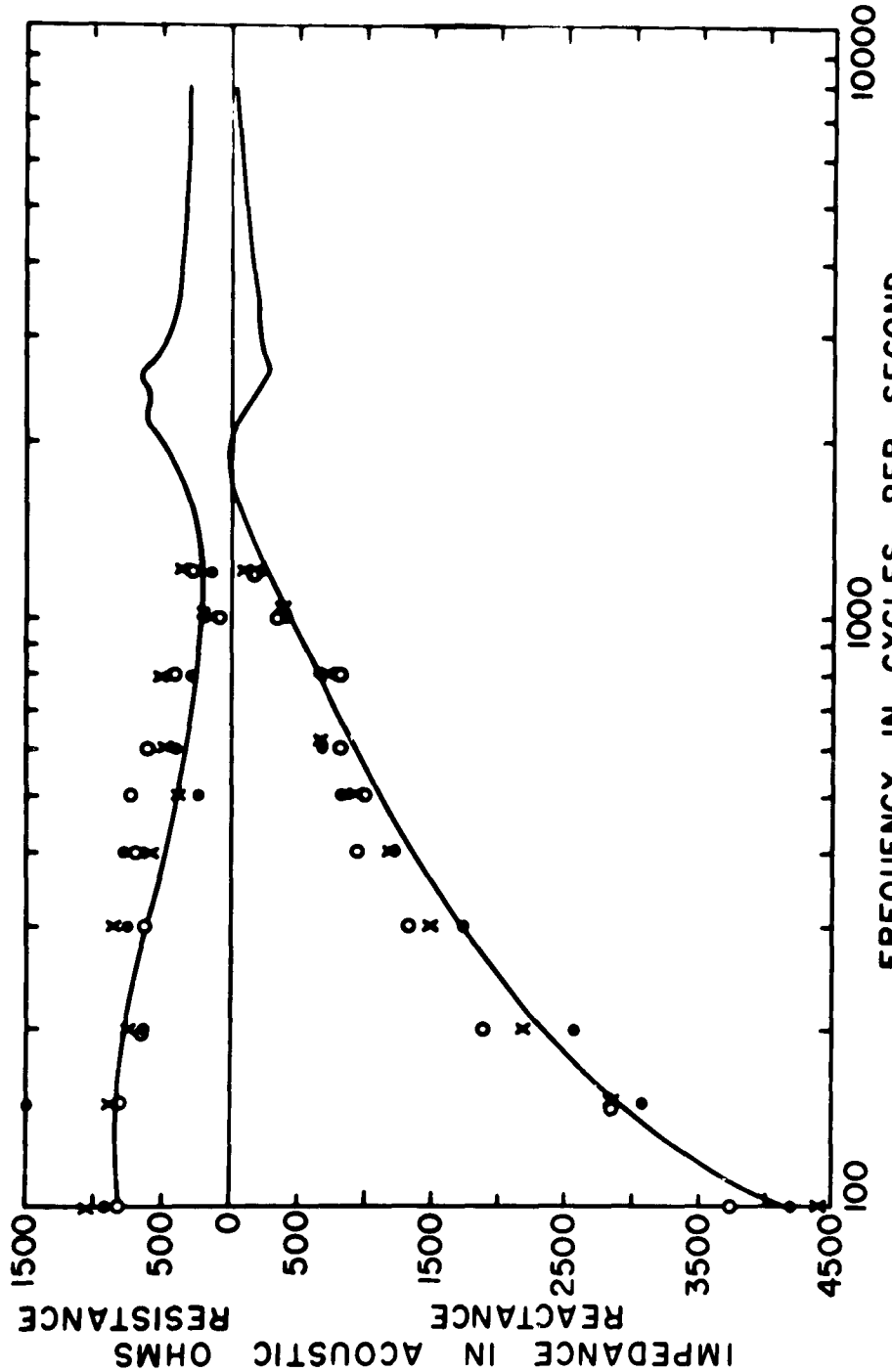

Fig. 9. Reactance and resistance at the eardrum of otosclerotic ears. Circles and crosses indicate data obtained on three patients; curves show analog results (same as in Fig. 8).

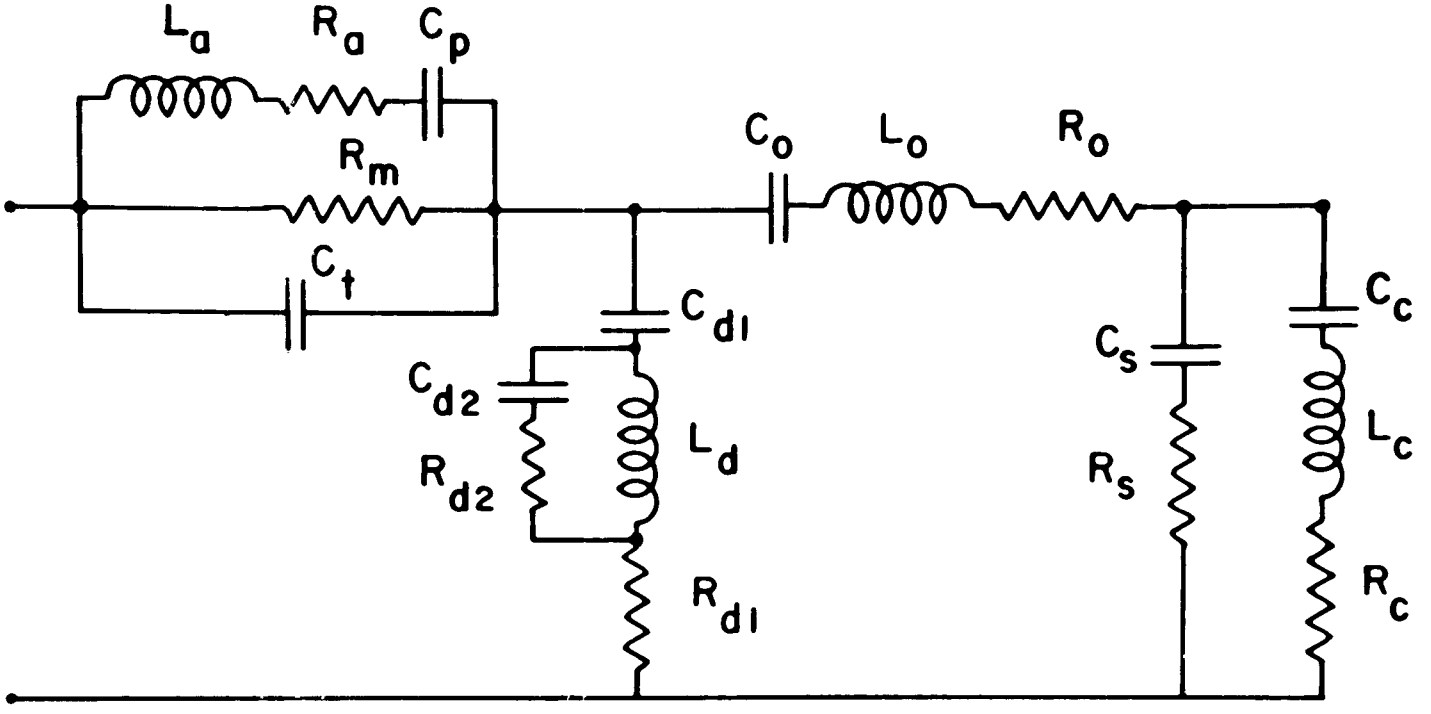

Fig. 10. Electric analog of the normal ear. Elements with subscript c are added to those of Figures 4 and 7; they belong to the cochlear complex.

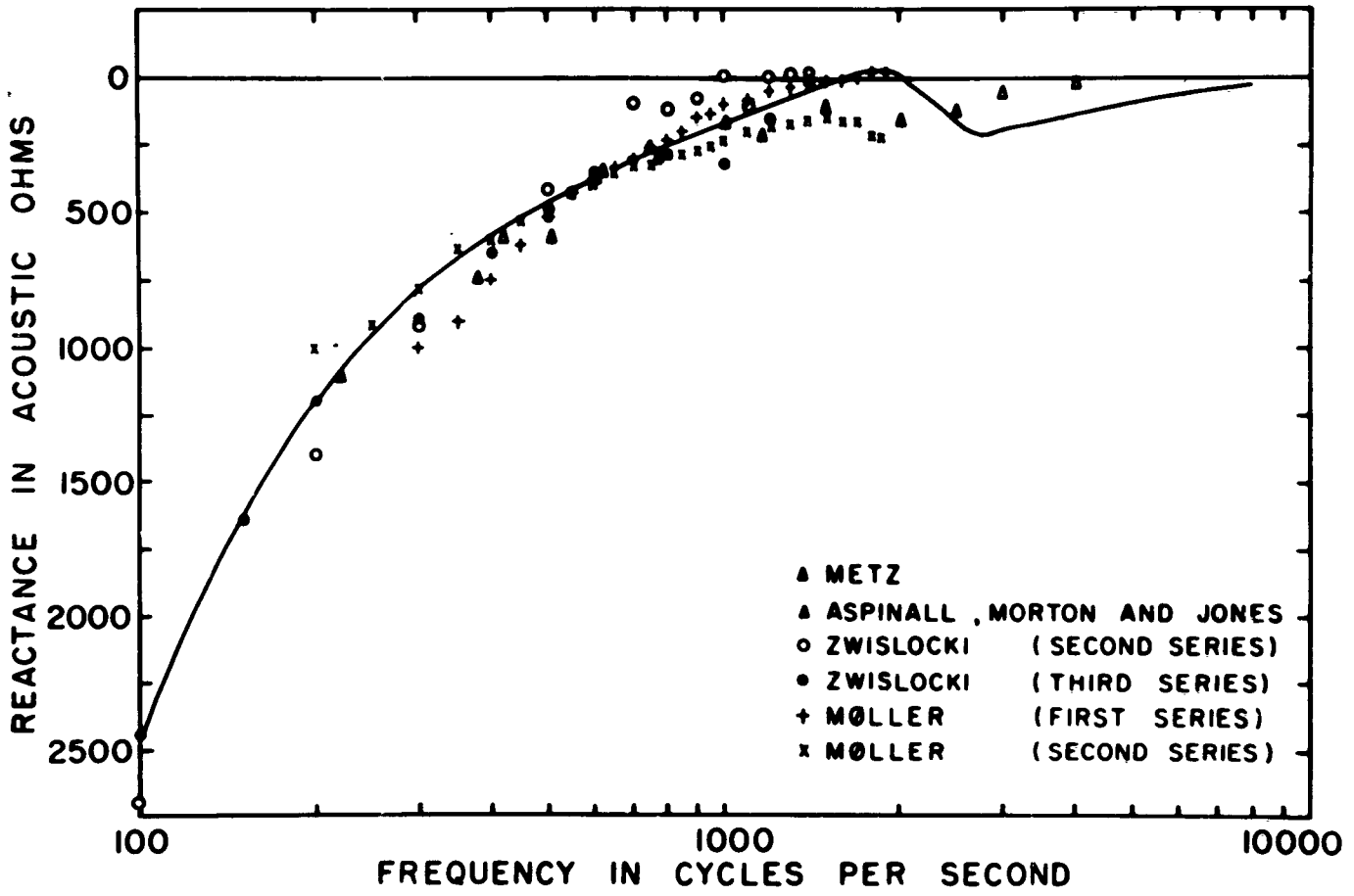

Fig. 11. Average reactance at the eardrum of normal ears. Symbols indicate data obtained by several investigators; curve shows analog results.

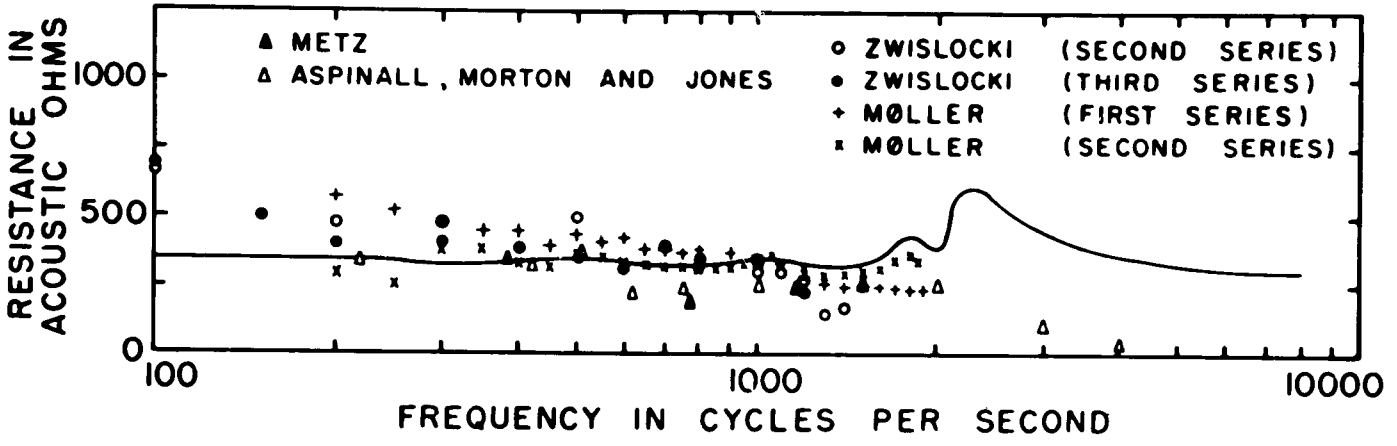

Fig. 12. Average resistance at the eardrum of normal ears. Symbols indicate data obtained by several investigators; curve shows analog results.

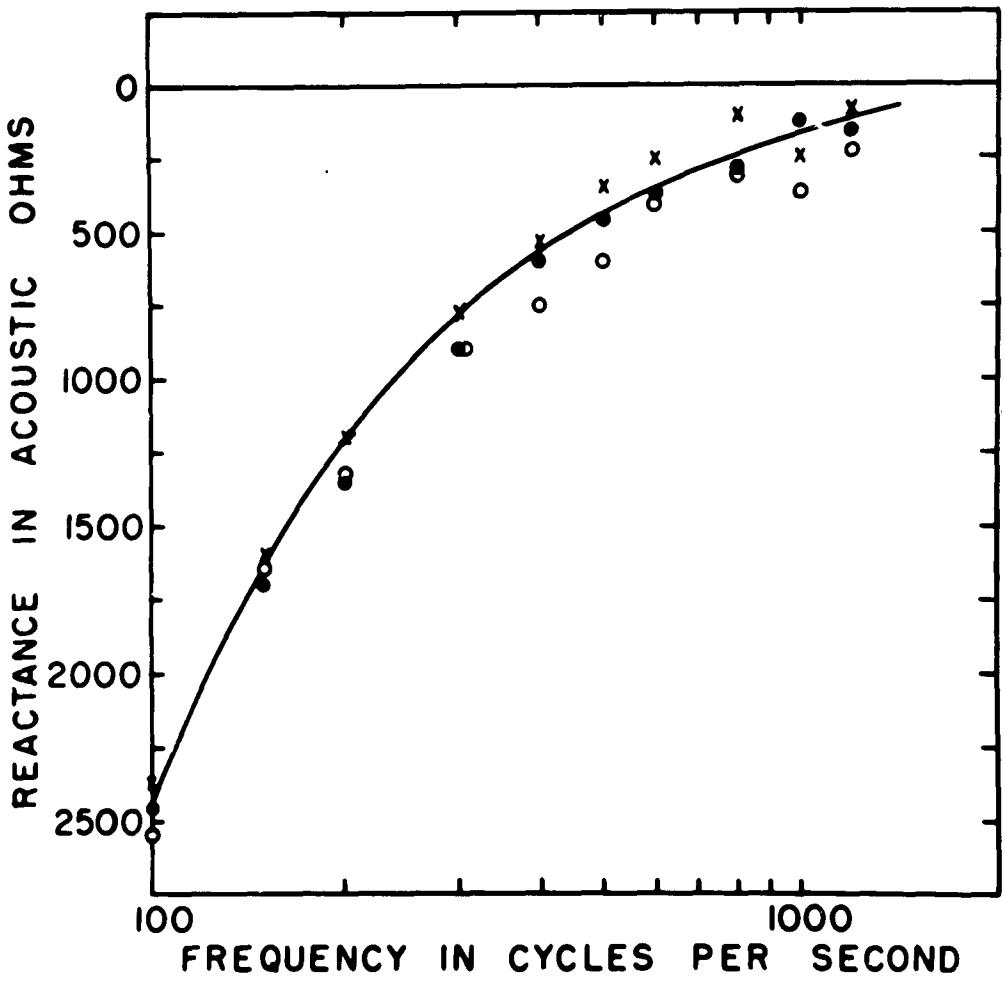

Fig. 13. Reactance at the eardrum of normal ears. Circles and crosses indicate data obtained on three subjects; curve shows analog results (same as in Fig. 11).

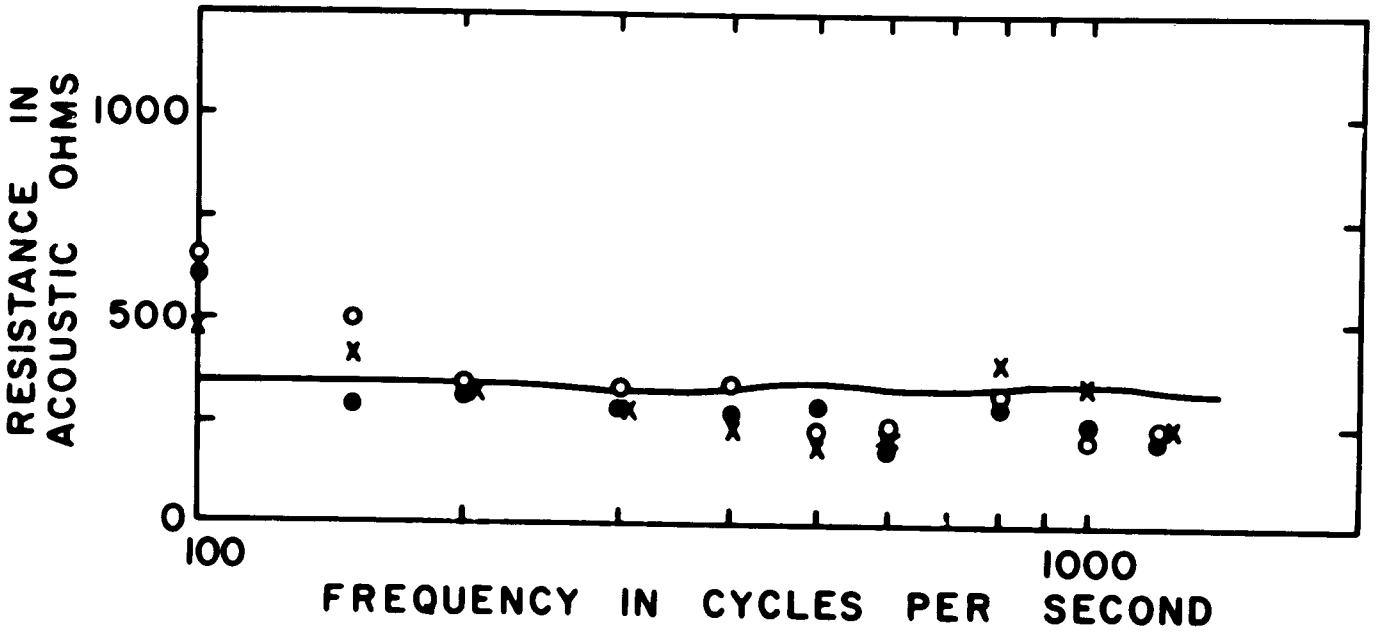

Fig. 14. Resistance at the eardrum of normal ears. Circles and crosses indicate data obtained on three subjects; curve shows analog results (same as in Fig. 12).

THE LABILITY OF THE RESTING AND REFLEX ACTIVITY OF THE HUMAN MIDDLE EAR MUSCLES

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Many of you know that I have been exploring middle ear muscle function manometrically, looking for individual differences in reflex responsiveness. Since the inner ear can derive some protection against extraneous noise through the activity of these muscles, their functional variability may be related to noise susceptibility problems or perhaps to noise tolerance. In keeping with the purpose of this Middle Ear Function Seminar, I should therefore like to run over a few experimental observations and interpretations briefly, and permit you to judge what they may imply regarding this field of research.

The indirect method developed for use in these experiments is based upon optimistic morphological and mechanical assumptions. Figure 1 is a line diagram of the ears which may help toward visualizing the situation. Acoustic stimulation transmitted from the drumhead through the ossicular chain to the inner ear fluid and auditory receptors may elicit reflex contractions of the stapedius and tensor tympani muscles. The tympanic membrane would be drawn inwardly if only the tensor were to contract, and outward movement of the membrane might be expected if only the stapedius contracted. Given the simultaneous contraction of both muscles, either protrusion or retraction could result depending upon relative predominance. Figure 2 illustrates one possibility: Here, a sooner and greater force of the stapedius is shown as being partially negated by a later and lesser force of the tensor, thus causing an intermediate resultant displacement. The chief purpose of this diagram is to warn us that the precise mechanical significance of a manometrically recorded reflex reaction is likely to remain uncertain. Because of this initial uncertainty, the tests were begun under the premise that reflex displacements registered from individual ears might serve as nothing more than an index of the comparative effectiveness or functional equivalence of different acoustic stimuli under controlled conditions.

Figure 3 shows the type of apparatus used to test the validity of the experimental hypotheses. In order to cope with the minuteness

and nature of the biological changes involved, the registration instruments require high sensitivity, low-frequency fidelity, stability, and freedom from random interference. From this point of view, the most notable element in the diagram is the screw micrometer with which the sensing and recording system could be re-checked and re-calibrated at any stage during the course of a manometric test on a subject.

When acoustic stimuli of controlled frequency, SPL, rise time, and duration were delivered to the contralateral ear, characteristic reflex deflections were recorded from the ears of some subjects. The shape of the deflection pattern was fairly uniform in any individual subject, but the magnitude of deflection elicited by presumably identical stimuli varied widely. Figure 4 shows the results of one of several tests in which the conditions were narrowed down so as to reveal the combined effects of systematic and of random influences. This experiment lasting about 2-1/2 hours used a single stimulus rise time, about 225 msec, and a single frequency, 500 cps. Stimuli were delivered to one ear at one intensity in irregularly spaced trains of 6 or 7 before proceeding to another SPL. The downstrokes from the base values shown in the graph represent the peak magnitudes of the reactions to the individual stimulations; the numbers are the mean deflections for the accompanying trains. It is clear that the means for a given SPL remained relatively constant throughout the test session, in contrast to the great variability in the size of reactions to the individual stimulations in the trains. Two additional experiments on the same ear and with closely duplicated test conditions gave about the same results. The averages of the daily reaction magnitudes for each of the intensities have been plotted graphically in Figure 5. The crosses mark the average reaction magnitudes for all three tests. The daily averages appear to be reproducible within an uncertainty range of about ± 1 db from the grand averages. This Figure and the previous one indicate that with very meticulous care the manometric method can yield reproducible quantitative data, although characterized by considerable instantaneous variability.

In Figure 6 normalized average values obtained from the same ear in 9 other experiments have been plotted. It is apparent that each of the three discrete tones and the audiometer masking noise used for stimulation introduce another source of response variability, of which three features can be detected: apparent threshold, dynamic growth range, and maximum deflection. Figure 7 shows a few of the recordings on one of the 9 days, and demonstrates that the reflex deflection-time patterns also vary with the acoustic frequency or spectrum of the stimulus.

However, there may be pitfalls in this type of study of the middle ear muscle reactions. Figures 8 and 9 show that the state of relaxation of the muscles at the time of stimulation must be considered. In Figure 8 we see that a persisting tone of 4 kc permitted a little relaxation to occur within the first half second, while relaxation was substantially complete by about 6 seconds. Yet in Figure 9 it is evident that a white noise may maintain the contractions of the middle ear muscles for many seconds with no indications of relaxation; in this particular test the effectiveness of steady stimulation was being compared with that of interrupted stimuli, but in other tests either type was found to retain its effectiveness for as long as the recording was continued, many minutes.

A less common type of variant is seen in Figure 10, that of the individual's reflex pattern. This subject's tympanic membrane has undergone inward reflex displacements in repeated tests over the past 5 years; but as the tracings show, on one occasion about 2 years ago the displacements were outward. Figure 11 is another example of a change in pattern obtained on the following day in another subject. Still another type of anomaly is shown in Figure 12: This subject had moderately large reflex retractions of the tympanic membrane as a rule, but the 512 cycle tone at 130 db (re .0002 ubar) resulted in much greater deflections; it was suspected that the incudo-stapedial joint became disarticulated when this stimulus was used. The administration of the nasal decongestant Benzedrex by inhalation abolished all observable reflex reactivity of the middle ear muscles for several minutes, after which the large reactions reappeared. From these and related experiences it was concluded that the patency of the Eustachian tube, the pressure gradient across the tympanic membrane, the resting length of the intratympanic muscles, and the pneumatic loading within the tympanic cavity constitute mechanical factors which can modify the appearance of manometric recordings, and that neurohumoral agents may have analogous importance. How such influences may be apportioned between modification of the acoustic stimuli reaching the cochlea and reflex motor effects remains to be determined.

Cochlear fatigue is an intermediate factor in the reflex arc, which may cause variations in response. Figure 13 is a table showing that the recovery rate of reflex excitability may be more rapid than that of auditory acuity, following the induction of fatigue. Other investigators have implicated the higher nervous system as a source of lability of the middle ear muscle reflexes, and more will follow on this subject.

One of the most disquieting aspects in the manometric testing of the middle ear muscle reflexes was that the thresholds appeared to be

considerably higher than those reported by many other workers, particularly those derived in acoustic impedance tests. While most of the apparent discrepancies might have been attributed to the designation of threshold values in different units, e.g., audiometric units or loudness units, without specifying the zero reference, rather than absolute physical values, another systematic error appeared possible also. Transmission across the head of the sounds used in one ear to elicit bilateral reflex contractions complicates the monitoring of acoustic impedance change, conceivably leading to a potential artifact. While such interferences could be discounted in the cases where well designed apparatus was being used properly, the published articles on acoustic impedance tests do not specify freedom from this latent hazard. Measurement of the "thresholds" at which this acoustic artifact appears and at which manometric reflex displacements of the tympanic membrane are observable was of obvious interest. Figure 14 indicates how this simultaneous comparison was made with a slight modification of Zwislocki's Resonator Earplug. (It may be noted in passing, that a tentative proposal for acoustic measurements similar to these appeared in the literature several years ago, but using bone conducted stimulation; a preliminary report by Anderson, Holmgren and Holst showed that characteristic differences had been recorded between normal ears at rest, during acoustic reflex stimulation, and in cases of middle ear pathology, suggesting that this might provide a useful clinical test. It has since been learned from Anderson that it was impractical to standardize the procedure because of the common differences in bone transmission. In the present connection we should note that this proposed method concerns the passive qualities of the middle ear muscles, whereas the manometric and presumably the acoustic impedance method depend upon the active contractions of the muscles.) To return to the diagram, representative recordings obtained from one of several subjects are shown in Figure 15. Clearly, the stimulus strength at which tympanic membrane displacement is detectable is considerably higher than that at which the condenser microphone picked up the sonic vibrations. In the few comparisons which have been made the difference in levels was of the order of 10 to 20 db. This suggests that if acoustic impedance testing should be done incautiously a stimulus artifact could lead to the acceptance of a spuriously low reflex threshold. Of course, acoustic impedance and manometry do not sample the physical events in the middle ear identically, and exact correspondence in results should not be expected. Furthermore, the demonstration by Wever, Vernon and Lawrence that most of the force exerted by the middle ear muscles anchors the attached ossicle more firmly while only a minor component is so directed as to move the ossicular chain longitudinally

probably means that the reflex threshold will appear lower with acoustic impedance tests.

The pursuit of individual differences in middle ear muscle reflexes is therefore attended by many uncertainties. Some of them may be ascribed to the method of test, but others are certainly due to biological intricacies which may affect the resting state and the reflex reactivity of the muscles. However, manometric tests appear to give fairly reliable results, even if their biophysical significance remains conjectural. In order to dispel some of the uncertainties it was desirable to make direct comparisons of reflex measurements in one group of subjects using several different methods. This was accomplished here at Fort Knox last year with the TTS-reduction and contralateral TS methods that John Fletcher and Mike Loeb had been using, and with simplified, mobile manometric apparatus.

Figures 16 and 17 summarize the TS and TTS results, respectively. These are the averages showing, respectively, the effectiveness of 100 db SL narrow band noise, and of 100 and 110 db SL clicks in shifting the threshold of perceptibility of a 500 cycle tone, and the effectiveness of the 100 db noise and the 110 db clicks in preventing the temporary shifts induced by 125 rounds of machine gun fire noise. In the latter testing, each gunfire burst was preceded by 150 msec with one of the acoustic reflex stimuli for comparison with the NO/AR condition. The magnitudes of the individual shifts by the two methods showed no significant correlations, implying that different psychoacoustic phenomena were involved in the two types of tests.

The manometric results appeared to be totally unrelated to either of the other two sets. One possible reason for the complete lack of demonstrable correlation might be that there was none indeed. This seems very unlikely since all three methods had been designed to test the same inherent biological function. Another reason might be that too many of the important factors had been uncontrolled. Still another reason might be that the methods had been inadequate to cope with practical exigencies arising during the testing. Regardless of the causes, we were left with a vast quantity of irreplaceable data from which some hints as to future planning should have been extractable through proper inquiry. This led to what I have named Desperational Analysis:

Figure 18 indicates what was done and how. Every likely factor in the experimental situation was considered and given a dichotomized classification for each subject. This weakened the quantitative value of any single factor, that might have existed, but it reduced the

possibility that any of the items had been graded improperly for whatever reason. Where there was a reasonable doubt as to the cutting value or the aspect that should be used in setting up a factor division, alternate items were tabulated. From this imposing looking but very elementary table, paired frequency distributions of scores were totalled up for each of the subjects, for contingency testing. Figure 19 shows the 136 contingency tables. According to Fisher's Exact Probability Test, the most conservative yardstick that could be applied, 16 of the 136 cells show significance at the .05 level or better. This outcome is several times greater than might have been anticipated on a basis of pure chance. On the other hand, there is no assurance that any of the significant correlations among the paired frequency distributions had not been a chance occurrence, even for the two .005 levels of significance. Nevertheless a few of the close associations shown by the analysis are of more than passing interest.

The population in the combined tests consisted of 21 subjects. Figure 20 is one of the contingency tables, relating contralateral TS with protection from TTS. It will be noticed that only 12 of the 21 individuals showed measurable audiometric losses after exposure to the machine gun noise. Of the 12, 8 received full protection, 4 only partial protection from acoustic reflex activation. But these 4 individuals developed greater contralateral threshold shifts from reflex activation while tracking the 500 cycle tone audiometrically. The tendencies for high TS to accompany less complete TTS-reduction, and for low TS to accompany full TTS-reduction are puzzling. It had been assumed that high TS would go with more rather than less protection against TTS. Several possible explanations come to mind. Perhaps the cochlear receptors were in differing initial states during the two sets of tests. Perhaps the resting condition of the middle ear muscles differed. Variations in the type of reflex contractions might be involved. The 150 msec interval between reflex activating stimulus and each gunblast might not have been long enough. The list of speculative explanations could continue, but it seems wiser to conclude that we need more information to solve the problem.

The three contingency tables in Figure 21 show that poorer audiometric acuity and greater unprotected TTS tended to go together among the 21 subjects. In a number of other studies on other populations and with other types of testing conditions the reverse finding has been reported, namely, that individuals with poorer auditory acuity develop lesser sensory deficits in fatigue tests. However, there are many dimensional complexities to consider. While the initial losses (10 db or more) were numerous among the 21 subjects, they were relatively

mild. Unprotected TTS also appears incipient rather than well developed in the population, as suggested by the fact that 9 of the 21 had none at all. Two categorical differences should also be kept in mind, one that we are dealing with explosive noise, rather than with steady tones or continuous noise; the other that these subjects had become more or less habituated to this type of auditory stress.

Figure 22 contains 4 contingency tables and suggests a greater complexity of interrelationships than we may be wont to think of. From rather rough estimates of the chronic noise exposures of the subjects, tendencies are indicated for more severe chronic exposure to accompany higher reflex thresholds and a characteristic that I have labelled "Tonic Contraction." It is reasonable to guess that habitual exposure should result in just these two features, high thresholds and a state of tonic contraction of the middle ear muscles. To the right of the figure we see again that the two trends of heightened muscle tone and higher reflex thresholds are associated closely, in the present data. Perhaps a word of explanation for the tonic contraction term is necessary. It has been applied in the cases where the pattern of the manometric recordings was particularly vigorous and particularly transient, in contrast to the other subjects whose records all resembled the ones shown in Figures 7, 8, 9, 10, 11, 12 and 15. It may be of interest to note that the pattern of reflex displacement was outward, suggesting predominant contraction of the stapedius muscle, in almost every instance interpreted as signifying tonic contraction. It would seem that the individuals with the high thresholds and the more forceful looking reflex tracings had developed a protective conditioning of the middle ear muscles. The sudden impulsive sounds to which the population was continually being exposed with or without advance warning would have had the most serious effects upon the sensitive structures to the inner ear when the middle ear muscles were maximally relaxed. To have protective value against this type of noise, the muscles would have to have been in a state of resting contraction, i.e., muscular tone. The adaptation to noise, about which many observers have remarked and which is evidently not always dependent upon the development of irreversible hearing loss, could be explained by an imperceptible conditioning of the middle ear muscles. This possibility merits direct inquiry in future investigations.

Another apparent behavioral adaptation was observed in one of the control subjects studied at ACEL before the combined tests were undertaken at Fort Knox. This man had high reflex thresholds. Partway through each of two manometric test sessions, the intense stimuli were followed by a series of spontaneous contractions, as indicated by vigorous, transient retractions of the tympanic membrane. The

twitches stopped after several minutes of quiet, but reappeared when stimulation was resumed, this time with stimuli 15 or 20 db below the reflex threshold. It still required the stronger acoustic values to elicit the smaller reflex retractions which had first been observed. After these tests had been completed, it was learned that the subject had been a sonabuoy instructor for a year or more during a previous duty assignment. Similar manometric results have not been noted before or since in recordings from over a hundred subjects. It is suggested that sonabuoy detection may be facilitated by sporadic contractions of the middle ear muscles. The ability of the reflex contractions to convert the ossicular mechanism into a high pass acoustic filter has been noted by many observers. In sonabuoy listening, the problem is to detect low frequency components within a background of mixed noise. It seems likely that sporadic contractions of the tympanic muscles would favor the discrimination of low frequency bands or peaks in mixed noise through the agency of a temporal comparison in the central nervous system. This could be one of the unappreciated results of training, experience and practice. Analogous spontaneous contractions in the middle ears of guinea pigs and sporadic contractions in the middle ears of bats and owls have been described, and these might represent similar accommodative mechanisms related to evasion and hunting, respectively.

In briefest summary, four points should be noted.

1. Each method of studying middle ear muscle activity in man may depend upon a different combination of factors, each with its own individual variability.

2. The resting activity of the muscles may modify the value of acoustic stimuli, and may also bias the possibility of measuring the effectiveness of the stimulation.

3. The concept of integrative as contrasted to differentiating studies of middle ear muscle function becomes implicit in the observations cited. This calls for control data regarding (a) auditory acuity with an absolute acoustic reference value, (b) the subject's previous exposures to fatiguing noise, (c) characterization of the chronic exposure, e.g., steady mixed noise, explosive or impact noise, siren noise, and so on, (d) estimates of past needs for protection against noise and for accommodations to execute auditory tasks in the noise, and (e) the resting condition of the middle ear muscles during testing.

4. Facts relevant to distinctions in individual noise tolerance may emerge from proper investigation within the broad frames of reference which have been implicated.

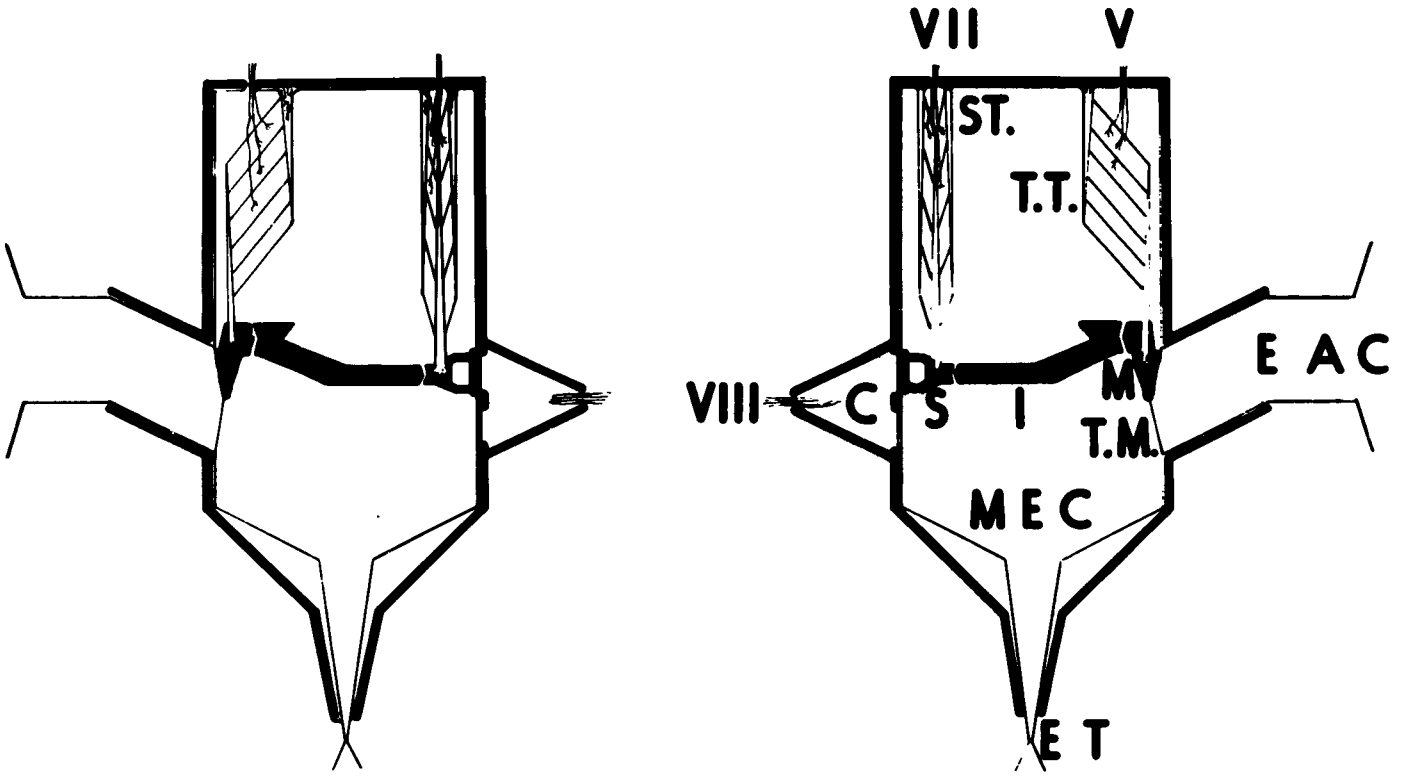


Fig. 1. Simplified diagram of auditory structures. EAC: external auditory canal; TM: tympanic membrane; MEC: middle ear cavity; M: malleus; I: incus; S: stapes; TT: tensor tympani muscle; ST: stapedius muscle; V: 5th cranial nerve; VII: 7th cranial nerve; VIII: 8th cranial nerve.

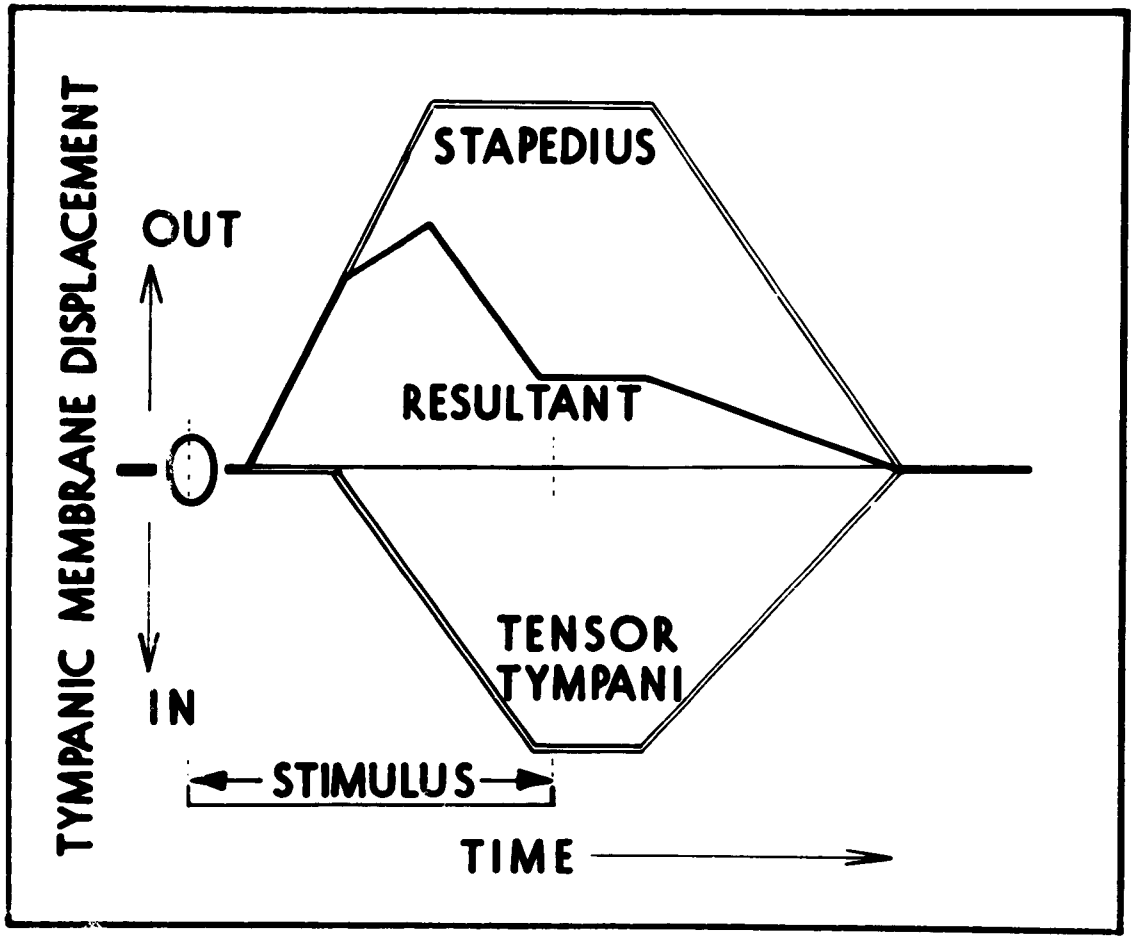


Fig. 2. Possible time and force functions during reflex contractions of the stapedius and tensor tympani muscles, and resultant displacements of the tympanic membrane.

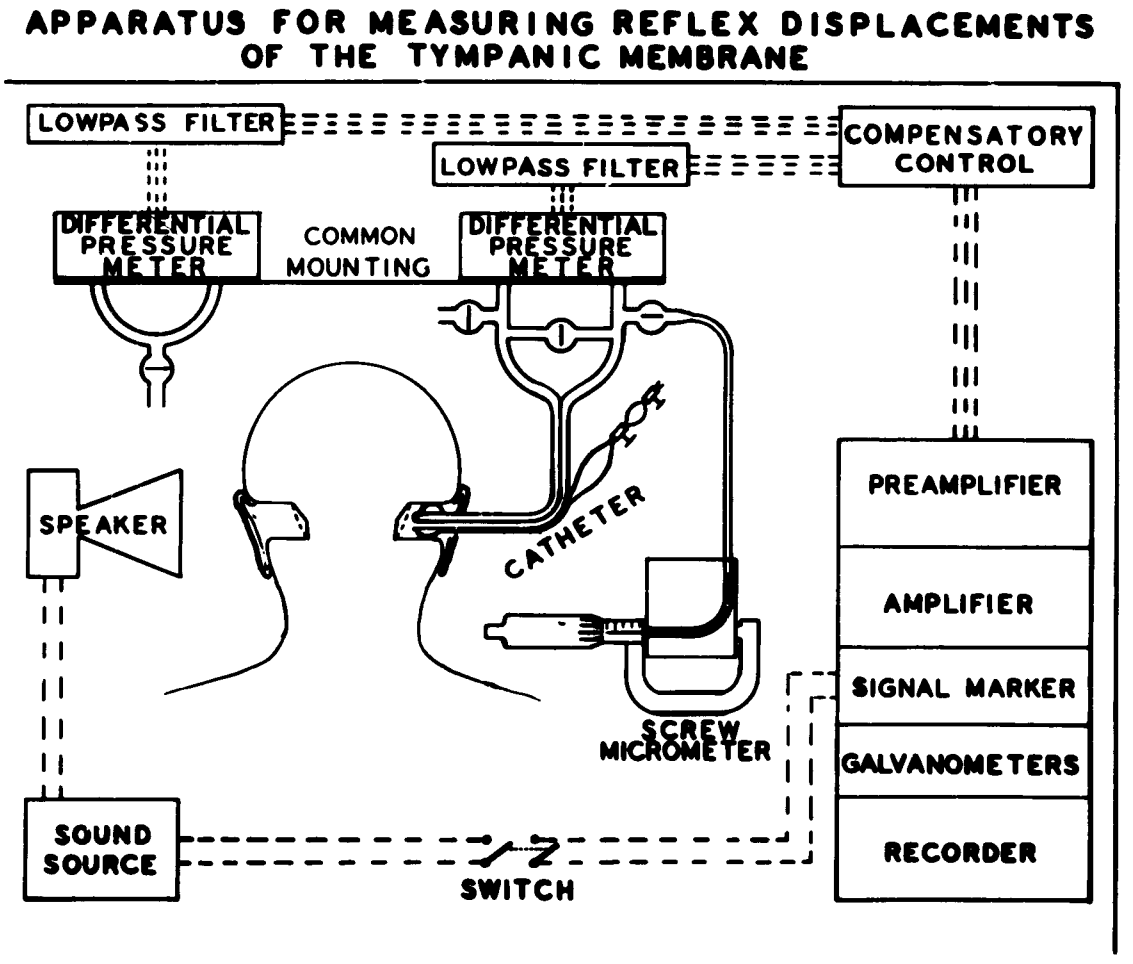


Fig. 3. Apparatus developed for cochleo-tympanic reflex testing.

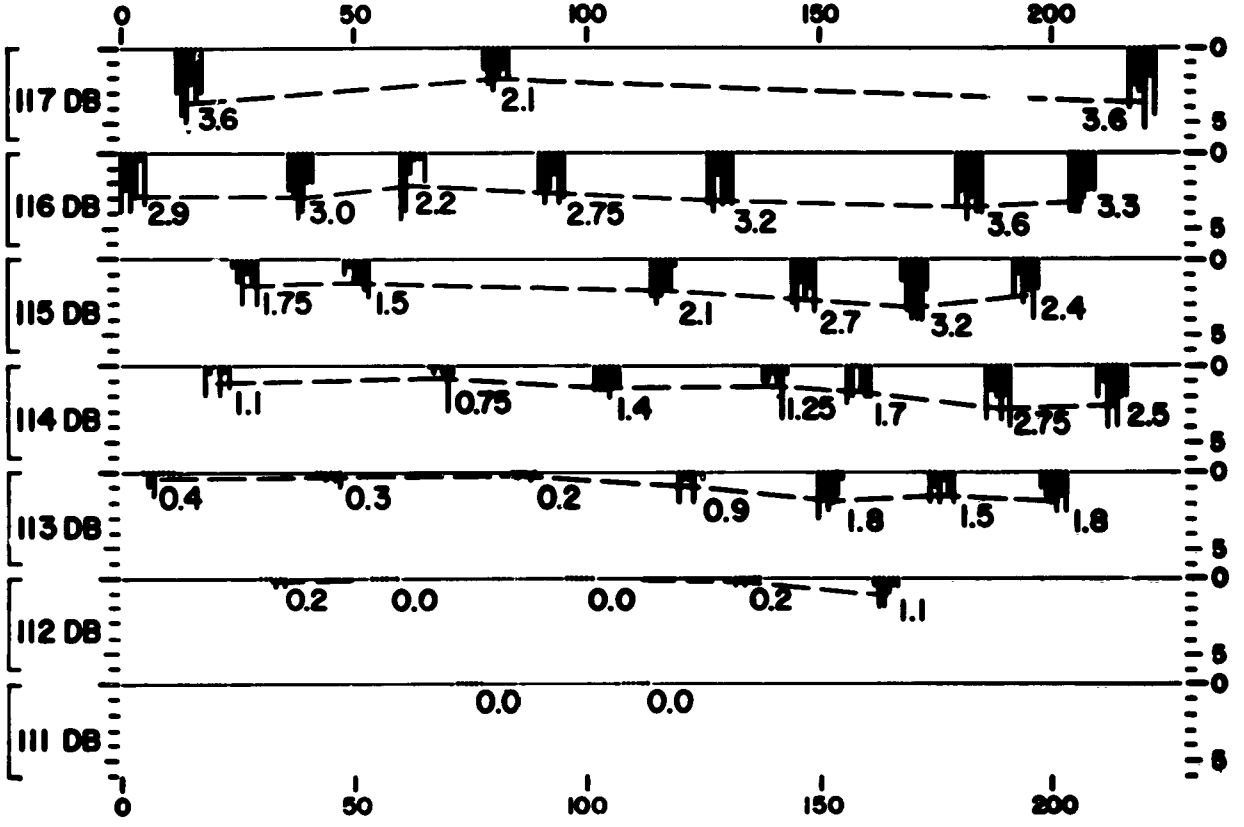


Fig. 4. Chronological sequence of stimulations and measured reflex displacements of tye tympanic membrane during 1 test session.

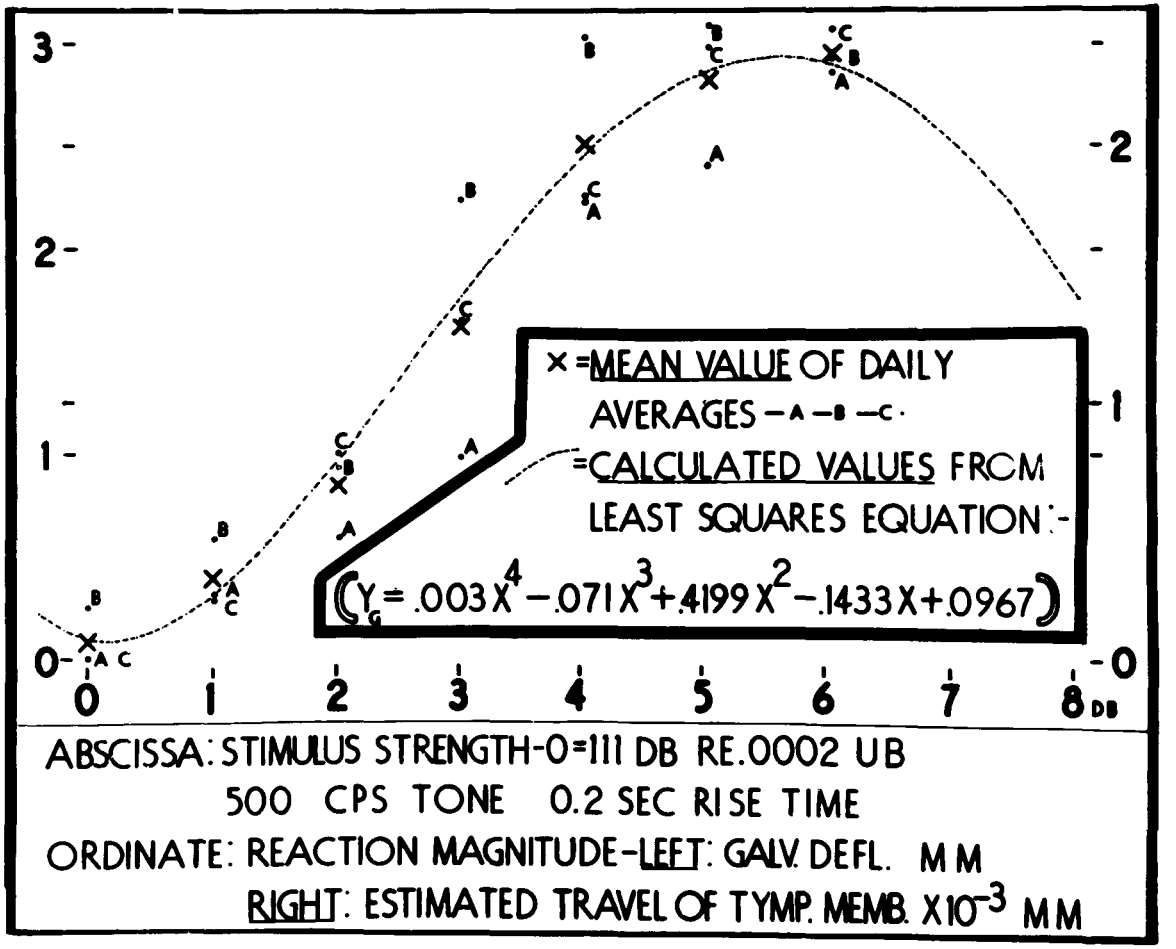


Fig. 5. Averaged stimulus - response relationship (one subject, 3 test sessions; 500 cps test tone; 225 msec rise time).

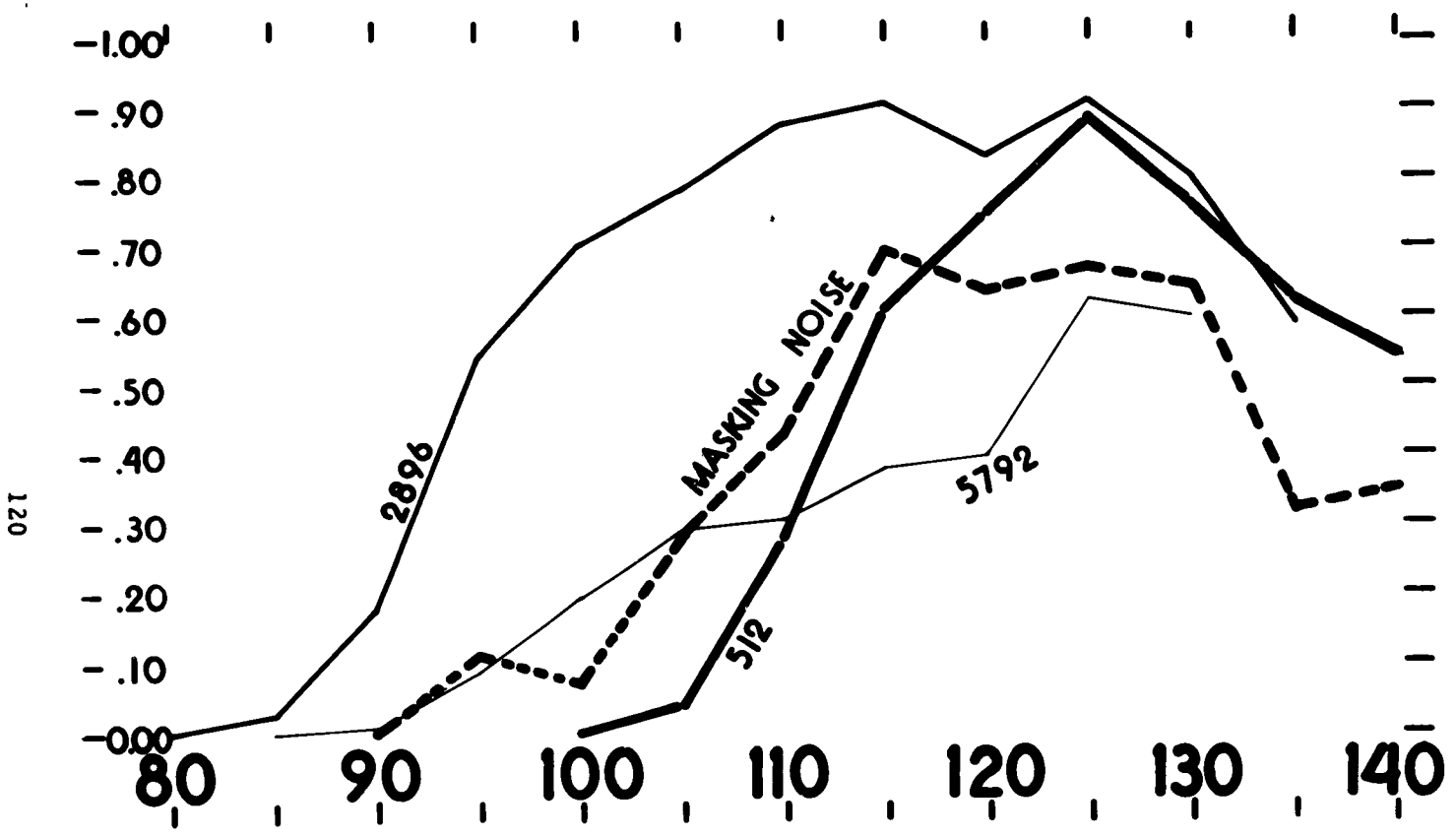


Fig. 6. Normalized, averaged reflex reactions in 9 preliminary tests on one subject (abscissa: SPL, db, 0.0002 microbar reference, for one of 3 single tones or audiometer masking noise).

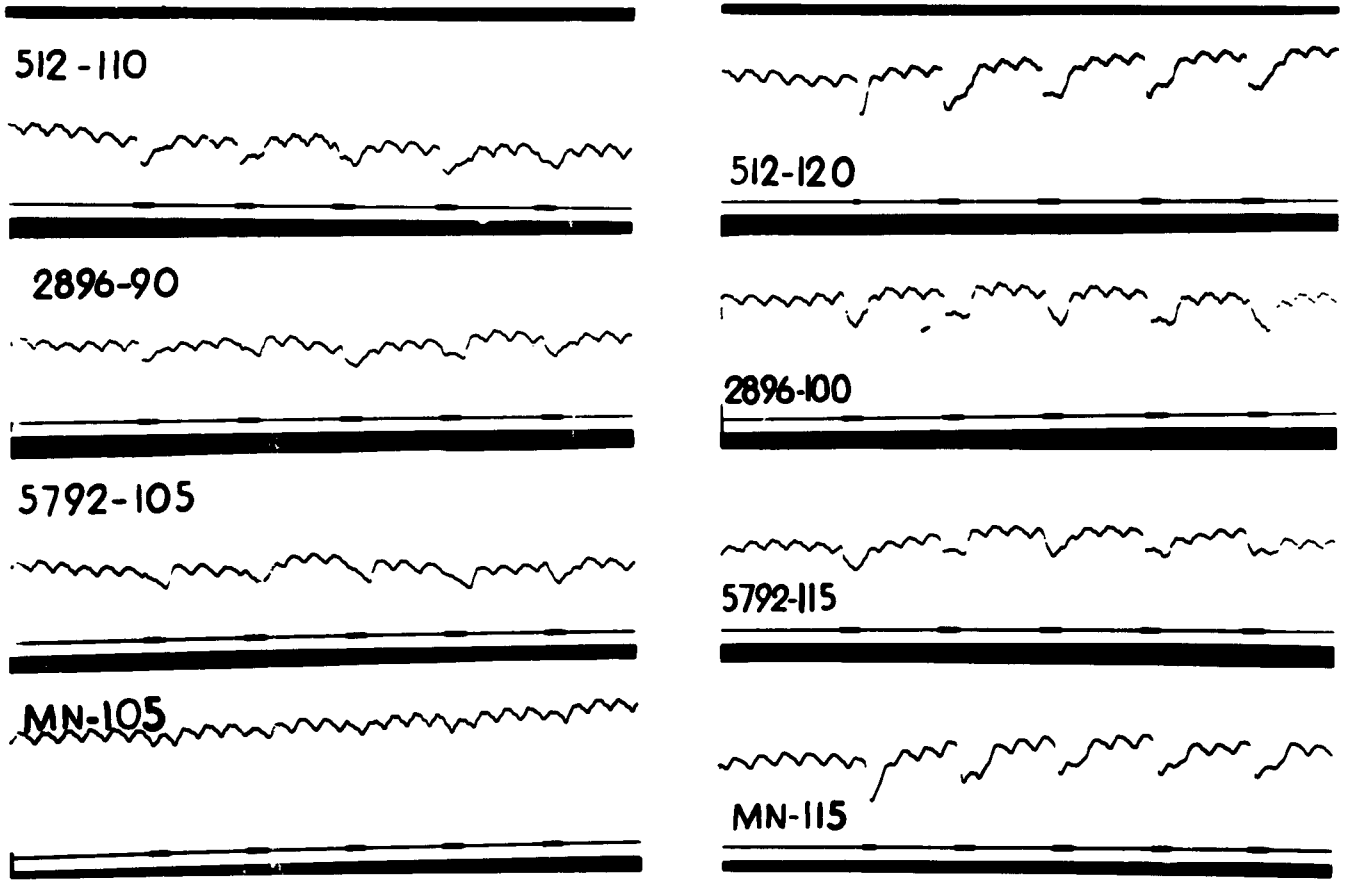


Fig. 7. Cochleo-tympanic reflex recordings. (Captions: stimulus frequency or masking noise; SPL, db. Lower tracing: stimulus marker; upper tracing: tympanomanometric registration from external auditory meatus, with superimposed cardiovascular pulsations.)

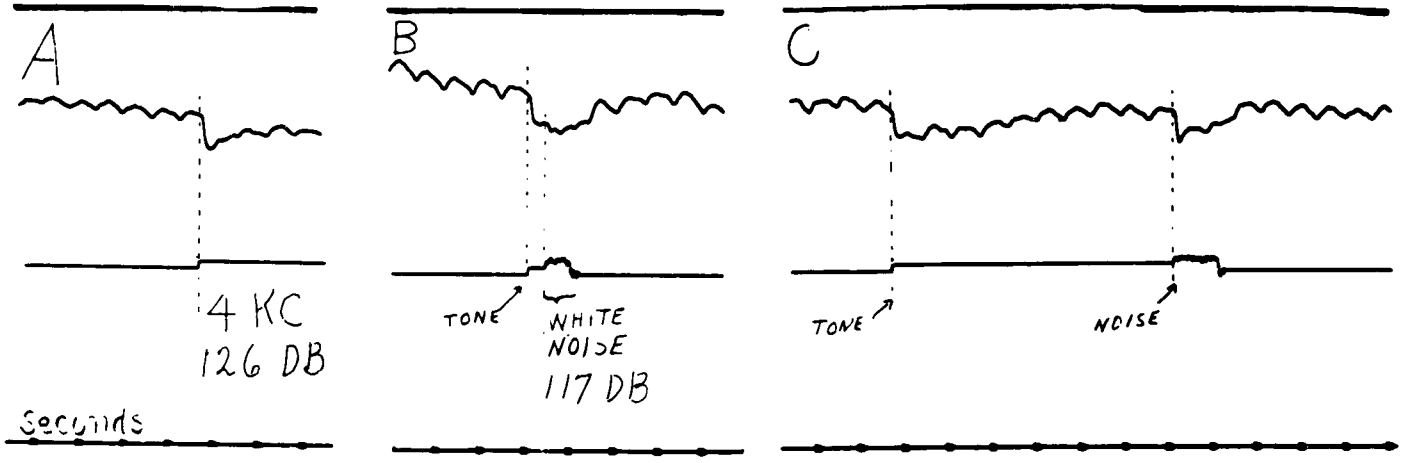


Fig. 8. Middle ear muscle relaxation recordings (lowest tracing: 1 sec time markings; middle tracing: stimulus marker; upper tracing: tympanomanometric registration).

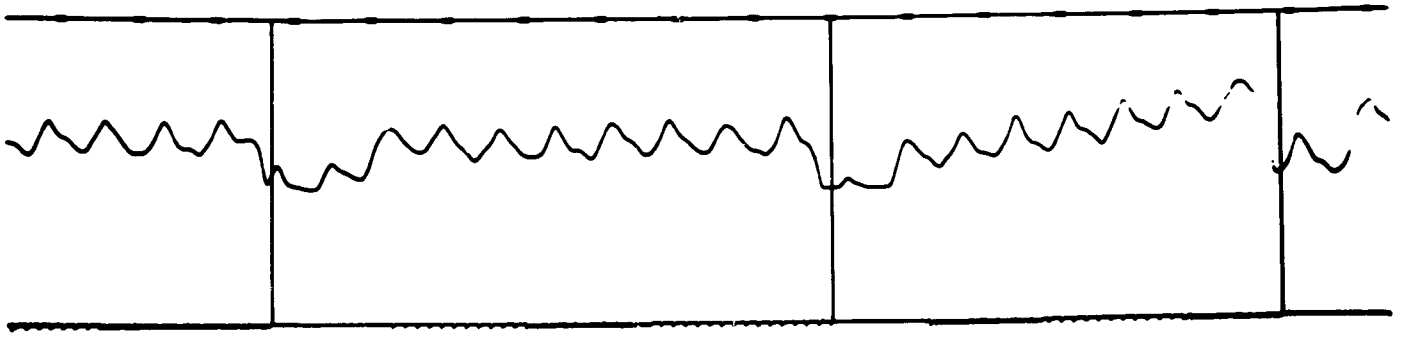


Fig. 9. Recordings of the effectiveness of alternating steady and intermittent bursts of noise. (Vertical lines: initial onsets of steady or intermittent white noise, rising to 120 db in 225 msec; critical cycling rate of intermittent noise at 0.45 duty cycle: 6-1/2 per second--with more rapid cycling, intermittent noise was less effective than steady noise; lowest tracing: 1 sec time markings; middle tracing: tympanomanometric registration; upper tracing: stimulus marker.)

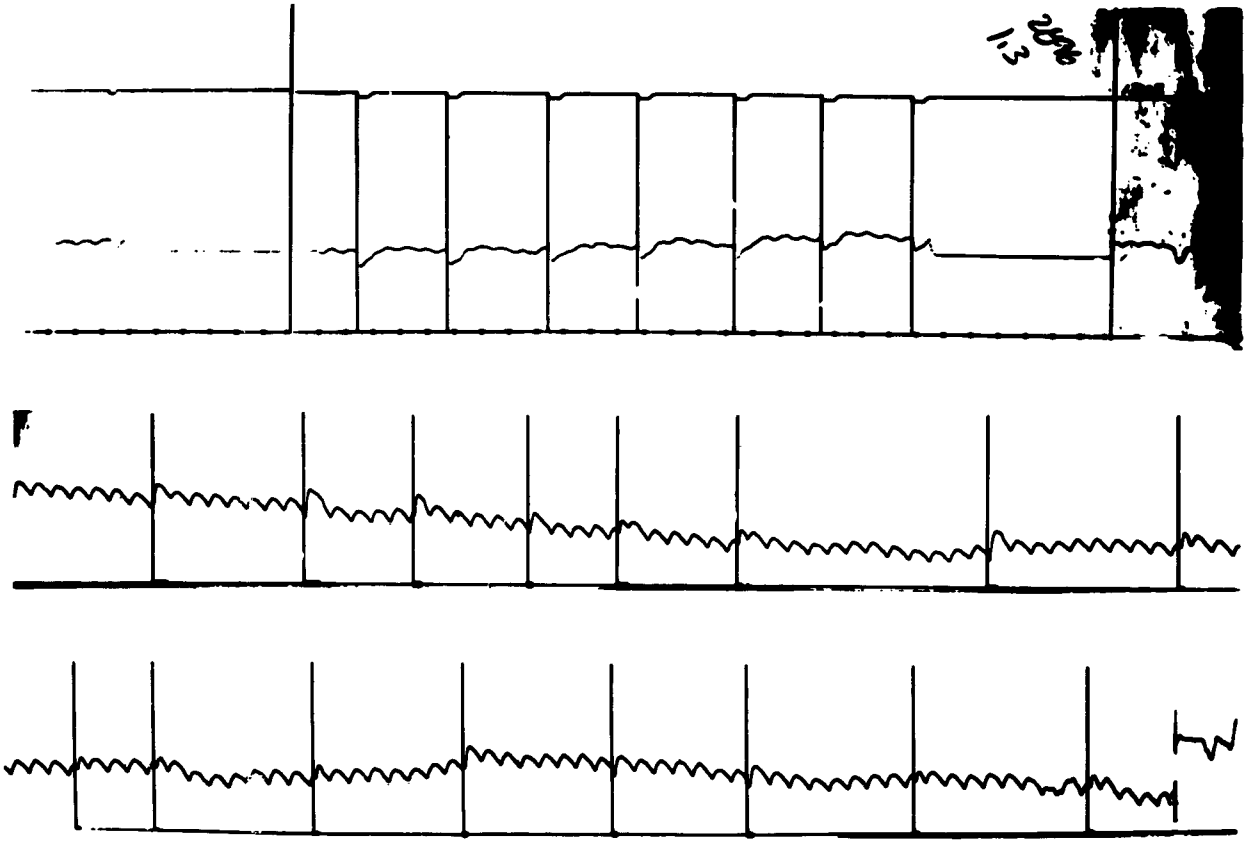
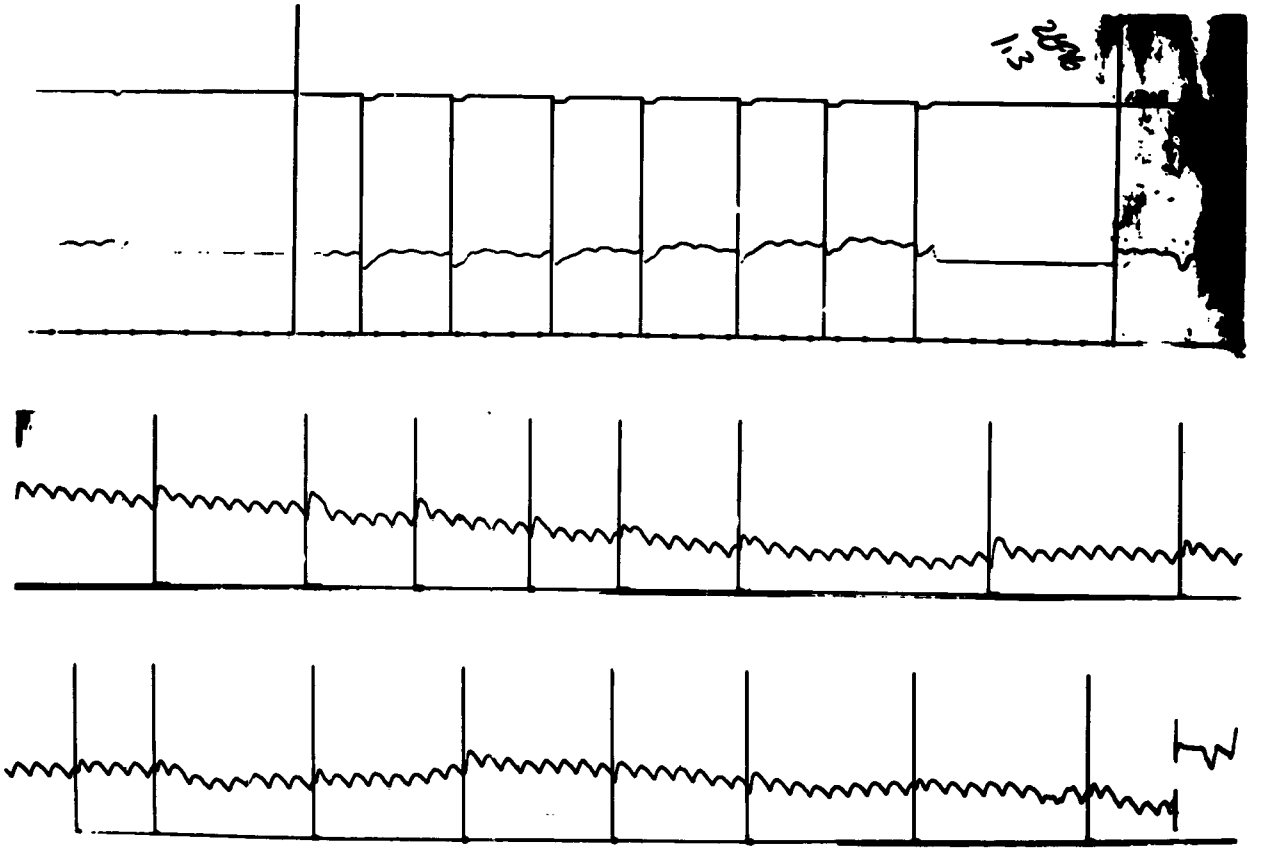


Fig. 10. Tests on Subject 22. (Upper record: 7/20/59, no parallax correction, catheter in left ear, 7 stimulations, 512 cps stimulus, 130-1/2 db; other records: 4/6/60, parallax correction .05 sec, catheter in right ear, 512 cps stimulus, 138 db, 8 stimulations; middle: 136 db, 8 stimulations; below: subsequent stimulations caused only downward reflex reactions.)



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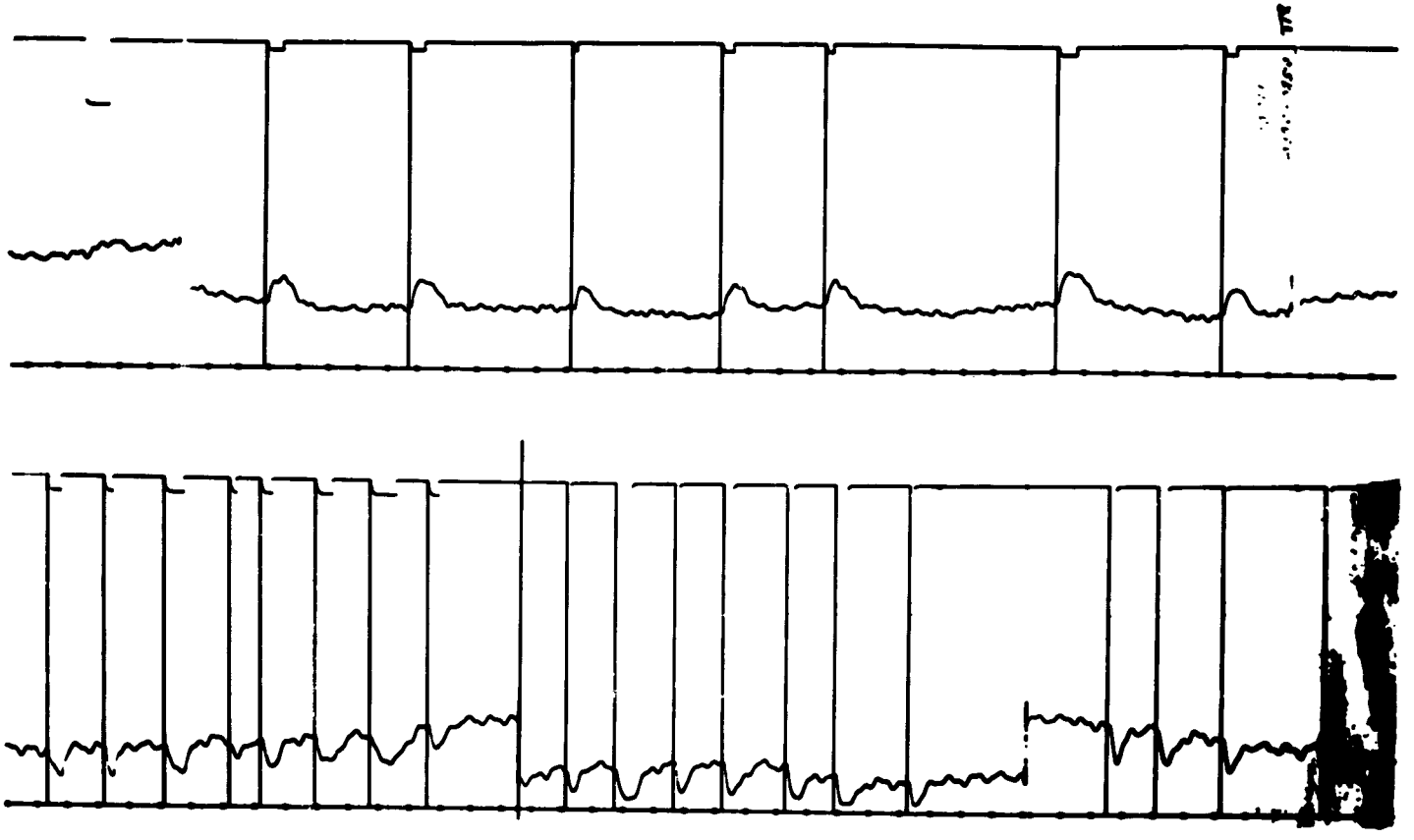


Fig. 11. Tests on Subject 69, 4/7/60. (Upper record: A. M., 7 stimulations, 0. 5 kc stimulus tone, 130 db; lower record: P. M., 20 stimulations, 2 kc stimulus tone, 110 db; subsequent reflex reactions resembled those in upper record.)

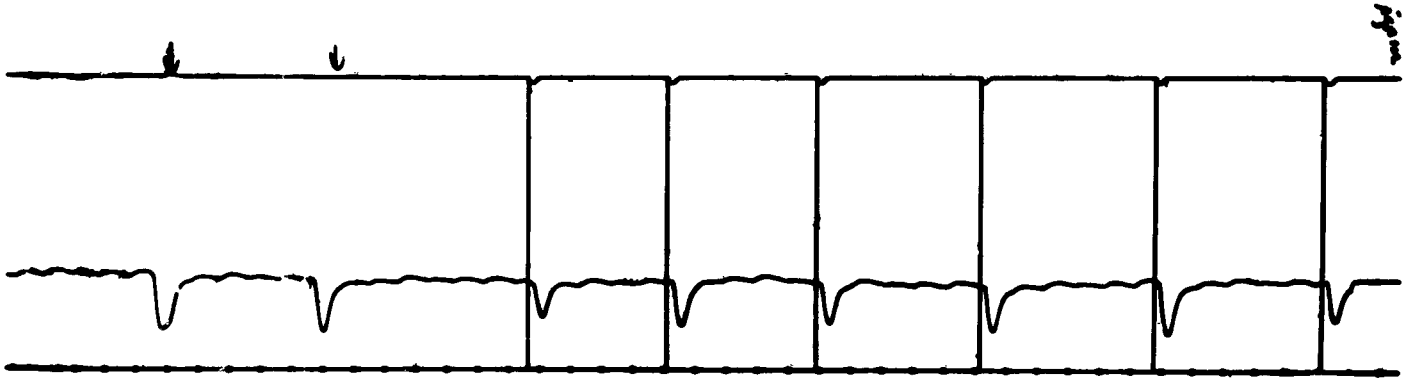


Fig. 12. Tests on Subject 61, 7/24/59. (Eight stimulations, 512 cps stimulus, 130 db--recording sensitivity had to be reduced by a factor of 4 below the usual value in order to keep the reflex reaction within scale.)

Time After Cessation of Fatiguing Stimulus	Residual Depression of Auditory Acuity	Residual Depression of Reflex Excitability
50 seconds	40 db	>>10 db
110 seconds	35-40 db	>10 db
230 seconds	35-40 db	<10 db
310 seconds	35 db	<5 db

Fig. 13. Recovery rates tested at 1500 cps, after fatigue with a 1000 cps tone for 1/2 minute at 140 db.

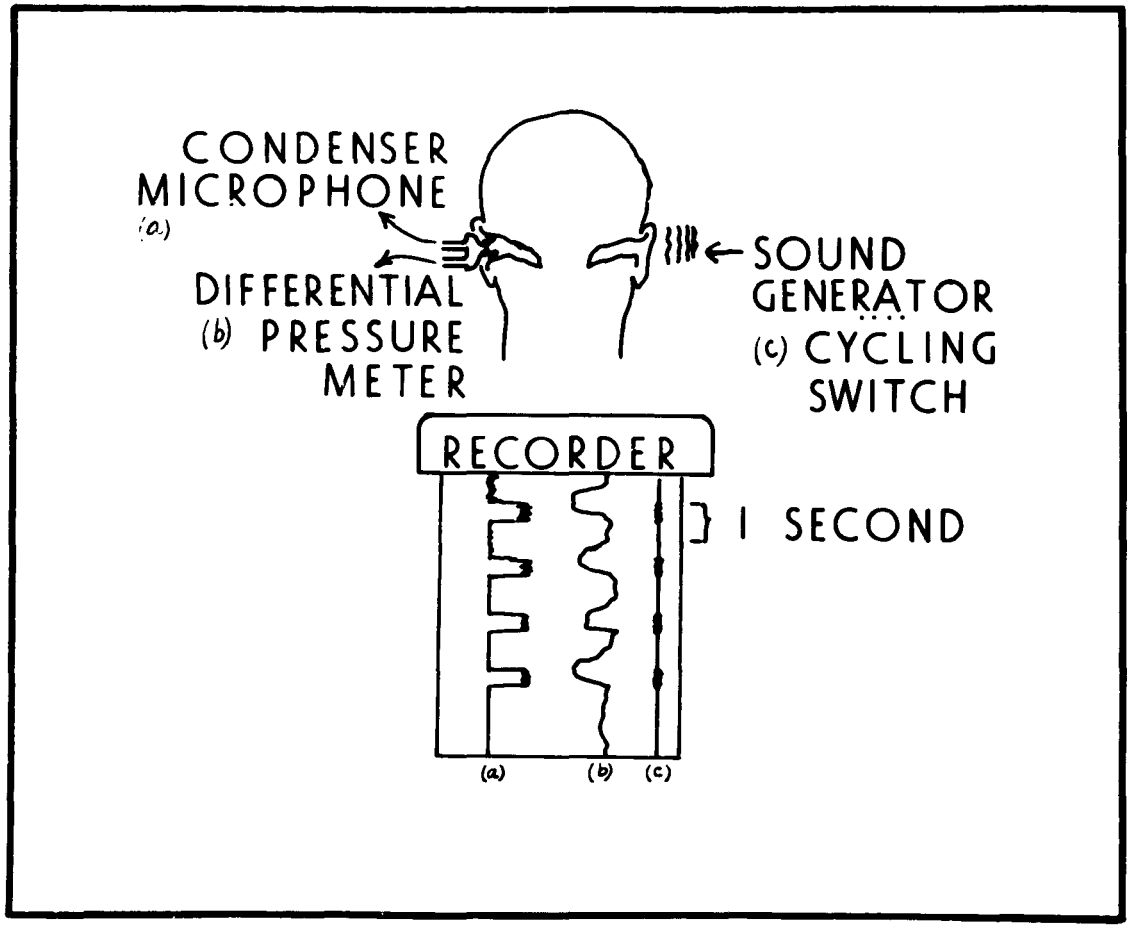


Fig. 14. Apparatus used for simultaneous recording of tympanic membrane displacement and sonic vibration, resulting from contralateral acoustic stimuli.

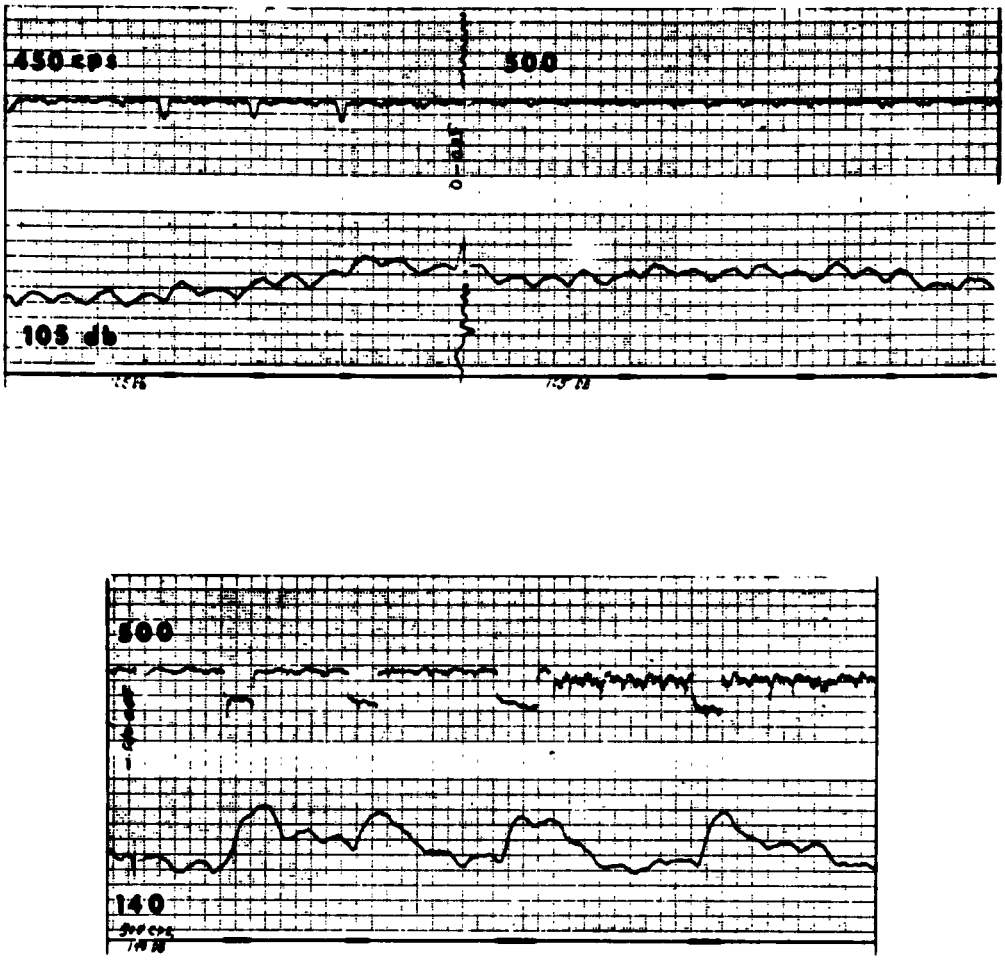


Fig. 15. Comparison of sonic vibrations (upper tracings, reflex displacement of tympanic membrane (middle tracings), and timing of contralateral stimuli (lowest tracings). (Stimuli; 450 cps, 105 db; 500 cps, 105 db; 500 cps, 140 db.)

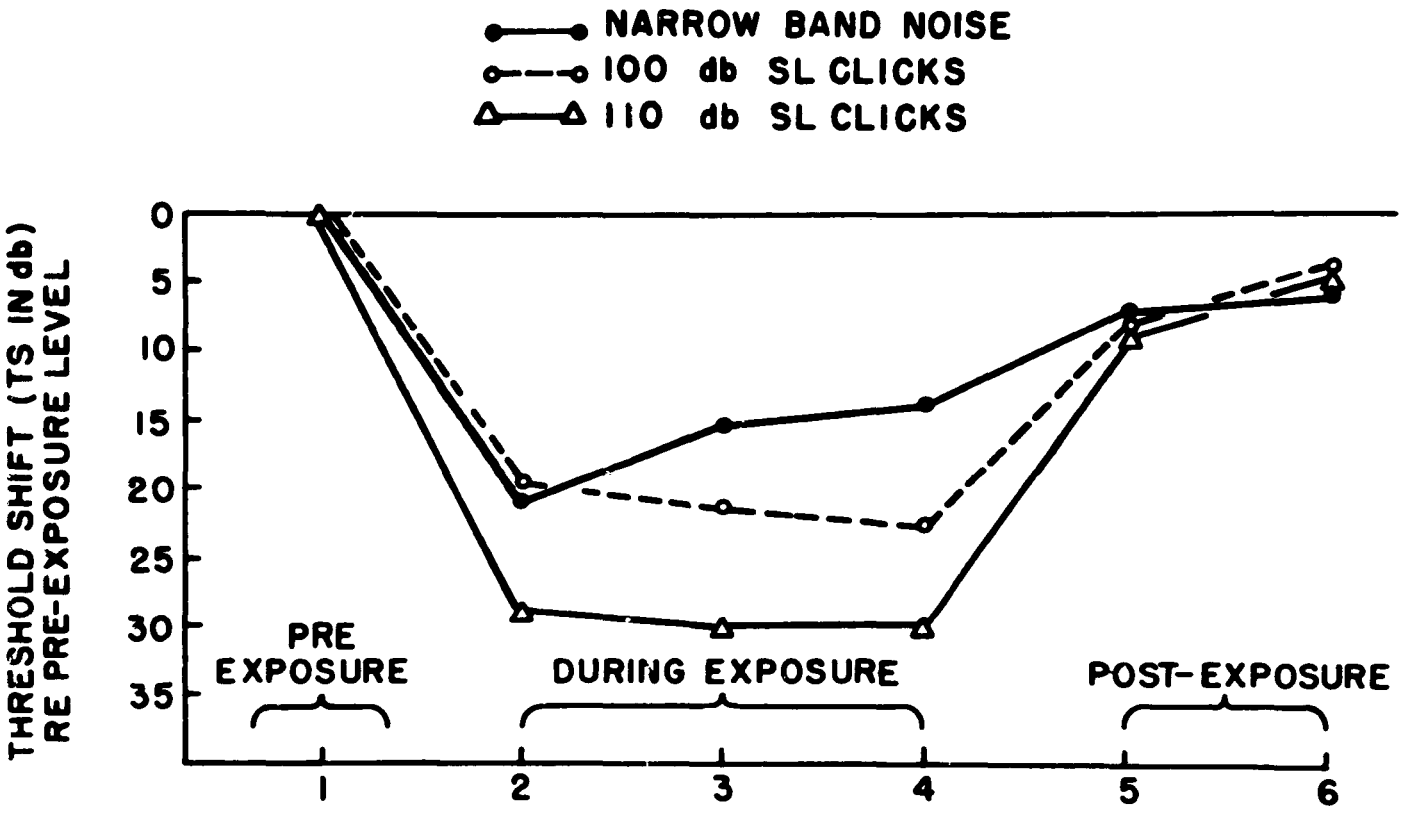


Fig. 16. Threshold shift during contralateral stimulation by click and noise.

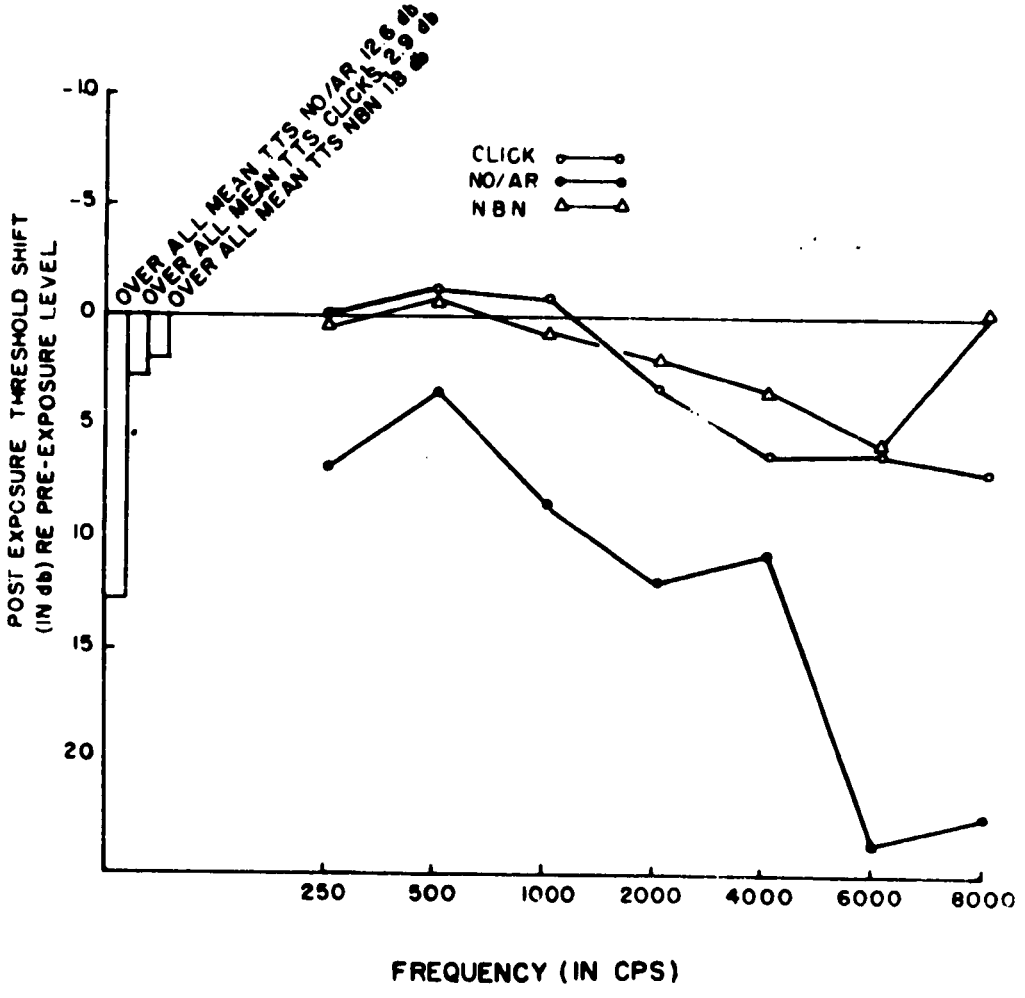
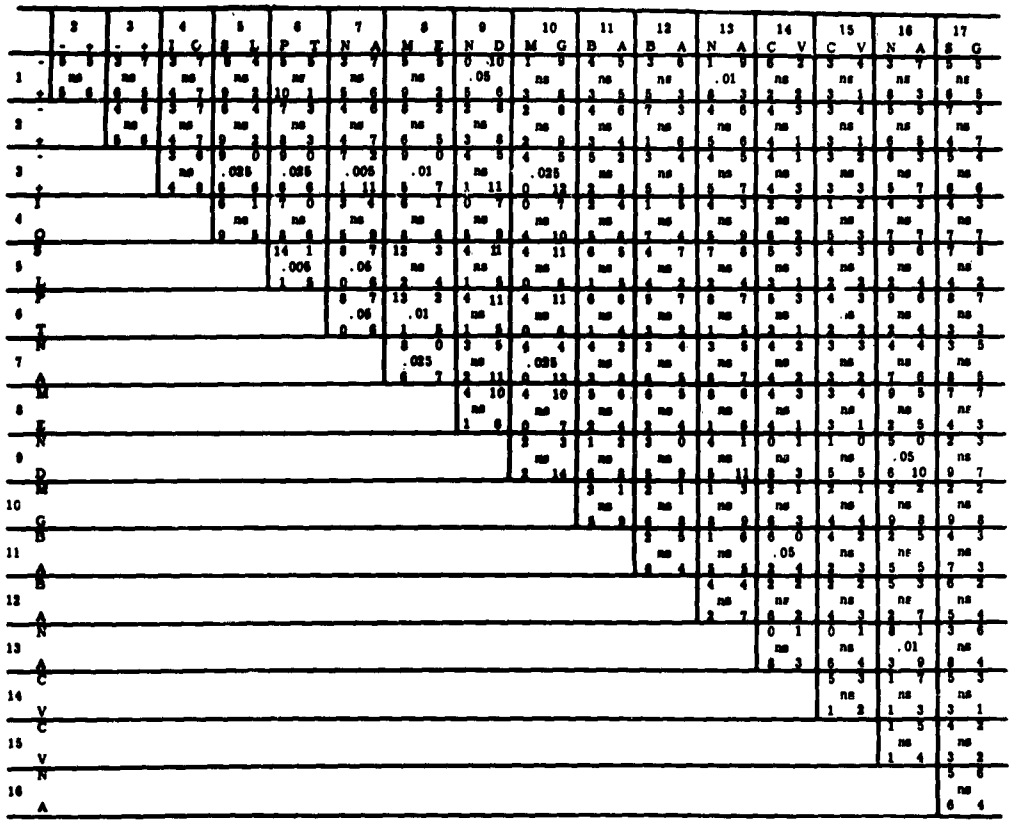


Fig. 17. TTS following firing without AR and with click and NBN AR stimuli.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Sub.	Seq.	Age	ReTh	Dir.	Mag.	Per.	Dim.[a]	Dim.[b]	A.Ac.	N.E.	TS[a]	TS[b]	TTS[a]	TTS[b]	TTS[c]	TTS[d]	M.I.
kjg	17	25	100	O	S	P	N	M	N	G	—	—	N	—	—	N	G
hs	11	23	103	O	S	P	N	M	N	M	A	B	A	V	C	N	S
go	2	20	105	I	S	P	N	M	D	G	B	A	A	C	V	N	S
m	20	20	108-	O	S	P	A	M	N	G	B	B	N	—	—	N	S
gb	16	25	108-	I	S	P	A	M	D	G	A	A	N	—	—	N	S
jh	13	23	110	O	S	P	N	M	D	M	B	B	A	C	C	A	S
esm	1	52	110	I	S	P	N	M	D	G	B	A	A	C	C	A	G
aj	3	27	110	O	S	P	N	M	D	M	B	A	A	C	V	A	G
ml	18	34	110	O	S	P	N	M	N	M	—	—	N	—	—	N	G
la	14	20	113	I	L	P	A	D		G	A	B	N	—	—	N	S
c	9	20	113	O	S	P	A	M		D	G	A	B	N	—	N	G
whb	7	21	113	O	L	T	A	M		D	G	A	B	A	V	A	S
rec	4	23	113	O	L	T	A	E		D	G	B	A	A	C	A	G
gh	21	35	115	I	S	P	A	M		D	G	—	—	N	—	N	G
ks	12	23	118-	I	S	P	N	M		D	G	A	A	N	V	A	G
jlf	19	35	118-	O	S	P	A	E		D	G	B	A	A	C	A	S
sp	15	24	118-	O	L	T	A	E	N		G	A	—	N	—	N	G
jc	6	24	127-	I	S	P	A	E		D	G	A	A	A	V	A	S
sm	5	27	127-	O	S	T	A	E		D	G	—	—	A	—	N	G
dh	8	19	131-	O	L	T	A	E		D	G	A	B	A	C	A	S
lc	10	39	131-	O	L	T	A	E		D	G	A	A	A	C	A	S

Coding of entries:
1-order in C-T test sequence (1 to 10, or 11 to 21)
2-Subject's age (19 to 23 years or 24 to 52 years)
3-Initial reflex threshold at 512 cps (100 to 110 db, or 113 to 131 db)
4-Predominant direction of Tympanic Membrane movement (Inward or Outward)
5-Relative magnitude of recorded drumhead displacement (Small, or Large)
6-Relative persistence of recorded drumhead displacement (Persistent, or Transient)
7-Diminution of C-T reflex reactivity (None, or Appreciable)
8-Diminution of C-T reflex reactivity (Minor, or Extreme)
9-Audiometric acuity (Normal, or Decreased by more than 10 db)
10-Subject's self-estimate of chronic noise exposure to noise (Moderate, or Great)
11-Maximum contralateral TS at 500 cps (Below 30 bd, or Above 25 db) - TS^a
12-Minimum contralateral TS at 500 cps (Below 20 db, or Above 15 db) - TS^b
13-Unprotected TTS at any test frequency (None, or Appreciable) - TTS^a
14-TTS Protection afforded by narrow band noise (Consistent, or Variable) - TTS^b
15-TTS Protection afforded by clicks (Consistent, or Variable) - TTS^c
16-Unprotected TTS at 8000 cps (None, or Appreciable) - TTS^d
17-Subject's self-estimate of degree of musical interests (Slight, or Great)

Fig. 18. Summary of individual observations on 21 subjects at AMRL, classified into 17 two-valued variables.



Coding follows order of Figure 18.

Indicator in center of block represents likelihood that frequencies in corners of blocks were chance occurrence (ns = no significance).

Fig. 19. Shared frequency distributions of each pair of classified variables for all subjects.

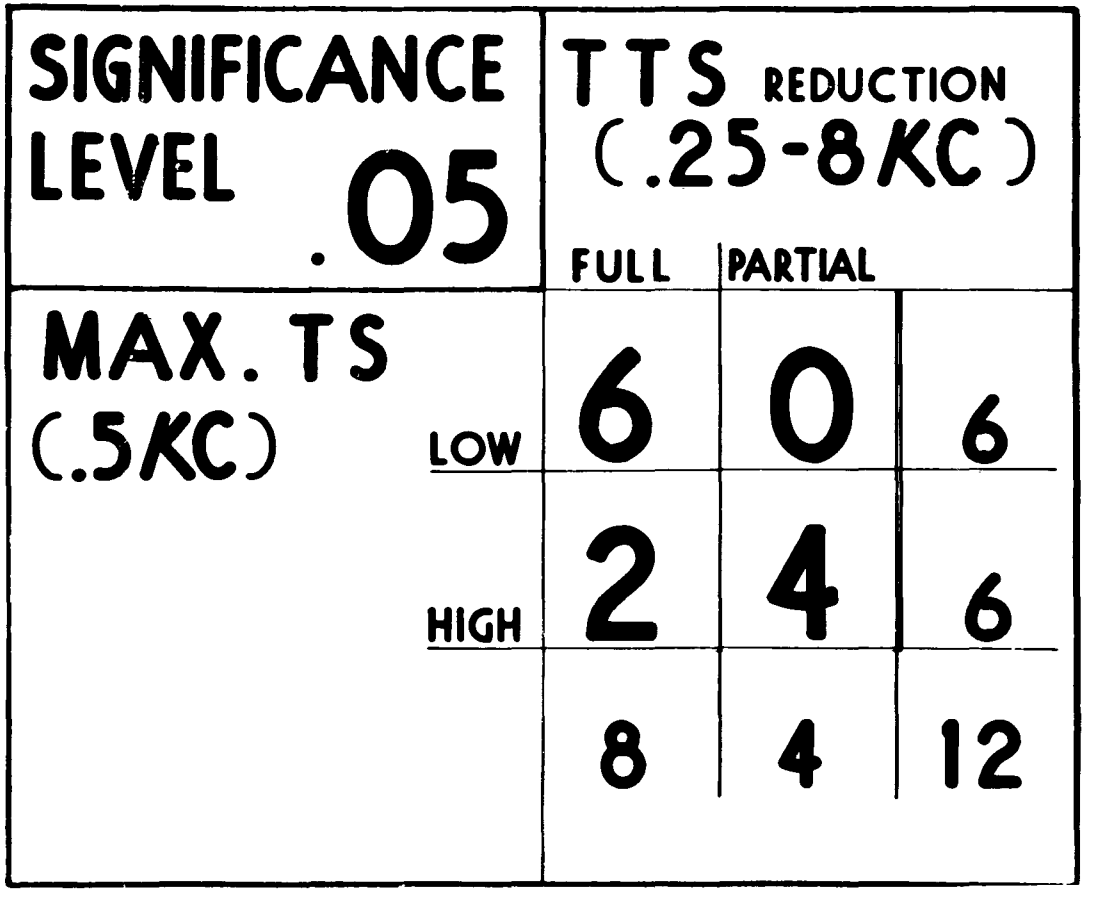


Fig. 20. Paired distributions showing correlation between 500 cps TS resulting from contralateral reflex activating stimuli, and reduction of gunfire - noise - induced TTS resulting from prior (150 msec) reflex activating stimuli.

SIGNIFICANCE LEVEL .05	AUDIOMETRIC ACUITY NORMAL	DEFECTIVE	
MANOM. TEST. SEQUENCE 1ST	0	10	10
2ND	5	6	11
	5	16	21

SIGNIFICANCE LEVEL .01	UNPROTECTED TTS (.25-8 KC) NONE	SOME	
MANOM. TEST. SEQUENCE 1ST	1	9	10
2ND	8	3	11
	9	12	21

SIGNIFICANCE LEVEL .05	UNPROTECTED TTS (8 KC) NONE	SOME	
AUDIOMETRIC ACUITY NORMAL	5	0	5
DEFECTIVE	6	10	16
	11	10	21

Fig. 21. Paired distributions relating (1) measures of unprotected temporary threshold shifts caused by gunfire noise, (2) audiometric acuity at any of 7 testing frequencies, and (3) sequential order in which subjects were tested manometrically.

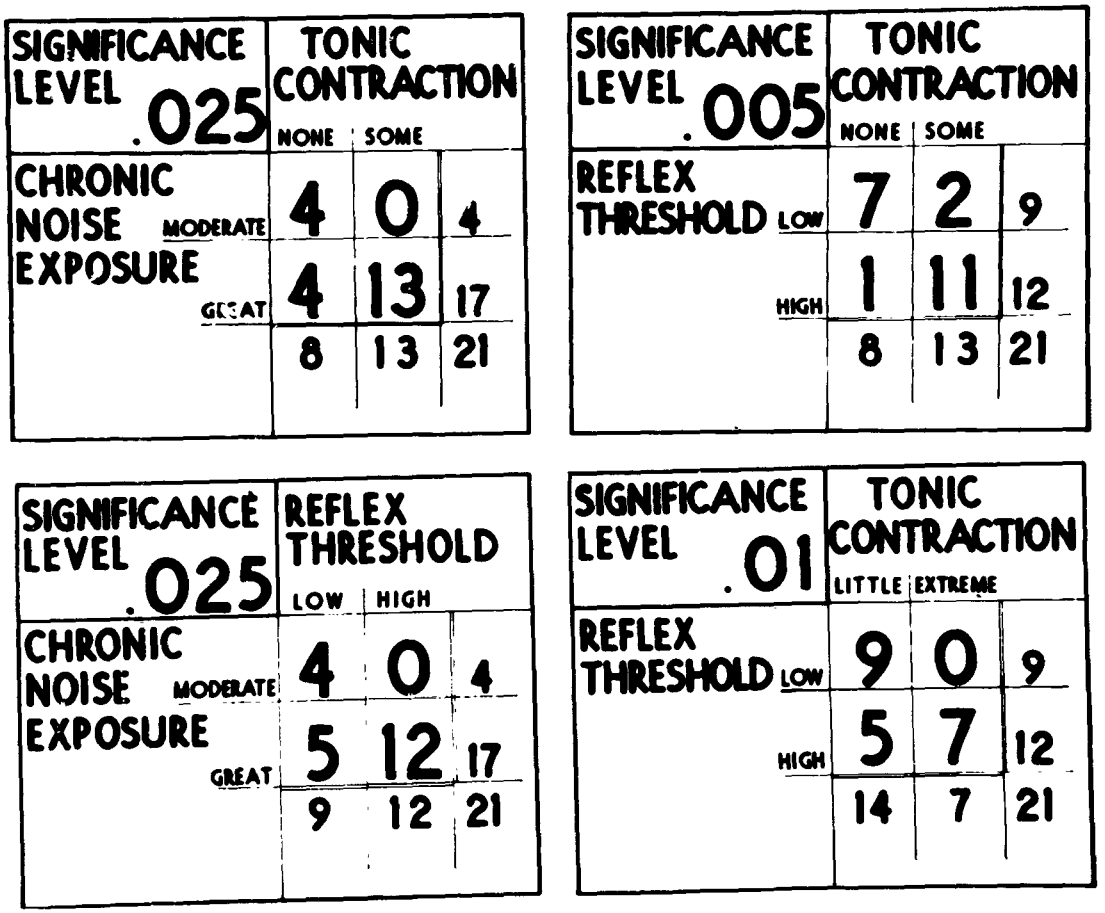


Fig. 22. Paired distributions among (1) subjects' self-estimates of severity of chronic noise exposure, (2) manometric reflex thresholds for 512 cps stimulus, and (3) adaptation tendencies suggesting tonic contractions of the middle ear muscles.

THE NORMAL HUMAN INTRA-AURAL MUSCLE REFLEX IN RESPONSE TO SOUND

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INTRODUCTION

In recent years, information processing in the nervous system in general and in the auditory system in particular has pre-empted the interest of our laboratory. The understanding of the function of the middle ear, interposed as it is between the source of an acoustic signal and the cochlea, is of vital importance when one attempts to define the actual input to the cochlea. This report will deal with a series of investigations on the activity of the middle ear muscles in response to sound. An attempt was made to clearly define the relative roles of the stapedius and tensor tympany muscles in the intra-aural reflex and to relate their participation to the parameters of the acoustic signal. The particular modifications of the method of tympanomanometry used are described, stress being placed on the simplicity of instrumentation and the high degree of sensitivity attained. It is to be noted that the first portion of the report deals with the results obtained using a tympanomanometric system with AC response characteristics thus limiting the information to the description of the threshold properties of the reflex as a whole. The second phase describes the results obtained with an improved DC electronics system and deals with the time-amplitude history of the intra-aural muscle reflex.

METHOD

Part I. As a technique for measuring the reflex in a normal human subject we have used the method of tympanomanometry. It is to be noted that this method takes advantage of the consensuality of this reflex permitting acoustic stimulation of one ear and reflex measurement on the contralateral ear. The ear in which the reflex was to be

measured was hermetically sealed with a molded, soft plastic insertion-type ear plug, V51R. For the test the plug was pierced through the center by a stainless steel tube with a collar at one end. A second collar was slipped over the steel tube and firmly held the plug, forming a tight seal (Fig. 1, Insert). The fully assembled unit is light in weight and easily held in place. As a pressure transducer for measuring the pressure changes in the sealed external canal, we used a capacity-type microphone, Altec 21C, fitted with a probe tube and connected to the tube of the plug unit by a length of polyethylene tubing (Fig. 1). The frequency response of the system was down 6 db at 250 cps and at 1/3 cps, e.g., an AC coupled system. Tests were conducted in an anechoic chamber. An Altec-Lansing speaker was used to deliver undistorted free-field signals up to about 115 db ref. 0.0002 dynes/cm^2 to each subject. A second microphone (W-E640AA) at the subject's unplugged ear monitored the acoustic signal and the output of both microphones was displayed on a split beam oscilloscope. For the purpose of permanent records the face of the oscilloscope was photographed. The sensitivity of the system is limited only by the electronic noise level of the Altec microphone and complement, in this case about 60 db ref. 0.0002 dynes/cm^2. Pressure changes measured at this limit of sensitivity are calculated to be in the region of 1.5×10^{-4} mm of Hg. If this value is translated into terms of displacement of the tympanic membrane, it is found to be equivalent to about 1×10^{-6} cm. Figure 2 depicts the time-amplitude envelope of the signals used. Time (in ms) is given in terms of rise time, duration of constant amplitude phase and decay time (e.g., 10-80-10). These values are confirmed from the output of the signal-monitor microphone (640AA) at the subject's ear. The pure tones were 400, 800, 1600, 3200, and 6400 cps. In addition, the signal, 50-0-50, was also presented as banded noise with center frequencies as above and band widths approximating those given by Fletcher (1953) as critical bands. Figure 3 is an example of our method of recording data. In the absence of sound stimuli the regular pertubations in the pressure trace are coincident with the subject's heart beat. The origin of this pressure pulse is unknown. With the onset of sound stimuli the pressure fluctutations coincident with the reflex response are seen superimposed on the pulse trace. The system is set up so that a downward deflection is inscribed by an increase in pressure. This is a typical biphasic trace recorded with this AC system.

We were, of course, aware that a severe limitation on interpretation of the time history of the reflex response was imposed by the lack of DC capabilities of the system. The questions which we might hope to answer had to be tailored with this limitation in mind. The

first experiments utilizing this system were designed to answer the following questions: 1) In what portion of normal young adults can the onset of an intra-aural muscle reflex in response to sound be demonstrated? 2) Within the limits of the sensitivity of the system, what are the threshold values for the reflex? 3) In what way is this reflex threshold affected by the subject's threshold of detection and the parameters of the signal?

Tests were conducted on a group of university male students screened at the outset to include only those with normal audiograms and no evidence of middle ear disease. A panel of ten was finally chosen consisting of only those students whose reflex could be elicited by the majority of the signals to be used (Fig. 2) at intensity levels below the 115 db limitation of our system. The signals were presented to each subject monaurally at a random rate, not exceeding 2/sec. In addition, each subject was required to make a sensation threshold decision on the basis of not more than 24 repetitions of the signal.

Part II. In order to consider problems concerned with the true time-history of the reflex, we changed from the Altec microphone (pressure transducer) to a Western Electric 640AA and changed to DC/FM electronics so that we could now record the true time history of the pressure changes in the external canal. Applied DC pressure loads, negative or positive (tested up to 20 mm of alcohol) could be maintained for periods up to 15 minutes or longer. As a recording device, we changed from the oscilloscope to a Computer of Average Transients (CAT - Mnemotron Corporation). With this instrument, we could now record the average response to multiple stimuli and thus come closer to an accurate picture of the reflex.

Figure 4 demonstrates the typical format of what we shall refer to as a CAT recording. Each figure is labeled as to the total time during which the CAT records information, i.e., analysis time, and the series of 20 dots on each trace gives time intervals in terms of the total analysis time. In the first trace, the analysis time is 32 seconds and therefore the time between dots is 1.6 seconds. The system is set up so that a positive pressure inscribes an upward deflection. The first trace represents a single sweep of the CAT, during which time a positive pressure was applied in the system. It is at once obvious that the DC pressure change is maintained for at least 16 seconds. Such a DC check was made before and after each test period. The other two figures represent a negative and a positive pressure response to similar signals in different subjects. These are typical responses in their general shape but are atypical in the presence of rather

large and regular oscillations, the first at a rate of about 3.5/sec and the second at 8.5/sec.

RESULTS

Part I. Only about 5% of the original group of 30 subjects tested did not respond to the signals presented with a measurable acoustic reflex. Of the 95% remaining, about 75% did respond to the majority of the signals at levels well below the 115 db limitation of the system. Ten of the latter subjects formed the panel used in the experiment.

It was clearly demonstrated that each subject had a characteristic reflex threshold. Figure 5 demonstrates the reflex and detection (sensation) level thresholds for the single signal, 50-0-50, individually plotted for each member of the panel. The reflex thresholds were repeatable within 1 db during the same period. However, from week to week, variations as much as 5 db were recorded. Figure 6 (A and C) depicts the average response of a panel of ten subjects to the battery of signals. The signals have been grouped according to increasing acoustic energy.* Group 1, the least power, is composed of 2-20-2, 10-20-10, and 50-0-50; Group 2 is composed of 10-40-10, and 10-80-10. Group 3, the greatest power, is composed of 25-500-25 and 25-1000-25. When classified in this way, it was demonstrated that threshold values for members of the group were not significantly different. The values at detection level, for the 500 and 1000 ms signals are identical and agree with the accepted free-field values for just-detectable monaural thresholds. The shapes of the two sets of curves, reflex

*Acoustic Energy per Unit Area $\left(\frac{\text{Watt sec}}{\text{cm}^2}\right) = \frac{(P)^2 t_1 \times 10^{-8}}{6.23} + \frac{(P)^2 t_2 \times 10^{-8}}{4.15}$

P = RMS amplitude of plateau in dynes/cm^2
t_1 = rise or decay time, seconds
t_2 = plateau duration, seconds

GROUP	I	II	III
	2-20-2 $= .20 \times 10^{-4}$	10-40-10 $= .15 \times 10^{-4}$	25-500-25 $= 5.0 \times 10^{-4}$
	10-20-10 $= .26 \times 10^{-4}$	10-80-10 $= .83 \times 10^{-4}$	25-1000-25 $= 9.8 \times 10^{-4}$
	50-0-50 $= .32 \times 10^{-4}$		

and detection, are remarkably similar both as a function of signal frequency and signal envelope. Within the range tested, the higher the detection threshold, the higher the reflex threshold. Most importantly, there appears to be a relation between the total length of the signal and its intensity with regard to its ability to reach both detection and reflex thresholds. That is, the longer the signal remains on, the less amplitude it need develop to reach threshold. There apparently is a limit to this phenomenon, since prolongation of the signal beyond 500 ms does not lower either threshold. When the same information is plotted on one graph (Fig. 6B) as the difference between reflex and detection thresholds, it is to be noted that the curves approximate a horizontal line. The average reflex threshold is about 92 db ± 4 db sensation level. That is, regardless of the frequency or the shape of the signal, the reflex threshold is about 92 db above the specific detection threshold.

Before going on, it is necessary to mention a few points. First, to bring attention to the 5% of subjects with apparently normal ears, whose reflex could not be demonstrated at stimulus levels up to 115 db. Secondly, to mention the ancillary findings that, in well-trained subjects, the contraction of the intra-aural muscles in response to sound stimuli can be heard by the subject himself. If the subject is permitted to control the intensity of the signal and is asked to determine his own reflex threshold using the disappearance of the sound of the muscle contraction as an end point, it is consistently found that this value is within 1 db of the threshold determined by visually monitoring on the scope the pressure changes in the same ear. And lastly, to call attention to the fact that the pressure change in the external canal in response to sound has been found to vary from subject to subject. In some, the change is a negative pressure and in others it is a positive pressure.

Part II. Using the modified system which provided DC recording capabilities, the first thing checked was the relative sensitivity of the AC system and the validity of the threshold information obtained up to this point. By averaging the response to 100 stimuli (indicated on each trace as x 100) we could now improve signal to noise ratio by 10 to 1 or about a 20 db increase in sensitivity. Using this method, the reflex thresholds were lowered less than 3 db, thus lending confirmation of the threshold values presented so far. The remainder of the report will concentrate on the time course of events of the reflex as a function of the particular acoustic signal.

Figure 7 demonstrates, in two subjects, the effect, on the time course of the reflex response, of gradually increasing the total duration

of the stimulus without changing its intensity. Each signal has a rise time of 50 ms and a plateau varying from 0 to 200 ms. The RMS sound pressure level was 100 db for each signal. Each figure represents the average of 100 responses. Here again it may be seen that signal duration alone may effect reflex response. At levels above threshold, the reflex response increases in amplitude as the signal length is prolonged, while holding signal amplitude constant until about 200 ms, when further signal prolongation simply lengthens the response but adds no amplitude. The evidence presented so far confirms the well-known temporal integration occurring at sensation threshold and suggests that a similar mechanism might be operative in the reflex both at threshold levels and above.

Up to this time, the time-amplitude envelope of the signals has been either triangular or trapezoidal. In order to test the reflex response to a linear increase in signal amplitude, it was necessary to change to a signal whose amplitude increased linearly over a relatively long period of time. The signal used was a ramp, with approximately a 40 db linear increase in sound pressure level, a duration of either 1 or 2 seconds, followed by an abrupt cut off. In Figure 8 it is seen, in two different subjects, that if the peak amplitude of the signal is close to reflex threshold, then the reflex is in operation for only the very last portion of the stimulus and never reaches a very great amplitude. On the other hand, if the peak value is more than 10 or 15 db above threshold, then the reflex reaches its zenith very early and in essence remains "pinned" for most of the duration of the stimulus. However, if the peak amplitude is set at some intermediate value, it may be demonstrated that the reflex will begin early, increase linearly with the signal amplitude, and reach its own peak just after the signal turns off. (It should be noted that in this series the signal turns off at the tenth time marker on each trace.) A close examination of these traces indicates that the span of linear response of the reflex is about 18 db. Note, that this does not indicate that the reflex provides 18 db of attenuation or "protection" but implies that if the reflex function is, in part, a protective one, then it can probably perform linearly in this capacity over at least an 18 db range above its threshold. The relatively steep slope of the trace after the sound is turned off and the tendency to proceed below the equilibrium point are characteristics of the response commonly seen using this signal shape. Note that the response to this signal configuration is similar whether the pressure change is predominantly positive pressure or negative.

Up to this point, we have limited ourselves to a description of the intra-aural muscle reflex as a unit--we have declined to recognize its

component parts, the stapedius and tensor tympany muscles. So far, we have defined for this unit, threshold values, relation of signal intensity and duration in its effect on the reflex response, the DC capability of the response and lastly, the ability of the reflex to respond linearly over a defined range. A thorough search of the literature will convince the reader that the problem of whether or not the tensor tympany muscle responds to acoustic stimuli in normal humans has not been solved. The stapedius muscle does respond to acoustic stimuli in animals and in man (Wersäll, 1958). The tensor has been seen to behave similarly in some animals. Without laboring the issue, it is fair to say that a large group of investigators hold that in the human, only the stapedius muscle responds to acoustic stimuli (Klockhoff, 1961). The first indication that we might have evidence for the action of both component muscles in response to sound was of course the fact that we early demonstrated both negative pressure and positive pressure changes as a response to acoustic signals, a situation difficult to explain on the basis of a single muscle reflex. However, the most suggestive evidence developed when we began using the ramp signal. The good possibility existed that the "plateau effect" was occasioned by the beginning contraction of a second, antagonistic muscle--that the oscillations occasionally seen were the effects of the two muscles pulling against each other--that the rapid return of the pressure trace to and then below the equilibrium point (quite unlike the relaxation of a single muscle) was the result of the unopposed action of the antagonist and the subsequent gradual return to baseline, a typical relaxation of a single muscle. One other point may be mentioned here--perhaps the reason for the existence of that 5% of subjects in whom no reflex could be demonstrated, is that in these few subjects, the two component muscles have very nearly the same thresholds and contract with nearly equal strength so that the resultant displacement of the tympanic membrane, in these cases, was so small as to be below the level of detectability of our system. A more obvious display of the action of the two muscles may be seen in Figure 9. In this case, near threshold level, a stimulus elicits a negative response with a slow return to baseline. As the signal is increased in loudness, less and less of the total response is formed by this negative response. By the time the signal has increased by 20 db, the major portion of the response is taken up by the positive deflection. Note the oscillations mentioned previously. In the past it has been suggested that the stapedius muscle can move the tympanic membrane outward and create a positive pressure in the external canal. Certainly, it is difficult to see how the tympanic muscle could do anything but pull the membrane inward, and produce a negative pressure. The results presented seem to indicate that both these statements are true.

The final answer to these questions lies in the examination of patients with only one functional muscle. Much difficulty is encountered in this attempt since most patients we have examined have had bilateral disease and it was impossible to deliver enough sound to the contralateral ear to be sure we were above the consensual reflex threshold levels. In Figure 10, the first trace is the average response to 100 acoustic stimuli of a patient without a stapedius muscle (post-stapedectomy for otosclerosis) and demonstrates the negative pressure response of the remaining tensor tympany muscle. The second trace is the response of a patient to sound stimuli before operation for otosclerosis. At operation, the stapes was completely fixed in oval window and the stapedius muscle intact, a situation which precluded the normal function of the latter muscle. The response is again negative, characteristic of the unopposed action of the tensor tympany muscle.

The last trace is from a patient with a preoperative diagnosis of otosclerosis. The obvious biphasic activity demonstrated in this preoperative test mitigated against the diagnosis. At operation, there was no demonstrable middle ear disease. This phase of this investigation, i.e., aid in clinical diagnosis, is one that should be explored further.

SUMMARY

1. The particular modification of the method of tympanomanometry as it is employed in our laboratory has proven to be a useful tool in the investigation of the normal human intra-aural muscle reflex.

2. Some of the signal-response relationships of this reflex have been investigated and discussed.

3. Evidence was presented which supports the thesis that both the stapedius and tensor tympany muscles in the human respond to sound stimuli.

4. The suggestion is made that this technique lends itself to increasing the accuracy of preoperative diagnosis of some forms of conductive hearing loss.

REFERENCES

Fletcher, H. Speech and Hearing in Communication. D. Van Nostrand Company, Inc., 1953.

Wersäll, R. The tympanic muscles and their reflexes. Acta Oto-Laryngol., 1958, Suppl. 139.

Klockhoff, I. Middle ear reflexes in man. Acta Oto-Laryngol., 1961, Suppl. 164.

Fig. 1. Apparatus as it appears during testing procedure. Plug unit in place, connected to Altec 21C microphone by length of polyethylene tubing. Insert demonstrates plug unit.

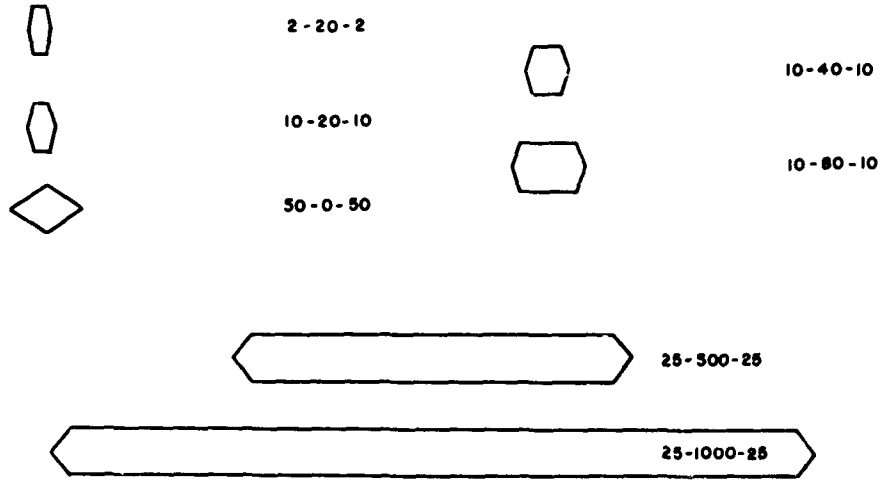

Fig. 2. Schematic diagram of acoustic stimuli tested. Time scale in milliseconds of rise time, constant amplitude phase, and decay time.

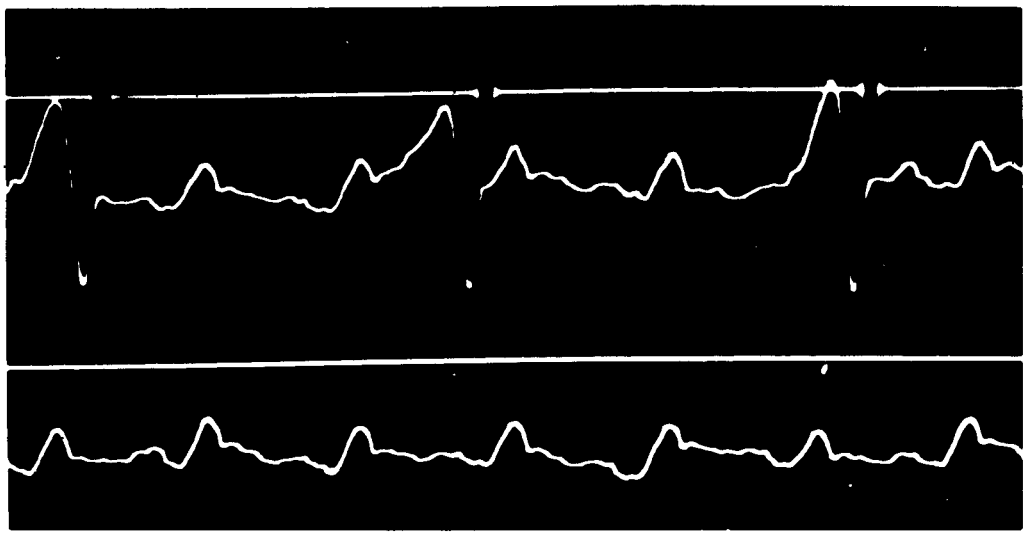

Fig. 3. Example of data record using AC system. Top pair of traces demonstrates the periodic pressure pulse fluctuations in the sealed external auditory canal in the absence of acoustic signal. Bottom pair of traces demonstrates the reflex in response to an acoustic signal superimposed on the pulse trace.

Fig. 4. Typical record from Computer of Average Transients (CAT). Positive pressure in the sealed external auditory canal inscribes an upward deflection. Time proceeds from left to right. Equally spaced time markers divide total analysis time into 20 equal intervals. First trace: Single sweep of CAT, analysis time of 32 seconds. Represents DC response of system to a positive pressure of 10 mm alcohol during last 16 seconds of record. Second trace: D.F., CAT record, average of 100 responses (X100), analysis time of 2 seconds, signal: 1600 cps, 40-1000-40. Typical response of a predominantly negative pressure reflex. Third trace: J.C., CAT record, X100, analysis time of 1 second, Signal: 1600 cps, 40-500-40. Typical response from another subject of a predominantly positive pressure reflex.

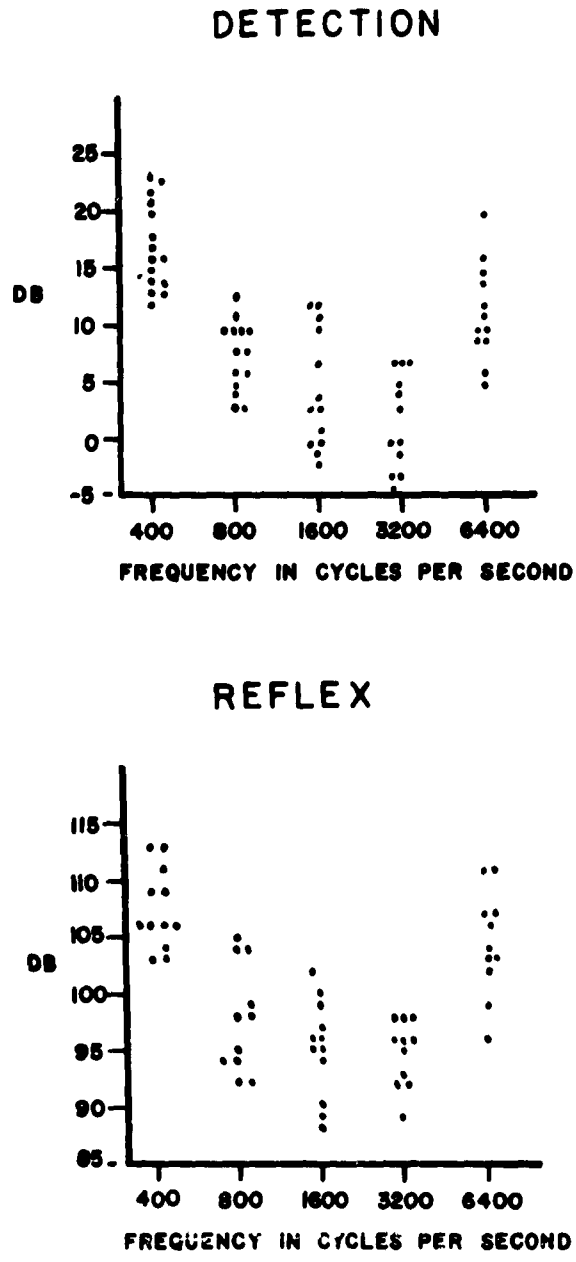

Fig. 5. Individual threshold values for members of the panel in response to signal 50-0-50. Signal amplitude in db. ref. 0.0002 dynes/cm^2.

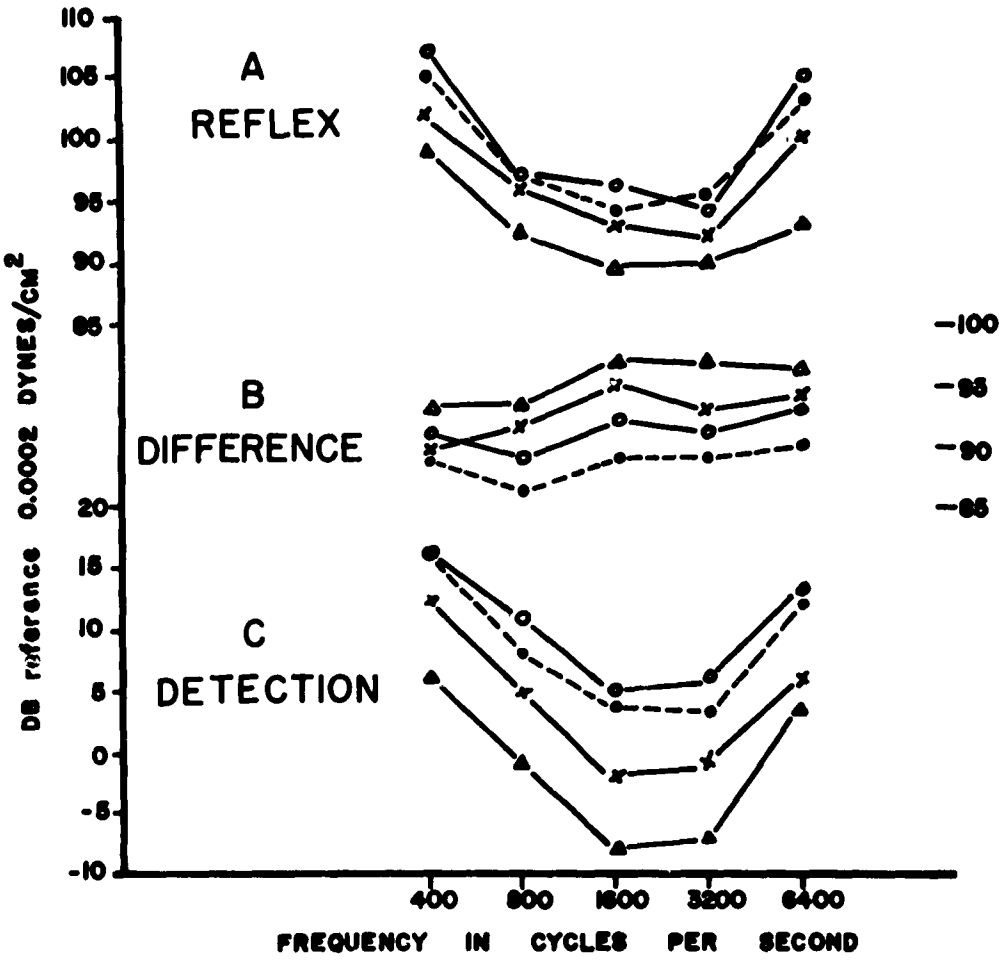

Fig. 6. Average response of panel members to acoustic signals at reflex threshold (A) and at detection or sensation threshold (C). Signals are grouped according to increasing acoustic energy. o -- group 1A = 50-0-50, 2-20-2, 10-20-10. • -- group 1B = 50-0-50 with banded noise. x -- group 2 = 10-40-10, 10-80-10. Δ -- group 3 = 25-500-25, 25-1000-25. The family of curves in B represents the average reflex threshold in db above sensation level, e.g., A-C = B.

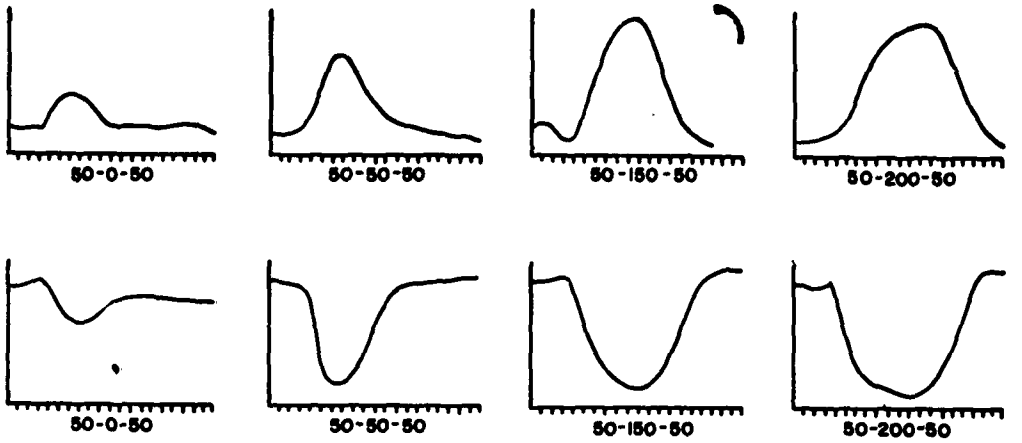

Fig. 7. Reflex response to increasing signal duration without changing signal amplitude (100 db). CAT record, X100, analysis time of 0.5 second, Signal: 800 cps. Time-amplitude envelope of signal given under each trace. Top group: D. D. Reflex response demonstrates positive pressure change. Plateau of response begins to appear with total signal duration of 250 ms. Bottom group: D. F. Reflex response is predominantly a negative pressure change and also forms a plateau when total signal duration reaches 250 ms.

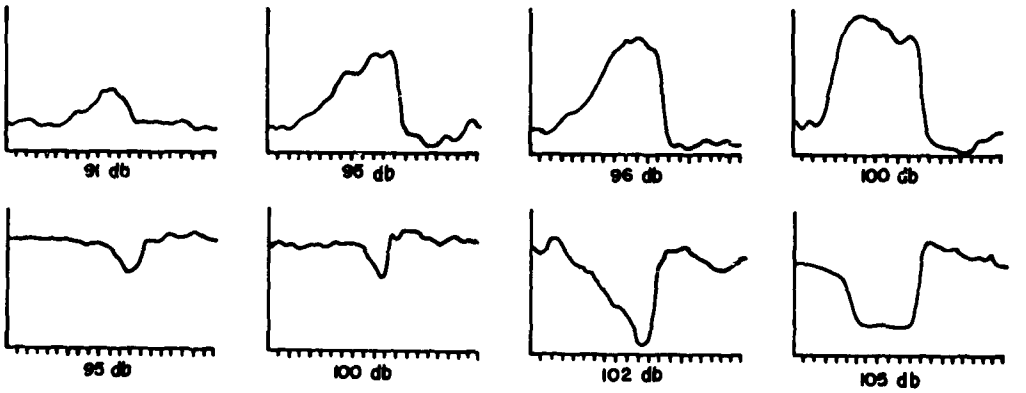

Fig. 8. Reflex response to ramp function; total signal duration of 2 seconds and total analysis time of 4 seconds. Final amplitude reached by ramp is noted under each trace. CAT record x 50, Signal: 800 cps. Ramp signal has linear increase in amplitude over range of about 40 db. Top group: H. W. Predominantly positive pressure response of subject to ramp signal. Note the linearity of the pressure curve when the peak of the signal is 95 db. Bottom group: D. F. Negative type pressure response to the same signal configuration. Note again the linear response, in this case when the signal peaks at 102 db.

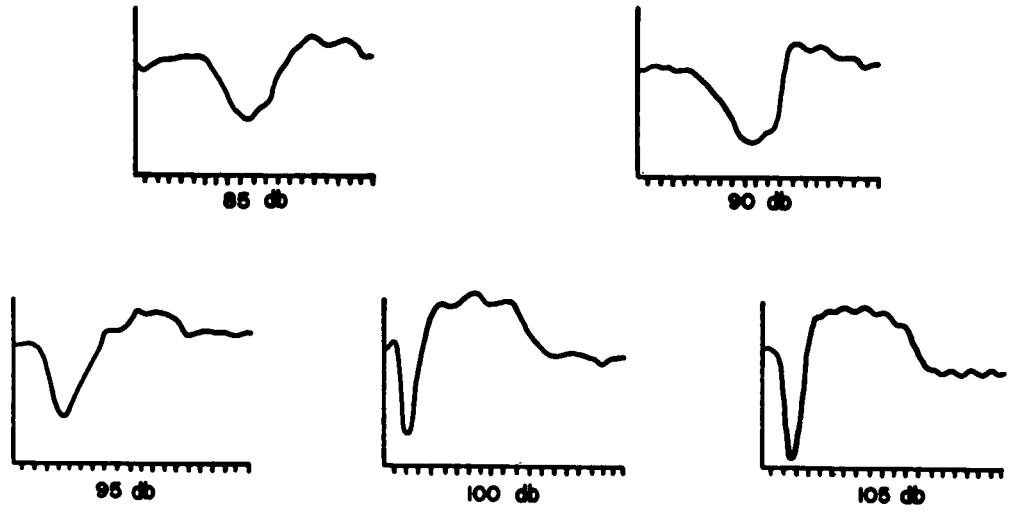

Fig. 9. Demonstration of conversion of monophasic negative pressure response to biphasic response by increasing signal amplitude. CAT record X50, analysis time 2 seconds, 800 cps, 1 second ramp signal.

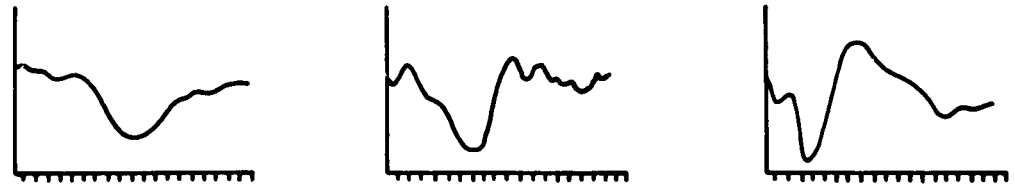

Fig. 10. First trace: Negative pressure reflex in 53 y. o. male, post-stapedectomy. CAT record X100, 1 second analysis time, 800 cps, 40-60-40. Middle trace: Negative pressure response in 40 y. o. male, pre-operative. Operative Diagnosis: severe stapes fixation. CAT recording X100, 1 second analysis time, 1600 cps, 40-60-40. Last trace: Biphasic pressure response in 28 y. o. male, pre-operative diagnosis of otosclerosis. Operative diagnosis of no middle ear disease. CAT record X100, 1 second analysis time, 1600 cps, 40-60-40.

PSYCHOPHYSICAL CORRELATES OF INTRATYMPANIC REFLEX ACTION

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In the past several years there has been a considerable resurgence of interest in the mechanism of the intratympanic acoustic reflex (AR). As Ward (1960) and others have pointed out, this is true not only because of the potential practical value which artificial elicitation of the reflex might have in protecting the hearing of individuals and in clinical diagnosis but also because of the realization of other possible implications. Recent research indicates that the intratympanic muscle action should possibly be taken into account in explanations of masking, remote masking, the scaling of loudness, mechanisms of attention, and intersensory effects.

NATURE OF THE REFLEX

The intratympanic muscle reflex is a reflexive response on the part of two small muscles of the inner ear. One, the tensor tympani, runs from the cartilaginous portion of the Eustachian tube to the manubrium or handle of the malleus (hammer); the other, smaller muscle, the stapedius, lies within the posterior wall of the middle ear and its tendon inserts on the head of the stapes (stirrup).

The tensor tympani is innervated by a branch of the trigeminal nerve and may receive sympathetic innervation (Byrne, 1938). It has also been reported recently that the tensor is innervated by a branch of the glossopharyngeal nerve (Lawrence, 1962). The stapedius is innervated by a branch of the facial nerve. Seemingly the muscles receive little or no sensory innervation (Blevins, 1962). In response to activating stimuli--usually loud sounds--the stapedius and tensor tympani pull in such a manner as to damp, limit, or reduce the acoustic input to the cochlea. The direction of pull of the muscles is at right angles to the axes of their respective ossicles in such a fashion that the direction of action of one is almost directly opposed to that of the other (Wever and Lawrence, 1954). It has long been known that the reflex is bilateral. Acoustic stimulation of either ear elicits the reflex in both ears (Pollak, 1886; Hammerschlag, 1899). While it is apparent

that the reaction to acoustic stimulation is bilateral, there is some question as to whether the homolateral and contralateral responses are equal in magnitude. Detailed descriptions of tensor tympani and stapedium anatomy and physiology are available elsewhere (Kobrak, 1957; Wever and Lawrence 1954; Perlman, 1960; Hilding and Fletcher, 1960).

It has been suggested that the noise-attenuating properties of the reflex are primarily attributable to action of the stapedius muscle (Galambos and Rupert, 1959; Simmons, 1959). As some observers have emphasized, the action of the muscle is somewhat analogous to that of the iris and blink reflexes of the eye. However, because of the sharp rise time and brief duration of certain acoustic stimuli, in comparison to the latency of the reflex, and the relatively long duration of other acoustic stimuli, in comparison with the fatigue time of the reflex, the protection offered the ear by the intratympanic reflex is not as adequate as that afforded the eye by the iris reflex.

MEASUREMENT OF MUSCLE ACTION

A remarkable diversity of techniques has been employed to measure the action of the intratympanic muscles. Large contractions may be seen with the naked eye or with a microscope, and somewhat smaller ones may be viewed by observing objects inserted into the muscle tendon. Hensen made direct observation of the tensor tympani in 1878 and demonstrated that the reflex was elicited by acoustic stimulation. With the stapedius, an appreciably smaller muscle, direct observation is more difficult. Kato (1913) made myograms of stapedial movements by tying a thread to the muscle and connecting it to a stylus on a rotating drum. Today myograms are usually made by monitoring the output of mechanoelectronic transducers fixed in the muscle tendons (see e.g., Wersäll, 1958).

The movements of the middle ear muscles, especially those of the tensor tympani, result in movements of the eardrum. Stapedius contractions produce an eardrum extrusion; tensor tympani contraction, a retraction. The drum has been directly observed (Hammerschlag, 1899; Waar, 1923; Kato, 1913; Lüscher, 1929), but small movements are quite difficult to detect. Köhler (1910) attached a small mirror to the drum and observed changes in the reflected light. While it is possible to detect smaller movements with this technique, there is probably some loading of the membrane. Other related methods include measurement of changes in volume or pressure produced by eardrum movement (Tsukamoto, 1934; Terkildsen, 1960; Mendelson, 1960, 1961)

and changes in acoustic impedance of the ear (Metz, 1951; Jepsen, 1951; Klockhoff, 1961). The acoustic impedance of the ear may be defined as resistance to motion on the part of aural structure when acoustic energy is applied. It is a function of the stiffness, mass, and resistance (friction) of the structures and may be estimated by certain pressure or acoustic measurements at the ear canal (cf. Zwislocki, 1961). Manometric, volumetric, and impedance change techniques are objective (although some experimenter judgment is required regarding presence or absence and magnitude of response) and fairly sensitive. It is possible that the impedance change method is more sensitive to tensor tympani than to stapedius action (Reger, 1960), and it is not always possible to determine both tensor and stapedius response on the same manometric recording (Mendelson, 1961). While the latencies of the muscle reflex vary, overlap is possible, and the changes produced by the two may cancel. Moreover, neither the manometric or impedance change procedure gives a direct indication of actual protection afforded by the reflex--the reduction of sound energy impinging on the oval window attributable to action of the acoustic reflex.

MEASURES OF ATTENUATION: REDUCTION OF COCHLEAR MICROPHONIC AND CHANGES IN PSYCHOPHYSICAL INDICES

One index of amount of protection afforded by the acoustic reflex is the reduction in cochlear microphonics produced by reflex activation (Wiggers, 1937; Wever and Bray, 1942; Galambos and Rupert, 1959; Simmons, 1959). A reduction in the ipsilateral or contralateral response to a loud tone may be noted after introduction of an activating stimulus. Estimates of maximum attenuation range from 10-20 db (Galambos and Rupert, 1959) to 40 db (Wiggers, 1937). It is reported that protection is greatest for tones below 2000 cps (Simmons, 1959).

While the cochlear microphonic technique has yielded the most data regarding amount of protection afforded by reflex action, it has certain limitations. Cochlear potentials are not readily recorded from humans, although in the course of certain surgical procedures they have been recorded (Ruben, Bordley, and Lieberman, 1961). A more serious objection is that at a moderately intense level, the cochlear microphonic no longer responds linearly to sound (Davis, 1961) and may even be distorted (Lawrence, Wolsk and Schmidt, 1962).

Alternatively, attenuation may be estimated by changes in several psychophysical indices--notably absolute threshold, loudness, and temporary threshold shift. Von Békésy and Rosenblith (1951) were perhaps

the earliest to report the use of psychophysical determination to measure attenuation afforded by the acoustic reflex. They introduced a loud pure tone into one ear and observed the attendant change of loudness in a tone presented to the contralateral ear. The changes observed indicated attenuation values of 5-10 db. Shapley (1954) introduced a loud thermal noise in one ear and noted changes in the loudness of a tone in the contralateral ear. (Loudness reduction was measured by requiring a subject to match the intensity of a tone presented during the contralateral stimulation to the intensity of a tone in the absence of such stimulation.) Attenuation values ranging from 6-15 db were obtained. There was some possibility, however, of direct masking due to bone conduction of the thermal noise from one side of the head to the other. Loeb and Riopelle (1960) employed a method in which observers were asked to track their threshold at 125, 500, or 1000 cps by a Békésy technique with and without a loud 2000 cps tone introduced into the contralateral ear. The difference in thresholds in the presence and absence of the contralateral 2000 cps tone was assumed to be an index of reflex attenuation. With test and activating tones at these frequencies, the attenuation through the skull is such that masking should not be a factor. Threshold shifts were quite small--approximately 3 db. Threshold shifts obtained by the conventional method of limits were of the same order of magnitude. In a second part of the same investigation a loudness balancing technique similar to that employed by Shapley (1954) was used, but a 2200 cps tone, rather than a thermal noise, was employed to activate the reflex. The data indicated that the loudness of intense tones was reduced to a greater degree than that of tones of lesser physical intensity. It was suggested, on the basis of these experiments and the discrepancy between the threshold shift data and earlier experiments employing cochlear microphonics that the reflex might act as a limiter rather than an attenuator, so that intense sounds may be attenuated considerably and faint sounds relatively little. Wever (1962) states that this hypothesis is contrary to other observations (not enumerated) and suggests instead a sort of constancy mechanism--a central process by which action of the reflex is taken into account. It is difficult to see, however, how this latter explanation could account for the small threshold shifts obtained. It should be pointed out that the results of loudness balance experiments by Reger (1960) do not support the hypothesis of differential attenuation of faint and intense sounds, while data from somewhat similar experiments by Prather (1961) tend to corroborate the Loeb and Riopelle (1960) findings.

An alternate explanation advanced (Loeb and Riopelle, 1960) was that subjects may be unconsciously--or consciously--inhibiting

the reflex during the psychophysical task. Smith (1943) and Reger (1960) have described individuals who have voluntary control of the acoustic reflex. According to Reger (1960), only one or two per cent of the population is so talented, but it is possible that a much larger percentage may inhibit their reflexes unconsciously when it is advantageous to do so. In any event, it has been demonstrated that if loud high frequency thermal noise is introduced into one ear and the resultant contralateral threshold shift is measured, larger shifts (as much as 15 db) are obtained than when the activating sound is a pure tone (Ward, 1961; Loeb and Fletcher, 1961). Fatigability of the reflex is also seemingly less when it is activated and maintained with a noise stimulus than when a tone is employed. Clicks are at least as effective in arousing and maintaining the reflex as noise (Fletcher and Loeb, 1962). Moreover, the difference is attributable to the changing quality of noise and click stimuli rather than their complexity, for square waves are no more effective than pure tones in producing contralateral threshold shift (Fletcher and Loeb, 1962). The fact that changing stimuli are more effective in producing contralateral threshold shift than steady ones might be interpreted as evidence supporting the notion that subjects may be somehow inhibiting reflex action during psychophysical measurements. Presumably with ever-changing activating stimulus, this should be harder to do. A more parsimonious explanation might be that reflex adaptation is very rapid for pure tone stimuli and less so for changing ones. Wersäll (1958) stated that the intratympanic muscle reflexes decline in strength over time but that presentation of a tone of different frequency brings the contraction back to full magnitude. Simmons (1959) noted that a frequency-modulated tone produces a more sustained reduction of cochlear microphonics in the contralateral ear than a steady one. Whether adaptation of the reflex is sufficiently rapid to account for the observed differences in contralateral threshold shift is debatable.

Another method of estimating reflex attenuation is the temporary threshold shift reduction technique (Fletcher and Riopelle, 1960; Fletcher, 1961; Ward, Selters, and Glorig, 1961). In this technique a train of loud impulsive noises sufficiently intense to produce a reversible loss of auditory acuity is presented to the observer, but a tone loud enough to activate the reflex is presented before each impulsive noise. (The impulsive noises themselves arouse the reflex, but the muscles do not achieve full contraction before the impulsive noises terminate.) Such a procedure has been found to reduce considerably the amount of temporary threshold shift produced by the traumatic stimulus. The average reduction was comparable to that

achieved by the use of earplugs known to attenuate pure tones by amounts ranging from 15 db at low frequencies to 45 db at high frequencies. An average figure, however, is not very meaningful. More TTS at low frequencies was noted when subjects wore earplugs and more TTS at high frequencies when observers were protected only by reflex elicitation.

Hilding (1961) employed a closely related technique. He demonstrated that both temporary and permanent losses in cochlear microphonic responses in cats, following exposure to impulsive noise, may be reduced or eliminated by presenting a tone loud enough to activate the acoustic reflex before each of the impulsive sounds. While such a technique has obvious advantages, it must be used with caution. Simmons (1960a) has shown that cochlear microphonic changes do not necessarily correlate with changes in neural sensitivity.

There have been some recent attempts to correlate certain physiological and psychophysical measures of reflex action. Loeb and Fletcher (1961) measured and correlated contralateral threshold shift and TTS reduction (TTSR) for 16 observers. Correlations between these were found to be negligible. Mendelson and Fletcher (1962) attempted to determine relationships between these same psychophysical indices and manometric indices of acoustic reflex action. In this instance some data suggestive of functional relationship between TTSR and the manometric techniques were obtained, but the relationships were not simple. A recent article (Loeb and Fletcher, 1962) presents data which may explain these results. It describes experiments in which estimates of reliability of contralateral threshold shift were determined. It was found that while contralateral threshold shift (CTS) in response to high frequency noise or clicks occurred quite regularly, the magnitude of the threshold shift varied somewhat, and reliability was surprisingly low (approximately 0.50 - 0.60 immediately after inroduction of the activating stimulus with experienced subjects and lower in magnitude at later intervals or with inexperienced observers). Reliability of temporary threshold shift was somewhat higher (approximately 0.70 - 0.80 immediately after termination of the traumatic stimulus with experienced observers, lower at later intervals or with inexperienced observers). Temporary threshold shift reduction reliability was not determined, but it might be expected to be somewhat lower than that of temporary threshold shift due to variability in the reflex action itself. With reliability of this magnitude it is apparent that non-significant correlations between the indices might very well be obtained. While similar information for manometric and acoustic impedance change techniques is not available in the form of

reliability coefficients, it is known that there is considerable within-observer variability in reflex action (Mendelson and Fletcher, 1962). It is also quite probable that a certain amount of variance is introduced in reading and interpreting the recordings obtained by manometric and acoustic impedance change methods.

LIMITATIONS OF REFLEX PROTECTION

It has already been mentioned that the latency of the acoustic reflex is such that little or no attenuation of very short, intense impulsive sounds (e.g. gunfire) occurs under normal circumstances. Various values have been given for the latency of the reflex. Some of this variance is attributable to the fact that the tensor tympani and stapedius have different latencies and some to the fact that latency may be defined either as the time before contraction begins or the time before it is complete. Kobrak (1959) proposed that the term "latency" be applied to the former interval and term "contraction time" to the latter, and this convention will be followed here. He states that the contraction time of the stapedius is approximately 20 msec and that of the tensor tympani 200 msec. Wersäll (1958) states that latency varies somewhat with stimulus intensity, averaging approximately 7-10 msec for stapedius and 12-16 msec for tensor tympani. (These values are for cats under superficial phenobarbital anesthesia, rather similar values were obtained with rabbits.) Contraction time (three cats) ranged from 22-38 msec for stapedius and from 56-122 msec for tensor tympani. All of Wersäll's values were obtained by myographic techniques. Klockhoff (1961) reported somewhat higher values, but as he employed an acoustic impedance change procedure in recording activity and elicited the reflex by non-acoustic means, the values should not necessarily be comparable. With manometric procedures (Mendelson, 1961) it is possible to distinguish stapedius and tensor tympani action because they produce opposite movements of the eardrums, but if the responses tend to overlap it is apparent that an accurate indication of latency and contraction time would be difficult to obtain. Cochlear microphonic experiments (Galambos and Rupert, 1957) have indicated reflex (presumably stapedius) latencies of the order of 10 msec. (Estimates of contraction time were not given and are not easy to determine from the records presented.) In any event, though estimates of latency and contraction time vary, it would seem that stapedius contraction requires at least 10 msec and tensor tympani contraction a longer period. There is some evidence that the tensor tympani response is part of the startle reflex pattern (Klockhoff, 1961) rather than a true response to acoustic stimulation, or that it plays little role in reducing acoustic input

to the cochlea (Galambos and Rupert, 1959; Simmons, 1959). If so, then the effective time of response can be taken as being between 10 and 50 msec. Since many impulsive sounds have rise times less than 0.1 msec and durations ranging from 10-75 msec (Doelling and Kryter, 1959) the protection against impulsive noise trauma afforded by the reflex is minimal.

A second limitation of the reflex attenuation is its gradual adaptation to a continuously presented sound. The rate of adaptation apparently varies with frequency, being relatively abrupt for a high frequency tone and considerably less so for lower frequency tones. Adaptation apparently begins immediately but may not be complete after 1.75 minutes (Simmons, 1959). If the frequency of the reflex-eliciting acoustic stimulus is continuously changing (Wersäll, 1958; Simmons, 1959) or if constantly changing in amplitude like a thermal noise or series of clicks (Ward, 1961; Fletcher and Loeb, 1962), the adaptation time may be very much lengthened. The practical consequence of the adaptation phenomenon is that reflex action not only is ineffective against short impulsive stimuli but also against relatively sustained stimuli. However, it is probable that some protection is offered under most circumstances, as animals with sectioned stapedius muscles show greater losses after exposure to continuous noise (Galambos and Rupert, 1959).

After termination of the activating stimulus, relaxation begins almost immediately, if the stimulus is at reflex threshold intensity, or shortly afterward, if the stimulus is considerably above threshold (Weiss, 1962). However, studies employing acoustic impedance change (Metz, 1946), myographic technique (Wersäll, 1958), and cochlear microphonic reduction techniques (Galambos and Rupert, 1959) all indicate that some contraction may be present for a period of as much as one second, though the relaxation period is generally shorter.

In addition to the differential adaptation rate for high and low frequencies previously mentioned, there is also, apparently a differential protection according to frequency (Wever and Bray, 1937, 1942; Wever and Lawrence, 1954; Simmons, 1959). In general, it is not entirely clear whether the difference is due to differential elicitation of the reflex by tones of different frequency or differential attenuation of tones of different frequency, though the Wever and Bray experiments suggest that the latter explanation is correct. (They found that when the tendons were pulled by external weights, attenuation was greater for lower frequency tones.) In an experiment by Wever and Vernon (1956) tension along the line of the tensor tympani

was related to changes in cochlear microphonic. While some animals showed fairly good relationships between tension and microphonic reduction, others did not. The stapedius activity was apparently not measured to any considerable degree. In any event, present data indicate that little protection may be expected from the reflex at high frequencies, especially above 7,000 cps (Simmons, 1959). Whether this is true, if the reflex is artificially excited, remains an open question.

The threshold for elicitation of the reflex is usually taken to be 70-90 db (Wersäll, 1958). Apparently the threshold is somewhat lower at higher frequencies (Wersäll, 1958; Simmons, 1960b), and it also varies with duration (Weiss, 1962). With repeated stimulation, post-tetanic potentiation may occur and the reflex threshold may be lowered approximately 24 db (Simmons, 1960b). It should be reiterated that at levels below the reflex threshold the muscles apparently maintain a state of contraction such that some sound attenuation is provided (Simmons, 1959; Lawrence, 1960).

It is usually assumed that the acoustic reflex is completely consensual (Wever and Lawrence, 1954). Studies of non-acoustic elicitation of the reflex indicate that reflexes so elicited may be completely homolateral (Klockhoff, 1961) or partially so (Pichler and Bornschein, 1957). There is also evidence that the reflex to acoustic stimulation may not be as great in the contralateral as in the homolateral ear. An experiment employing the acoustic impedance change technique (Möller, 1961) and another employing a loudness balance technique (Reger, 1960) yielded data best interpreted in this fashion.

NON-ACOUSTIC ELICITATION

It has long been known that contraction of the middle ear muscles may be elicited by non-acoustic means. Lawrence and Wever (1954) reviewed the claims of several investigators who describe voluntary contractions of the middle ear muscles. They suggested (citing Schrapinger) that some (not all) individuals who believe that they are contracting the tensor tympani are actually contracting the veli palatini, a muscle of the palate attached to the Eustachian tube. Smith (1943) reported an individual with voluntary control of middle ear muscles and measured changes of threshold during contraction. It is interesting that the changes in loudness reported during contraction were not necessarily correlated with threshold changes. Reger (1960) also reported individuals with voluntary control and described results somewhat similar to Smith's. Reger's findings previously cited indicate

that only one to two per cent of the population has voluntary control of muscle movement.

Since, through conditioning, a number of responses have been shown to be capable of elicitation by previously neutral stimuli, it seems at least possible that the intratympanic muscle response may also be so elicited. Simmons, Galambos, and Rupert (1959) have in fact reported that if light flashes are repeatedly paired with loud auditory stimuli the light flashes alone will eventually elicit the muscle response. Ward and Fleer (1961) on the other hand, found that there was no difference in temporary threshold shift produced by trains of high intensity clicks with periodic and aperiodic inter-click intervals. This indicates that the reflex may not be temporally conditioned.

A number of researchers have reported eliciting middle ear muscle reflexes with cutaneous stimuli. Kato (1913) reported contractions in response to various types of aural and facial stimulation. Wersäll (1959) elicited muscle contraction by mechanical stimulation of the pinna and auditory canal. Klockhoff (1916) reported that electrical stimulation of the auditory canal produced a unilateral stapedius reflex, while tensor tympani action could be elicited only bn an orbital air jet. The latter response was interpreted as being a component of a startle reaction. Pichler and Bornschein (1957) report a 10-15 db increase in the homolateral threshold, attributed to intratympanic muscle action, as a result of electrical stimulation of the external auditory canal. A small contralateral threshold shift (up to 5 db) was also reported. Attenuation of lower frequencies was appreciably greater than that for higher frequencies, and by anesthetic techniques the possibility of direct stimulation of the muscles at the intensities employed was ruled out. Djupesland (1962) elicited homolateral and contralateral stapedius reflexes (indicated by impedance change) and an occasional small tensor tympani response by aural air puffs. It is not certain whether the reflex was due to cutaneous or acoustic stimulation.

Hugelin, Dumont, and Paillas (1960) report data indicating elicitation of response of the intratympanic muscles by action of the mesencephalic reticular system. This is discussed below.

APPLICATIONS

The most obvious application of the research reported is in construction of a new kind of ear protective device. Fletcher and

Riopelle have applied for a patent on an apparatus which will present tones or other reflex-eliciting stimuli a short period before the presentation of impulsive sounds. Such a device would have advantages over other protective devices--e.g., earmuffs or earplugs--in that no active cooperation on the part of the subject is necessary. (Anyone who has tried to elicit such cooperation will appreciate the importance of this factor.) It is probable that more effective reflex eliciting stimuli will be discovered. Chisman and Simon (1961) suggest that reflex-eliciting tones of gradual onset might be less injurious. However, Fletcher's data (1961b) indicate that his activating tones with abrupt onset did not induce appreciable auditory fatigue and so presumably are not injurious. In addition, there is evidence that tones of abrupt onset elicit the reflex more effectively (Weiss, 1962).

Present indications are that the artificial elicitation of the reflex will be of practical value primarily for protection against impulsive rather than continuous traumatic noise. An attempt to reduce TTS following exposure to loud continuous noise by presenting a moderately loud monaural tone during exposure to the noise was unsuccessful (Fletcher, 1961a). However, when either narrow band noise or a train of clicks was presented monaurally during a 25-minute exposure to broad band noise in the contralateral ear, the TTS produced by the broad band noise was significantly reduced (Fletcher, 1961b). Whether protection of this kind would be noted following a longer exposure and whether a system incorporating the reflex-eliciting principle would be practical for long continuous-noise exposures is not known, but further exploration of these problems is planned.

A second major application of the findings is in the determination of criterion for exposure to impulse noise. Rather widely accepted criteria for safe exposure to continuous noise already exist (Glorig, Ward, and Nixon, 1961) but comparable standards for exposure to impulsive noise are not available. In the past, it was generally assumed that as impulsive noises are more closely spaced, opportunity for recovery declines, and for the same number of impulses, the danger to hearing increases. However, if the impulses are spaced together closely enough, the reflex activated by one impulse should reduce the inner ear input resulting from the successive one (Ward, 1962). Carter and Ball (1962) have actually demonstrated such an effect. They find that intense impulses presented at 5 and 10 pulses per second are less damaging than the same number of impulses presented once per second. Similar results have been reported by investigators at the Army Human Engineering Laboratory at Aberdeen, Maryland (Donley and Smith, 1961).

A third major application would be the determination of reflex excitability for use in clinical diagnosis. Klockhoff (1961) presented data indicating that if a stapedius reflex may be elicited, either through acoustical or electrical stimulation, some middle ear conduction must be present. Djupesland (1962) presented data indicating that this principle may not be entirely general, but his method of reflex elicitation (aural air puff) differed from Klockhoff's somewhat. Absence of such a response would indicate conductive loss. (It might also indicate fifth nerve impairment, but in the absence of other symptoms this is improbable.) If a tensor tympani response may be elicited (by orbital air puff stimulation) and the stapedius reflex may not, this is indicative of otosclerosis involving stapes ankylosis. Failure to elicit either reflex indicates a more extensive conductive loss. Failure to elicit the stapedius reflex when auditory function appears normal and the tensor tympani is readily elicitable may be taken as a diagnostic sign for facial nerve lesion (Klockhoff, 1961; Jepsen, 1955).

IMPLICATIONS FOR RESEARCH

It has been suggested that changes in remote masking (masking of low frequency sounds by high frequency sounds) may be attributable to acoustic reflex activation (Ward, 1961; Burgeat and Hirsh, 1961). It is probable also that a great deal of the effect usually attributed to direct masking (masking of high frequencies by low) is in part due to acoustic reflex action rather than simply to overlap of activated neurons. If so, the degree of masking should change as a function of time, at least for steady masking sounds. Moreover, our perception of the relative loudness of sounds is in all likelihood influenced by acoustic reflex action (Reger, 1960; Ward, 1961). Reger suggests that the slope of the equal loudness contour for intense pure tones reflects this function. Loudness of pure tones might be expected to increase as a function of time due to reflex adaptation, but effects of this kind would be opposed by effects due to adaptation and fatigue.

A number of researchers (e.g., Hernández-Péon, Scherrer, and Jouvet, 1956; Galambos, Sheatz, and Vernier, 1956) have demonstrated reduction in evoked potentials elicited by auditory stimuli at various central loci as a function of repeated non-reinforced presentation or exposure to distracting stimuli as well as facilitation of such potentials following pairing with reinforcing stimuli. These phenomena have been explained on the basis of activity of the descending reticular activating system (Hernández-Péon, Scherrer, and Jouvet, 1956).

Hugelin, Dumont, and Paillas (1959) report that reticular stimulation reduces dorsal cochlear nucleus potentials in "encéphale isolé" cats. However, if the cats are curarized (thereby eliminating intratympanic muscle action) the effect is not obtained. They conclude, therefore, that the physiological mechanism discussed involves reticular system control of the intratympanic muscles. On the other hand, Mousheigan, Rupert, Marsh, and Galambos (1961) have shown that the habituation, conditioning, and distraction effects described may be recorded in cats without middle ear muscles. The conflict of results may be more apparent than real. Desmedt (1960) has presented data indicating that descending pathways not in the reticular system may be responsible for centrifugal control of auditory input. However, the possibility that the intratympanic muscles may exert some such regulatory influence may not be ruled out at this time.

It follows, further, that possible acoustic reflex action should be taken into account in other contexts. If sensory interaction experiments involving cutaneous facial stimulation and acoustical stimulation are performed, possible elicitation of the reflex and resultant attenuation of the sound should be considered. If a study of backward conditioning employs an air puff or an electric shock presented before a tone, it should be realized that the observer may, because of reflex action, not be experiencing the tone at full intensity. Wherever an intense stimulus, especially a cutaneous stimulus to the face or ear, is applied concomitantly with or shortly preceding an auditory stimulus, the possible effects of the reflex should be taken into account in interpreting findings.

SUMMARY

A review is presented of research on the intratympanic muscle reflexes. Among topics covered are a discussion of the methods employed for observation of reflex action, discussion of the natural protection against traumatic noise afforded by the reflexes and its limitation, and discussion of various types of stimulation (especially acoustic and cutaneous) which may be used to elicit the reflexes. Also described are possible practical uses of artificial reflex elicitation to provide enhanced protection against noise and for certain kinds of clinical diagnosis. Implications for damage risk criteria and for certain areas of psychological and physiological research are discussed.

REFERENCES

Blevins, C. E. Fine innervation patterns of the middle ear muscles. In Fletcher, J. L. (ed.) Seminar on middle ear function, US Army Med. Res. Lab. Rep., Fort Knox, Ky., 1962.

von Békésy, G. and Rosenblith, W. A. The mechanical properties of the ear. In Stevens, S. S. (ed.) Handbook of psychology. New York: Wiley, 1951, 1075-1115.

Burgeat, M. and Hirsh, I. J. Changes in masking with time. J. acoust. Soc. Amer., 1961, 33, 963-965.

Byrne, I. G. Studies of physiology of the ear. London: Lewis and Co., 1938.

Carter, N. and Ball, J. Studies of temporary threshold shift caused by high intensity acoustic transients. Bolt, Beranek and Newman, Inc., Tech. Rep., Cambridge, Mass., 1962, No. 916.

Chisman, J. A. and Simon, J. R. Protection against impulse-type industrial noise by utilizing the acoustic reflex. J. appl. Psychol., 1961, 45, 402-407.

Davis, H. Mechanism of excitation of auditory nerve impulses. In Rasmussen, G. L. and Windle, W. F. (eds.) Neural mechanisms of the auditory and vestibular systems. Springfield, Ill.: Charles C. Thomas, 1960, 21-39.

Desmedt, J. E. Neurophysiological mechanisms controlling acoustic input. In Rasmussen, G. L. and Windle, W. F. (eds.) Neural mechanisms of the auditory and vestibular systems. Springfield, Ill.: Charles C. Thomas, 1960, 152-164.

Djupesland, G. Intra-aural muscular reflex elicited by air current stimulation of the external ear. Acta oto-laryngol., 1962, 54, 143-153.

Doelling, N. and Kryter, K. D. Characteristics of noise produced by several contemporary Army weapons. Bolt, Beranek, and Newman, Inc., Tech. Rep., Cambridge, Mass., 1959, No. 630.

Donley, R. and Smith, G. Personal communication, 1961.

Fletcher, J. L. TTS following prolonged exposure to acoustic reflex eliciting stimuli. J. aud. Res., 1961, 1, 242-246 (a).

Fletcher, J. L. Prolongation of the action of the acoustic reflex. J. aud. Res., 1961, 1, 306-310 (b).

Fletcher, J. L. and Loeb, M. The influence of different acoustical stimuli on the threshold of the contralateral ear: a possible index of attenuation by the acoustic reflex. Acta oto-laryngol., 1962, 54, 33-37.

Fletcher, J. L. and Riopelle, A. J. Protective effect of the acoustic reflex for impulsive noise. J. acoust. Soc. Amer., 1960, 32, 401-404.

Galambos, R. and Rupert, A. Action of the middle ear muscles in normal cats. J. acoust. Soc. Amer., 1959, 31, 349-355.

Galambos, R., Sheatz, G., and Vernier, V. G. Electrophysiological correlates of a conditioned response in cats. Science, 1956, 123, 376-377.

Glorig, A., Ward, W. D., and Nixon, J. Damage risk criteria and noise induced hearing loss. Arch. Oto-laryngol., 1961, 74, 71-81.

Hammerschlag, V. Ueber den Tensorreflex. Arch. Ohrenheilk, 1899, 46, 1-13.

Hensen, V. Beobachtungen über die Thätigkeit des Trommelfell-spanners bei Hund und Katze. Arch. Physiol., 1878, 2, 312-319.

Hernández-Péon, R., Scherrer, H., and Jouvet, M. Modification of electric activity in cochlear nucleus during "attention" in unanesthesized cats. Science, 1956, 123, 331-332.

Hilding, D. A. and Fletcher, J. L. The protective value of the middle ear reflex. Int. Rec. Med., 1960, 173, 369-374.

Hilding, D. A. The protective value of the stapedius reflex: an experimental study. Trans. Amer. Acad. Ophthalmol. Otolaryngol., 1961, 69, 297-307.

Hugelin, A., Dumont, S., and Paillas, N. Tympanic muscles and control of auditory input during arousal. Science, 1960, 131, 1371-1372.

Jepsen, O. The threshold of the reflexes of the intratympanic muscles in normal material examined by means of the impedance method. Acta otolaryngol., 1951, 39, 406-408.

Jepsen, O. Studies on the acoustic stapedius reflex in man. Ph. D. Thesis, Univer. Aarhus, Aarhus, Denmark, 1955. Cited by Klockhoff (1961).

Kato, T. Zur Physiologie der Binnenmuskeln des Ohres, Pflüg. Arch. ges. Physiol., 1913, 150, 569-625.

Klockhoff, I. Middle ear muscle reflexes in man. Acta oto-laryngol., Suppl. 164, 1961.

Kobrak, H. G. The human ear. Chicago: Univer. Chicago Press, 1959.

Köhler, W. Akustische Untersuchungen. Zeitschr. Psychol., 1909, 54, 241-289.

Lawrence, M. Acoustic effects of middle ear substitution. Trans. Amer. Acad. Ophthalmol. Oto-laryngol., 1960, 64, 235-247.

Lawrence, M. The double innervation of the tensor tympani. In Fletcher, J. L. (ed.) Seminar on middle ear function. US Army Med. Res. Lab. Rep., Fort Knox, Ky., 1962.

Lawrence, M., Wolsk, D., and Schmidt, P. Inner-ear response to high-level sound. J. acoust. Soc. Amer., 1962, 34, 102-107.

Loeb, M. and Fletcher, J. L. Contralateral threshold shift and reduction in temporary threshold shift as indices of acoustic reflex action. J. acoust. Soc. Amer., 1961, 33, 1558-1560.

Loeb, M. and Fletcher, J. L. Reliability and temporal course of temporary and contralateral threshold shifts. J. Speech Hearing Res., 5, 284-291, 1962.

Loeb, M. and Riopelle, A. J. Influence of loud contralateral stimulation on the threshold and perceived loudness of low-frequency tones. J. acoust. Soc. Amer., 1960, 32, 602-610.

Lüscher, E. Die Funktion des Musculus stapedius beim Menschen. Zeitschr. Hals-Nasen-und Ohrenheilk., 1929, 23, 105-132.

Mendelson, E. S. Improved method for studying tympanic reflexes in man. J. acoust. Soc. Amer., 1961, 33, 146-152.

Mendelson, E. S. and Fletcher, J. L. Some indices of middle ear muscle activity. U.S.N. Air Material Center Air Crew Equipment Lab. Rep., Philadelphia, Pa., 1962, No. 473.

Metz, O. The acoustic impedance measured on normal and pathological ears. Acta oto-laryngol., Suppl. 63, 1946.

Metz, O. Studies on the contraction of the tympanic muscles as indicated by changes in the impedance of the ear. Acta oto-laryngol., 1951, 39, 397-405.

Möller, A. Bilateral contraction of the tympanic muscles in man. Roy. Instit. Technol., Speech Transmission Lab. Rep., Stockholm, 1961, No. 18.

Mousheigan, G., Rupert, A., Marsh, J. T., and Galambos, R. Evoked cortical potentials in the absence of middle ear muscles. Science, 1961, 133, 582-583.

Perlman, H. B. The place of the middle ear muscle reflex in auditory research. Arch. Otolaryngol., 1960, 72, 201-206.

Pichler, H. and Bornschein, H. Audiometrischer Nachweis nichtakustisch ausgelöster Reflexkontraktionen der Intra-auralmuskulatur. Acta oto-laryngol., 1957, 48, 498-503.

Pollak, J. Ueber die Function des Musculus tensor tympani. Medez. Jahrb., 1886, 82, 555-582.

Prather, W. F. Shifts in loudness of pure tones associated with contralateral noise stimulation. J. Speech Hearing Res., 1961, 4, 182-193.

Reger, S. N. Effects of middle ear muscle action on certain psychophysical measurements. Ann. Otol. Rhinol. Laryngol., 1960, 69, 1-20.

Ruben, R. J., Bordley, J. E., and Lieberman, A. T. Cochlear potentials in the human being. J. acoust. Soc. Amer., 1961, 33, 830.

Shapley, J. L. Reduction in the loudness of a 250-cycle tone in one ear following the introduction of a thermal noise in the opposite ear. Proc. Ia. Acad. Sci., 1954, 61, 417-422.

Simmons, F. B. Middle ear muscle activity at moderate sound levels. Ann. Otol. Rhinol. Laryngol., 1959, 68, 1-18.

Simmons, F. B. Middle ear protection from the acoustic trauma of loud continuous sound. An electrophysiological study in cats. Ann. Otol. Rhinol. Laryngol., 1960, 69, 1-9 (a).

Simmons, F. B. Post-tetanic potentiation in the middle-ear muscle acoustic reflex. J. acoust. Soc. Amer., 1960, 32, 1589-1591 (b).

Simmons, F. B., Galambos, R., and Rupert, A. Conditioned response of middle ear muscles. Amer. J. Physiol., 1959, 197, 537-538.

Smith, H. D. Audiometric effects of voluntary contraction of the tensor tympani muscle. Arch. Oto-laryngol., 1943, 38, 369-372.

Terkildsen, K. Acoustic reflexes of the human musculus tensor tympani. Acta oto-laryngol., 1960, Suppl. 158, 230-238.

Tsukamato, H. Zur Physiologie der Binnenohrmuskelnn. Zeitschr. Biol., 1934, 95, 146-154.

Waar, A. C. Mikoskopishce Wahrnehmungen der Funktion der Mittleohrmuskein beim Menschen. Acta oto-laryngol., 1923, 5, 335-358.

Ward, W. D. Studies on the aural reflex. I. Contralateral remote masking as an indicator of reflex activity. J. acoust. Soc. Amer., 1961, 33, 1034-1045.

Ward, W. D. Studies on the aural reflex. II. Reduction of temporary threshold shift from intermittent noise by reflex activity; implications for damage-risk criteria. J. acoust. Soc. Amer., 1962, 34, 234-241.

Ward, W. D., Selters, W., and Glorig, A. Exploratory studies on temporary threshold shift from impulses. J. acoust. Soc. Amer., 1961, 33, 781-793.

Ward, W. D. and Fleer, R. E. Temporal conditioning of the auditory reflex. Amer. Psychologist, 1961, 16, 441.

Weiss, H. The intra-aural muscle reflex in man. In Fletcher, J. L. (ed.) Seminar on middle ear function. US Army Med. Res. Lab. Rep., Fort Knox, Ky., 1962.

Wersäll, R. The tympanic muscles and their reflexes. Acta Otolaryngol., Suppl. 139, 1958.

Wever, E. G. and Lawrence, M. Physiological acoustics. Princeton, N.J.: Princeton Univer. Press, 1954.

Wever, E. G. Hearing. Ann. Rev. Psychol., 1962, 13, 225-250.

Wever, E. G. and Bray, C. W. The stapedius muscle in relation to sound conduction. J. exp. Psychol., 1942, 31, 35-43.

Wever, E. G. and Vernon, J. A. The control of sound transmission by the middle ear muscles. Ann. Otol. Rhinol. Laryngol., 1956, 65, 5-14.

Wiggers, H. C. The functions of the intra-aural muscles. Amer. J. Physiol., 1937, 120, 771-780.

Zwislocki, J. Acoustic measurement of the middle ear function. Ann. Otol. Rhinol. Laryngol., 1961, 70, 1-8.

CHANGES IN AIR CONDUCTION AND BONE CONDUCTION SENSITIVITY ASSOCIATED WITH VOLUNTARY CONTRACTION OF MIDDLE EAR MUSCULATURE

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In a historical review, Wever and Lawrence (12) reported that Fabricius ab Aquapendente stated in 1600 that he could voluntarily contract his middle ear muscles. It was not until 1943 that quantitative measurements of the transmission loss through the middle ear resulting from voluntary contraction of the intratympanic muscles were presented by Smith (10) on one subject. Figure 1 shows Smith's measurements. His data indicate that voluntary contraction results in significantly greater attenuation of frequencies below 2000 cps than for higher frequencies. It is worthy of note that the subject reported the 2000 cps tone was increased slightly in loudness during contraction, although the audiometric measurements obtained with a 5 db intensity step attenuator did not confirm this subjective impression.

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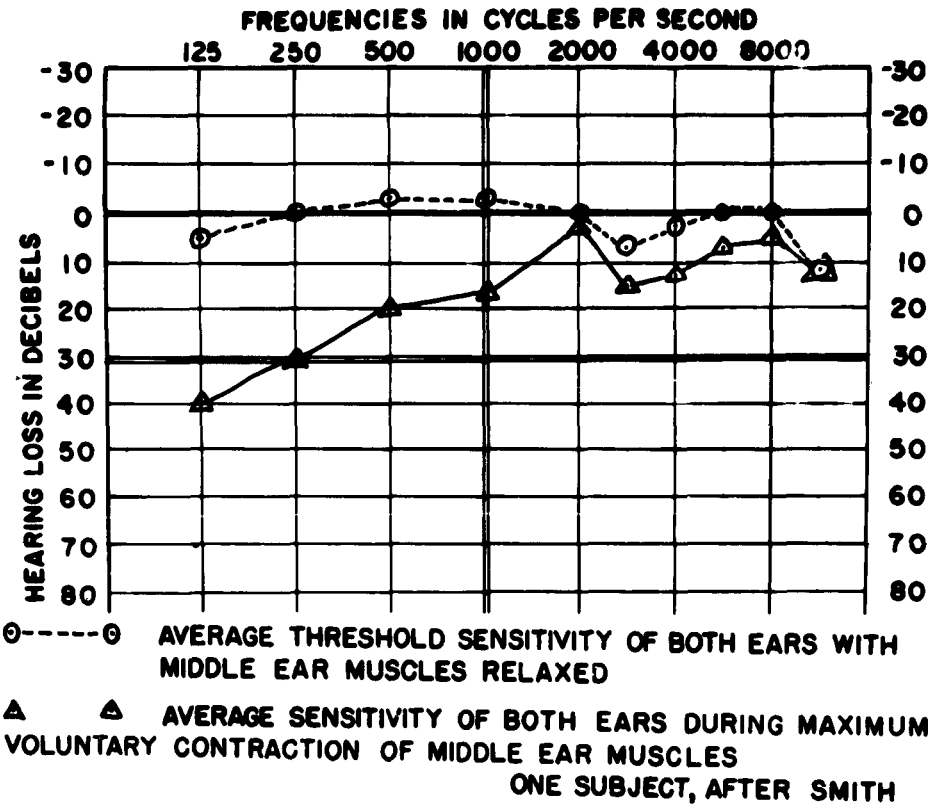

Figure 1

Figure 2 reproduces a graph, published by one of the writers (8) in 1960, which shows the average attenuation of air conducted sound of the eight ears of four subjects during maximum voluntary contraction of their intratympanic muscles. Each of these subjects was able to sustain voluntary contraction for 20 to 30 seconds before loss of control, at which time renewed effort reestablished the effectiveness of the contraction. Two of the subjects reported a noticeable increase in loudness of the 2000 cps tone and one a slight increase in loudness of the 4000 cps tone during maximum contraction.

The following discussion presents additional data on the transmission loss for both air conducted and bone conducted sound associated with voluntary contraction of the intratympanic muscles of three well trained, highly motivated subjects, who are the joint authors of the present paper. Thresholds were obtained before and during

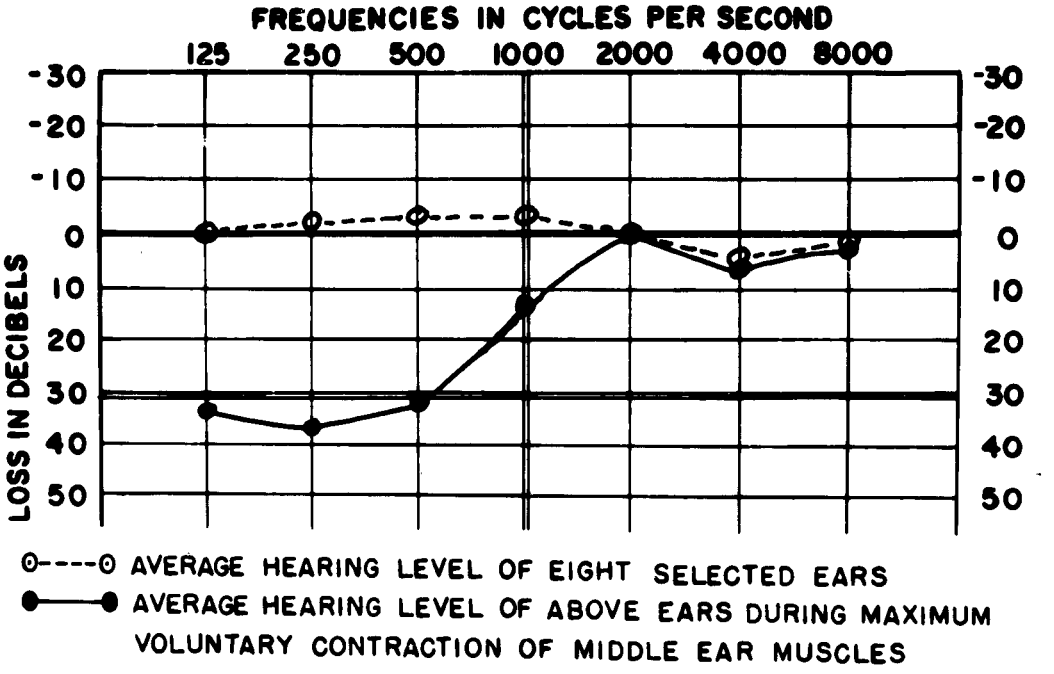

Figure 2

contraction by means of conventional 5 db step audiometry, Békésy audiometry and by the method of adjustment by the listener with a 1 db step attenuator. Each subject had a negative ear history, a negative ear, nose and throat examination and possessed auditory sensitivity within normal variation limits.

Figure 3 shows the mean sensitivity for the air conduction measurements of the six ears of the three subjects as measured by the three different testing techniques. In this and the following figures the threshold sensitivity curves of the subjects before contraction are superimposed on the zero hearing level reference line; the changes resulting from contraction are recorded with the db differences adjusted relative to that line. The small circles above and below the measurements resulting from contraction indicate the extreme plus and minus ranges about the associated means.

The mean data of Figures 1, 2 and 3 agree reasonably closely. It must be emphasized that all the subjects used in these three illustrations were selected because of their exceptional ability to produce relatively large transmission losses by contraction of their intratympanic

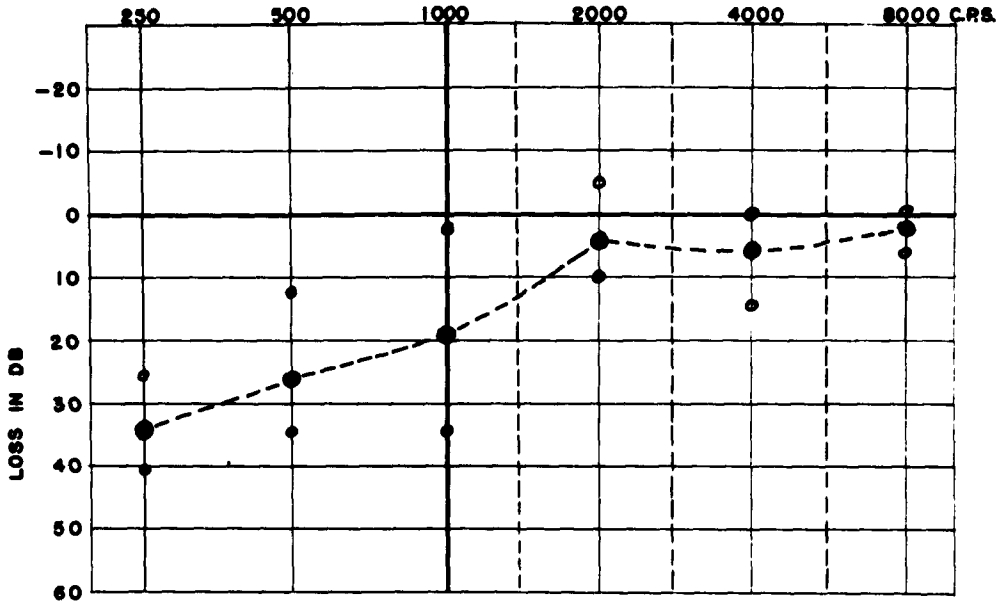

Fig. 3. Mean transmission losses of six ears of three subjects for air conducted pure tones during voluntary contraction of their middle ear muscles. 0 = extreme intensity ranges.

musculature for relatively long periods of time. Therefore, these data fall at the end of the normal distribution curve which shows maximum values and are not representative of the mean transmission loss which results from contraction of these muscles. As the result of considerable measurement experience by one of the writers with individuals who can voluntarily contract their intratympanic muscles, it appears that individual differences in the magnitudes of transmission losses and the durations of contractions show as much variability as individual differences in susceptibility to noise exposure.

Figure 4 shows the results of the bone conduction (BC) measurements. The BC vibrator was mounted in a special headband which held the vibrator with a force of approximately 520 grams against the midline of the forehead. No attempt was made to obtain BC sensitivity in the separate ears and masking was not employed while making the measurements.

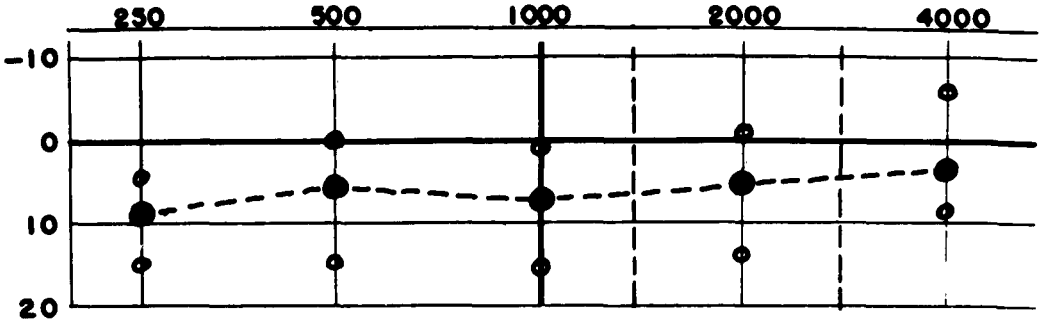

Fig. 4. Mean transmission losses of three subjects for bone conducted pure tones during voluntary contraction of their middle ear muscles with open external auditory canals. 0 = extreme intensity ranges.

DISCUSSION OF RESULTS

The air conduction transmission losses shown in Figures 1, 2 and 3 indicate somewhat greater attenuation for the lower frequencies than has been observed in animal experimentation. A loss of about 20 db is the maximum reported by animal investigators, with the exception of Wiggers (13), who found losses as great as 40 db for low frequencies resulting from non-acoustically activated spontaneous contractions in the guinea pig. However, there appear to be significant differences in middle ear muscle action between man and animals. Acoustic stimuli elicit contraction of both intratympanic muscles in the commonly used experimental animals. It is worthy of note that Kato (3) was unable to observe any definite tensor tympani contractions as a result of acoustic stimulation in monkeys. Recent work on man, particularly by Jepsen (2), Pichler and Bornschein (7), Klockhoff and Anderson (4), Klockhoff (5), Møller (6) and Djupesland (1) indicates it is highly probable that only the stapedius muscle in man contracts in response to acoustic stimuli. However, it must be noted that Terkildsen (11), using a sensitive manometer to measure slight variations in pressure in the external auditory canal, reported that muscle reflexes could be elicited by acoustic stimuli in 95 per cent of a group of normal ears; "both tensor and stapedius reflexes were found in 40 per cent, while in 33 per cent and 22 per cent respectively only stapedius or tensor reflexes were evident."

Most animal investigators have reported that the stapedius is more effective than the tensor in attenuating sound conduction across the tympanum. Simmons (9), working with cats in which electrodes have been implanted near their round windows, reported that: "... most of the dynamic action of the middle ear muscles in response to moderate sound can be credited to the stapedius."

The writer is not aware of any research which describes the relative roles of the intratympanic muscles in man during voluntary contraction. It is not known at present if only the tensor tympani or both muscles react to such contraction. A certain introspective observation supports the conclusion that at least the tensor is involved in voluntary contraction. While listening to low frequency pure tones or low frequency narrow band noise at a 110 db SPL, the subjects were requested to alternately contract and relax their middle ear muscles. Contraction invariably resulted in a marked decrease in loudness. It is assumed that such low frequency stimuli at the 110 db SPL are adequate to elicit the reflex, which, as noted above seems to involve the stapedius in man. The reduction in loudness accompanying the voluntary contraction may be due in part to reinforcement of the tension of the stapedius exerted by the contraction; however, it appears equally plausible that the reduction in loudness results in part or in toto from the voluntary contraction of the tensor.

Figure 4 shows that the attenuation for BC sound is considerably less and also more uniform over the indicated frequency range than the attenuation of air conducted sound.

INTROSPECTIVE OBSERVATIONS

As reported previously by one of the writers (8), the percentage of persons who can contract their middle ear muscles voluntarily is conspicuously low. "From the 120 students in the Junior Medical Class at the State University of Iowa, only one or two are found each year who are able to contract their middle ear muscles voluntarily, but usually in limited degree." However, an appreciable number of individuals can develop this ability by guided practice. Several subjects first noticed the noise of muscle tremors and the attenuation of ambient sounds while yawning. Those who in addition also experienced some sort of kinaesthetic sensation in their ears while yawning found it relatively easy to learn to contract their middle ear musculature at will. Many persons who state they cannot contract their middle ear muscles voluntarily can be taught to do so in the following manner: expose the subject to a 2000 pulse of 1 second duration at a 100 db

SPL. At the termination of the pulse middle ear muscle tremor noises usually can be heard as the muscle relaxes. If a kinaesthetic sensation also is experienced, transfer to voluntary control often becomes possible. An air jet directed into the external auditory canal or the eye, or a tactile sensation, such as a mild electric shock in the external canal, also usually causes the tensor tympani to contract. If the subject experiences middle ear muscle tremor noises or kinaesthetic sensations during contraction or relaxation of the muscles, voluntary control may be facilitated.

Extreme individual variations exist in the durations of the contractions and in the quantitative values of the transmission losses related thereto. One of the three subjects in the present paper could maintain contraction for only 20 seconds, one for about a minute and a half and one for about three and a half minutes. The subject who could maintain contraction for the 20 seconds experienced the minimum air conduction threshold transmission loss and the one who sustained contraction for three and a half minutes experienced the maximum air conduction threshold transmission loss. These relationships may have been coincidental. Some subjects can maintain contraction over considerably greater intervals by periodically renewing the contracture effort before complete relaxation occurs following an initial effort.

Individuals who can voluntarily contract their middle ear muscles are aware of "fluttering" or "trembling" muscle tremor noises during the contraction, except while in noise levels sufficiently intense to mask the tremor noises. In an earlier study one of the writers (8) "...found that the sound produced by a 20 cps tone reproduced in an earphone approximated the sound resulting from intra-tympanic muscle tremors when this tone was presented at the 18 db sensation level" in four subjects. This 20 cps tone was not reproduced by the earphone as a pure tone but as a complex tone with numerous harmonics. It appears unlikely that middle ear muscle tremor noise is sufficiently high in intensity to produce sufficient masking to account for the magnitudes of the air conduction transmission losses reported in Figures 1, 2 and 3.

How can an experimenter be certain that his subjects can voluntarily contract their middle ear muscles? The following subjective tests are of value: (1) present to the ears a 110 db SPL wide band thermal noise or low frequency pure tone and request the subject to contract and relax his intratympanic musculature. If he reports a marked reduction in loudness during contraction, it can be assumed with reasonable certainty that he possesses the ability in question;

(2) present a 110 db SPL complex tone, having a 100 to 250 cps first partial which contains considerable audible energy. Effective contraction of the musculature in question alters the tone quality of the complex tone decisively; (3) obtain the subject's threshold sensitivity at 250 cps. Then present this tone at his 25 db SL. If the tone is attenuated to inaudibility during contraction, he has attained considerable competence in ability to contract his middle ear musculature. The muscle tremor noise is not sufficiently high in energy to mask out perception of a 25 db sensation level, 250 cps pure tone.

Use of an impedance bridge provides a more objective indication of ability to contract the intratympanic musculature. However, the bridge may show imbalances as the result of swallowing or opening of the Eustachian tube orifice in the nasopharynx, which sometimes is associated with the contraction.

It must be emphasized that the awareness of muscle tremor noise during what may be assumed to be intratympanic muscle contraction is not necessarily an indication of ability to contract the middle ear muscles. Certain individuals who cannot contract these muscles are able to exercise voluntary control over some of the musculature in proximity to the opening of the Eustachian tube in the nasopharynx, which also may result in audible tremor noises.

Many individuals can contract their middle ear muscles without extending the effort visibly to other musculatures. Some accompany such effort with visible contracture of certain muscles of the face and neck. Many individuals who can contract their middle ear muscles voluntarily can also exert a certain amount of control over other muscles which usually lie dormant in some persons. One subject has been studied who, in addition to contracting his middle ear muscles in either ear separately or in both simultaneously, could wiggle either auricle separately or both simultaneously. could contract the muscles around his nares on either side separately or both sides simultaneously, could contract certain scalp muscles on various areas of his head and could voluntarily open his Eustachian tubes. The opening of the tubes was accompanied by a "click" as the mucous covered surfaces separated. On the other hand, patients have been encountered who can voluntarily open their Eustachian tubes who cannot contract their middle ear muscles; and others who can voluntarily contract their middle ear muscles cannot wiggle their ears.

Tinnitus is rarely observed during or following voluntary contraction, except for the low frequency fluttering noise which is due to the

muscle tremors of the contracted stapedius and tensor. No dizziness or nystagmus has been observed to date.

Pitch changes of sustained pure tones are occasionally noticed during voluntary contraction. One of the co-authors, while exposed to a 100 db SPL, 250 cps pure tone, reported during voluntary contraction a change in quality so extensive it was difficult to ascribe any pitch to the sound. Other subjects have noted sufficient change in quality of low frequencies during contraction to make it difficult to determine if they experienced a change in pitch. Several reported that the voices of others appear to exhibit markedly reduced bass response for speech, which is most readily noticed in the male voice.

Some observers report a subjective impression of hearing frequencies above 1000 cps with increased loudness during voluntary contraction. Audiometric measurements rarely confirm this observation. Others never experience a loudness increase for any frequency at any sensation level while contracting.

Admittedly, the above introspections have been obtained from a limited sample which exhibits a phenomenon in which large individual variations are the rule rather than the exception. It is intended that this entire paper be regarded as a preliminary early study of a process which is little understood at the present time. The authors also regard their statements and conclusions as tentative, and subject to revision or modification as more data become available. Critics please note.

BIBLIOGRAPHY

1. Djupesland, G. Intra-aural muscular reflexes elicited by air current stimulation of the external ear. Acta Oto-Laryngol., 1962, 54, 143-153.

2. Jepsen, O. Studies on the acoustic stapedius reflex in man. Thesis at the University of Aarhus, Denmark, Universitets - forlaget i Aarhus, Aarhus 1955.

3. Kato, T. Zur Physiologie der Binnenmuskeln des Ohres. Pflügers Arch. ges. Physiol., 1913, 150: 569-625.

4. Klockhoff, I. and Anderson, H. Reflex activity in the tensor tympani muscle recorded in man. Preliminary Report. Acta Oto-Laryngol., 1960, 51, 184-188.

5. Klockhoff, I. Middle ear reflexes in man. Acta Oto-Laryngol., 1961, Suppl. 164, 1-92.

6. Møller, A. R. Bilateral contraction of the tympanic muscles in man. Ann. Otol., Rhinol. and Laryngol., 1961, 70, 735-753.

7. Pichler, V. H. and Bornschein, H. Audiometric proof of non-acoustic reflex reactions of the intra-aural muscles. Acta Oto-Laryngol., 1957, 48, 498-503.

8. Reger, S. N. Effect of middle ear muscle action on certain psycho-physical measurements. Ann. Otol., Rhinol. and Laryngol., 1960, 69, 1179-1198.

9. Simmons, F. B. Middle ear muscle activity at moderate sound levels. Ann. Otol., Rhinol. and Laryngol., 1959, 68, 1126-1143

10. Smith, H. D. Audiometric effects of voluntary contraction of the tensor tympani muscle. Arch. Otolaryngol., 1943, 38, 369-372.

11. Terkildsen, K. Acoustic reflexes of the human musculus tensor tympani. Acta Oto-Laryngol., 1960, Suppl. 158, 230-236.

12. Wever, E. and Lawrence, M. Physiological Acoustics. Princeton: Princeton University Press, pp. 454, 1954.

13. Wiggers, H. C. The functions of the intra-aural muscles. Amer. J. Physiol., 1937, 120, 771-780.

CONTRALATERAL REMOTE MASKING

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This talk was originally supposed to be on contralateral remote masking. As it turns out it will be partly on this but only partly, because recent evidence has led me to believe that contralateral remote masking may not properly belong in this particular conference. The logic is inescapable that if reflex activation does indeed produce low frequency attenuation, if then you put a high-frequency tone or band of noise in one ear, you should be able to measure a change in the threshold of a low-frequency tone in the opposite ear. Von Békésy, of course, pointed this out many years ago, but nothing was done with it until Shapley wrote a thesis on it in 1954, which was followed by a study a little bit later by Loeb and Riopelle. I became interested in the phenomenon and ran a group of studies after a conference of the Subcommittee when Scott Reger was visiting us. Dr. Reger excited our interest with a brief account of Shapley's work. We, therefore, studied this "remote contralateral masking" in some detail.

Figure 1 illustrates the reasoning involved. The experimental procedure here was to determine the threshold change at 500 cps in the left ear under five different conditions: 300-600 cps noise in the same ear, 2400-4800 cps noise in the same ear, these noises in the opposite ear, or 3400 cps pure tone in the opposite ear. Clearly, the curve furthest to the left is ordinary masking--direct masking--because the noise included the critical band that is involved in perception of 500 cps. The dotted one illustrates the phenomenon of remote masking which the CID** researchers have shown to be due to low frequency distortion products: distortion products produced in the inner ear by the high-intensity, high-frequency band of noise.

In the case of 300-600 cps in the opposite ear, two plausible effects can be involved, perhaps even three. For one thing, eventually you get to the point where the sound leakage across the head (the transcranial attenuation in this case amounts to 60 db) becomes great enough

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that with a 10 db increase in intensity of the arousal noise there is a 10 db increase in threshold. So again, that is just ordinary masking. What happens at the lower levels of this curve, though, is a little more complicated: there must be either (a) some reflex arousal (which does not seem very reasonable) or (b) central masking. Now, central masking is a subject that has not received very much attention, but it is quite evident that it is present here. If it is true that the separation of these two curves implies that there is a 60 db attenuation between the two ears, then when one has a 60 db 300-600 cps noise, if one calculates what the level of the critical band is and subtracts 60 db, this transcranially conducted sound is so low that it should not affect the threshold in this ear at all by means of any direct physical masking. Therefore, we must postulate that some channels in the higher centers that are responsible for threshold response to 500 cycles in one ear are being preempted by this 60 db band of noise and that, therefore, the threshold is raised slightly. This phenomenon was noted by Ingham somewhat earlier.

Finally, what happens then when we put 2400-4800 cps noise in the right (contralateral) ear? This result is the one of major interest because we are hoping to be able to factor out all other causes of threshold shift and come out with a number that indicates the decibels of attenuation of the 500 cycle tone that is due to the arousal of the acoustic reflex. I have finally realized that I really do not have enough points. We must measure ipsilateral remote masking up to 130 db if we are to determine what the reflex arousal attenuation will be at 130 db. This is necessary because the amount of ipsilateral remote masking indicates the effective level of the low-frequency distortion products, and the effective level of these distortion products will determine the degree of central masking in the other ear which must be subtracted from the observed contralateral remote masking in order to obtain the attenuation due to the reflex.

Let me go through the complete argument for 110 db. At 110 db on the curve marked "2400-2800 cps in LE" there are distortion products to produce a masking of about 46 db of a 500 cycle tone in the same ear. If we next ask how much low-frequency noise is needed to produce this same masking, the "300-600 cps in LE" curve tells us that this is about 70 db. This means that the effective level of these low-frequency distortion products (effective for producing central masking), is about 70 db. So, dropping down to the "300-600 cps in RE" curve, we see that the 70 db low-frequency noise produces central masking to a stimulus in the other ear of about 4 db, and this is the amount that we have to correct the 100 db point on the "2400-4800 cps in RE curve" in

order to determine how much of the observed contralateral remote masking (13 db here) is due to the reflex arousal. Therefore, the highest point in which we can place any confidence in this particular display is that for 110 db of 2400-4800 cps noise in the contralateral ear: this, I have some assurance, produces about 9 db of attenuation at 500 cps due to the reflex. Unfortunately, ipsilateral remote masking was not measured at 120 and 130 db, so it is not possible at the moment to go through the gymnastics to determine the reflex attenuation produced by these levels. Therefore, the curves presented in my previous article are not, as I implied in the article, reflex attenuation alone but are rather a sum of reflex attenuation plus some undetermined amount of central masking. More research must be done to define central masking phenomena.

Therefore, let me leave the topic of contralateral remote masking and describe some experiments we did to answer a question of some practical importance: What are the optimum characteristics of a reflex-arousal tone if it is to be used around gun emplacements?

There are two opposing considerations that are involved. The reflex arousal stimulus should be as intense as possible and it should precede the impulse long enough and last long enough that everybody's reflexes are aroused and have not yet started to relax when the impulse arrives. On the other hand, you do not want to make it so long or intense that you produce more temporary threshold shift (TTS) with your arousal stimulus than you are preventing.

Some aspects of this situation are fairly well understood now. In regard to the spectral characteristics, there is apparently complete latitude. The most recent evidence is that the strength of the short-term arousal of the reflex is constant for a given sensation level (or perhaps loudness) regardless of the frequency, and we can probably therefore use almost <u>any</u> pure tone or noise band, or even a klaxon or other horn (which is usually easier to obtain). The TTS probably is not much of a problem if you are dealing with ordinary rates of fire because of the fact that TTS from a noise that occurs in short bursts is proportional to the on-fraction. Therefore, if the arousal stimulus is on only for 1/100 of the time you only get 1/100 of the TTS that you would if the noise of the arousal stimulus were on all the time.

More uncertainty, however, regards the latency that should be used. It is clear from Møller's records that the temporal course of the change of impedance associated with short tone pulses varies greatly from person to person. Although latency of the first action

potentials in the stapedius is known to be on the order of 10 msec, appreciable changes in the acoustic impedance are not manifested until after 30 to 40 msec. The maximum change is not reached in some subjects for nearly 300 msec. Metz has shown that the original balance is not restored until about 1 second after cessation of the arousal stimulus. The cutaneous stapedius reflex study by Klockhoff shows even longer latency. In a group of 10 subjects, the time from onset of the shock stimulus applied to the ear canal to the first reflex contraction (presumably this represented only the action of the stapedius) varied from 120 to 190 msec. He attributes this increase in latency to the need for substantial temporal summation of the afferent impulses from the skin in order to initiate the muscle action. More germane here is the fact that some 150 msec elapsed after onset of reflex action before the maximum was reached.

Now, on the other side of the coin, Terkildsen reports that in some cases where he believes the observed impedance change represents only stapedius action, the latency of the arousal was as short as he could measure--about 10 msec. However, he does not actually show records, so we cannot judge whether this is an initial twitch or the beginning of a later rise in impedance. He points out that the tensor tympani has a much longer latency (on this all studies agree). Klockhoff's theory is that the stapedius by its contraction induces a reflex contraction of the tensor tympani and that is its normal mode of reaction. I have not heard that mentioned here yet this morning but I think it is worth discussing later.

If it is true, therefore, and it certainly seems likely from animal experiments, that the stapedius is primarily responsible for the protection produced by the acoustic reflex, perhaps an arousal-to-impulse interval somewhat shorter than that implied by the impedance measurements would be better. Terkildsen argues as follows: "The rather long latencies found with measurements of the impedance are then best explained through the assumption that a real fixation of the whole ossicular chain does not take place until both muscles are in action. As the tensor muscle is the slower of the two, such measurements actually record the latency of the muscle, which for protective purposes is of minor importance." I suspect that there is some disagreement with this point of view among some of the members of this seminar. But at any rate the optimum interval for spacing between the arousal stimulus and the impulse is not at all obvious, so the best way to do this is to check, that is, actually put in an arousal stimulus at various times before the arrival of the impulse and see what sort of effective protection is provided. One could also, of course, record the changes in

impedance associated with different latencies. That would be a much easier method, but I doubt that all of us here are convinced that the instantaneous acoustic impedance is a completely isomorphoric indication of the effective protection that exists at that moment. Therefore, our experiment was set up.

A 250-msec, 100 db SPL 1000 cps tone was used throughout. This was presented to one ear of a listener at four different delay times: zero (actually, no arousal tone at all), 25 msec delay, 6 msec, and 100 msec preceding the arrival of a pulse that was generated by a high intensity speaker that can generate pulses of peak levels up to about 155 db. The procedure, grossly, was as follows. First, an exposure of about 1 min to clicks at a rate of one every 2.4 sec was given. We used this particular interval in order to guarantee that there was no residual reflex arousal (from one pulse) still present when the next one arrived, in the no-deliberate-arousal-stimulus condition. After a minute of exposure to this one level we measured the threshold at 4,000 cps (interrupted tone), and if the TTS at about 1/2 minute of recovery exceeded 20 db an audiogram was made. If, however, less than 20 db of TTS was present, the ear was exposed for 1 min at the next higher pulse level. This means one obtains some sort of cumulative TTS measure. One measures the TTS after 135 db clicks then after a minute of testing the subject is given another minute of exposure to the next higher level clicks--138 or 138-1/2 db (the steps are about 3-1/2 db apart), etc--and one gets a curve that eventually goes up to 20 db. Now, when the reflex arousal stimulus is being given, if there is a shift of this whole curve to the right, the implication is that you have gotten an amount of protection equal to the separation of the curves. I must also mention that in order to measure the amount of the effective attenuation of course you must use people with less resistant ears, because if you have someone who shows no TTS after exposure to the highest intensity when no reflex arousal stimulus is present, obviously you cannot demonstrate any effective protection. About one-half of the subjects that we tested were too tough-eared for us to use so this experiment was done on the most susceptible 50 per cent of the people.

Some of the results are shown in Figure 2. This shows 4 of the 10 observers; it should be clear why an "average" curve is not attempted. Listener MK's right ear displays the simplicity of results I was hoping we would get for all listeners. Without reflex-arousal stimulus (short-dashed line) he begins showing TTS almost immediately--at least after the second exposure. Then when the onset of the arousal tones preceded the pulse by 25 msec, the growth of TTS was unchanged (solid line). Similarly, a 62-msec gave no change. But for 100 msec

delay the curve has been shifted about 13 db to the right. Similarly, MK's other ear shows about 10 db shift at 100 msec, none at lower delays. Apparently, he does not get any protection from a reflex arousal tone if the delay is less than 100 msec. He is, therefore, a fairly slow responder.

The fastest responder I had was KA, who showed some reduction of TTS even for the 25 msec delay. He was the only one who showed more than about a 3 db difference between the curves for 25 msec delay and the no-arousal-tone condition, and this may not actually represent anything more than day-to-day variability.

EW's results are more ambiguous. With delays of 62 and 100 msec, a plateau is apparently reached. YT's results are the most extreme in this regard. At some point his left ear makes an abrupt transition from no TTS to about 20 and then there is no further increase as the level is raised even higher.

Figure 3 shows results from three other subjects. JM showed very little effect up to 100 msec delay and therefore was given 150. The large effective attenuation finally produced indicates that she (this was the only girl in the panel) has a very slowly-responding reflex. (I don't think this is a sex-linked characteristic.) On the other hand, subject HM shows no additional change as the delay increases from 100 to 150 msec, which implies that he simply has weak muscles. They are responding all they can as soon as the delay reaches 100 msec but their best is not much.

VA's right ear is the most unusual of all. Notice that not only does the TTS rise swiftly, but that he shows much more TTS at the lowest intensity used than all the other ears even relative to his other ear itself. Further, increasing the reflex arousal tone latency all the way up to 150 msec did not result in the slightest change in the TTS. The obvious implication is that he has an inoperative stapedius reflex. We later were able to compare the impedance changes produced in the two ears by contralateral stimulation and indeed there was a great difference. There were no recordable changes from the right ear when the arousal noise was presented to the left but large shifts in impedance when the noise was in the right ear and measurements were made on the left ear. Fortunately, VA is one of my student assistants, so he has been used in this sort of study for about 3 years. Therefore, I went back and looked at the records that he gave before, particularly on contralateral remote masking. His right ear nearly always showed less contralateral remote masking than the others, but it still showed

a gradual change with time that I assumed earlier to indicate a relaxation of reflex activity. If indeed the reflex is in this case inoperative, this means that part of the gradual change with time of contralateral remote masking is ascribable to a change in time of central masking. At the moment, therefore, I am willing to entertain the hypothesis that what we have been doing in our studies on contralateral remote masking has been merely measuring prestimulatory fatigue by a perhaps crafty but very roundabout method.

At any rate, you can see that it would be difficult to get an average of all these 10 subjects. Three of them are not on here, but they don't differ from some of the others. One can, however, calculate medians. These median values of effective protection came out to be 0.5 db for the 25 msec delay, 5 db for 62 msec delay, 13 db for 100 msec. Of course, one cannot obtain a dependable median value for 150 msec delay because only a few ears were given this long latency. However, these results definitely do imply that it takes a long time for full reflex protective action to develop, and so any aspect of reflex arousal that is over in a very short time is not by itself sufficient to produce the protection. This implies perhaps that the tensor tympani may have more effect than has previously been thought.

At any rate, the upshot is that a reflex arousal tone should precede gunfire by 150 msec because this much time is needed for the slowest people to develop full reflex action. By coincidence this is just about the value the Fort Knox investigators have been using in their experiments. I should say that you need at least 150; maybe 200 would be better.

At least, this particular study answers one empirical question, and I think it does so rather unambiguously. I think that it is better that I presented these results here than a series of confusing, challenging, and ambiguous results using contralateral remote masking.

I have made a list of the questions* that apparently are the issues involved in contemporary middle ear reflexology, and perhaps I can present them later. So far, there have been partial answers to some of the questions that have come up here. Usually, they have not been specifically stated but I am sure that we are making headway.

The experimental method used in the present study is a very cumbersone way, though, to measure the reflex latency--the latency

*See Appendix.

of effective protection. Since these results do agree fairly well with the temporal delay that is implied by Møller's results, I would suggest at this point that what we should do is use impedance-bridge measurements to establish the point at which one obtains maximum impedance; then perhaps, as a last validation, do a study along the present lines to make sure that you indeed do have the most protection when the ear presents the maximum impedance to any incoming signal.

Figures 4 and 5 show the results of a study, just completed, having to do with the effect of spacing of impulses. As some of you may know, several of us got together at Aberdeen a few months ago as a CHABA* working group to discuss TTS from impulses and damage risk criteria for them. We decided at this time to do the same experiment in various laboratories around the country. Everybody would use an interpulse interval of 1, 3, and 9 seconds in order to find out what sort of a correction had to be made for variability of interpulse intervals. As Dr. Loeb pointed out today the Ward, Selters, and Glorig results imply that perhaps the total number of impulses determines how much TTS is produced, and the spacing is a less important factor. In our case we have pulses generated by an Altec 20801 speaker; other laboratories will use different pulses, including machine gunfire.

The exposure in this study was always just 60 pulses. For each observer, I first found the click level that would just give about 20 db or a little more of TTS 1 min after exposure for 2 minutes to these clicks at 24 clicks per minute, and this level was used for the entire experiment. These 60 clicks could be delivered either once a second, once every 3 seconds, once every 9 seconds, or finally once every 30 seconds. Inclusion of the 30-sec interpulse interval was almost mandatory after it became evident, as you can see from Figure 4, that the TTS at 4,000 cycles did not change as the total time exposure increased from 1 minute to 9 minutes. Obviously, there had to be some point at which the TTS began to decline, as the interval became longer and longer. This finally happened for the 30-sec interval (the total exposure in this case is 30 minutes because 60 clicks were delivered in each condition). This shows that when pulses are 30 seconds apart there is an appreciable recovery between them, but that this is not true when they are 10 seconds or less apart.

Finally, Figure 5 shows the terminal audiograms, not just at 4,000 cps but at many different frequencies. This is the TTS at about

*Armed Forces-National Research Council Committee on Hearing and Bio-Acoustics.

5 minutes recovery. I cannot explain the secondary peak at 10,000 cps. This figure illustrates the close similarity of results even at the 30-sec interpulse interval condition--only at 3, 4, and 6 kc are the 30-sec interval shifts smaller than all three of the other conditions.

As far as our topic today is concerned, the fact that the 1-sec interval did not give less TTS than the 3-sec interval indicates that there was no residual action of the middle ear muscles due to the preceding pulse. This implies that full relaxation occurs within 1 sec after cessation of the arousal stimulus, as Metz showed by a different method.

I have tried, then, to make three main points:

(1) Measurement of contralateral remote masking is not sufficient to give an accurate measure of the effective attenuation produced by the reflexes, since an unknown amount of central masking by low-frequency distortion products of the high-frequency noise is involved.

(2) The time required for the muscles to reach full contraction varies from person to person, some individuals requiring at least 150 msec. Therefore, a reflex-arousal tone designed to reduce hearing loss from gunfire should precede the gunshot by 150 to 200 msec.

(3) The time required for complete relaxation of the reflexes is about 1 sec; no residual protection can be expected after this time.

APPENDIX

1. What is the adequate stimulus for each muscle?

2. What are the exact relations among the various measures from which we infer the strength of reflex arousal: impedance change, drum movement, and effective attenuation?

3. How does the time course of reflex activity vary with arousal level, duration, and spectral composition?

4. What causes the "adaptation" or "relaxation" in the presence of a continued arousal stimulus? (That is, is this relaxation correlated with the growth of prestimulatory fatigue?)

5. Is the reflex threshold more nearly constant with frequency for equal SPL, SL, or loudness?

6. Do low-frequency sounds produce more activation (at supra-reflex-threshold levels) than high-frequency sounds?

7. With activation of the muscles, how is the transmission of sound altered as a function of frequency?

8. Is the ipsilateral contraction really greater in strength than the contralateral contraction?

9. Is there a difference in latency between ipsilateral and contralateral contraction?

10. In voluntary contraction of the muscles, how much of the observed threshold shifts are indicative of additional attenuation, and how much can be ascribed to masking by the low-frequency noise generally reported to accompany such voluntary contraction?

11. Is the effect of middle ear contraction a simple attenuation, or instead is there peak- or center-clipping of the signal?

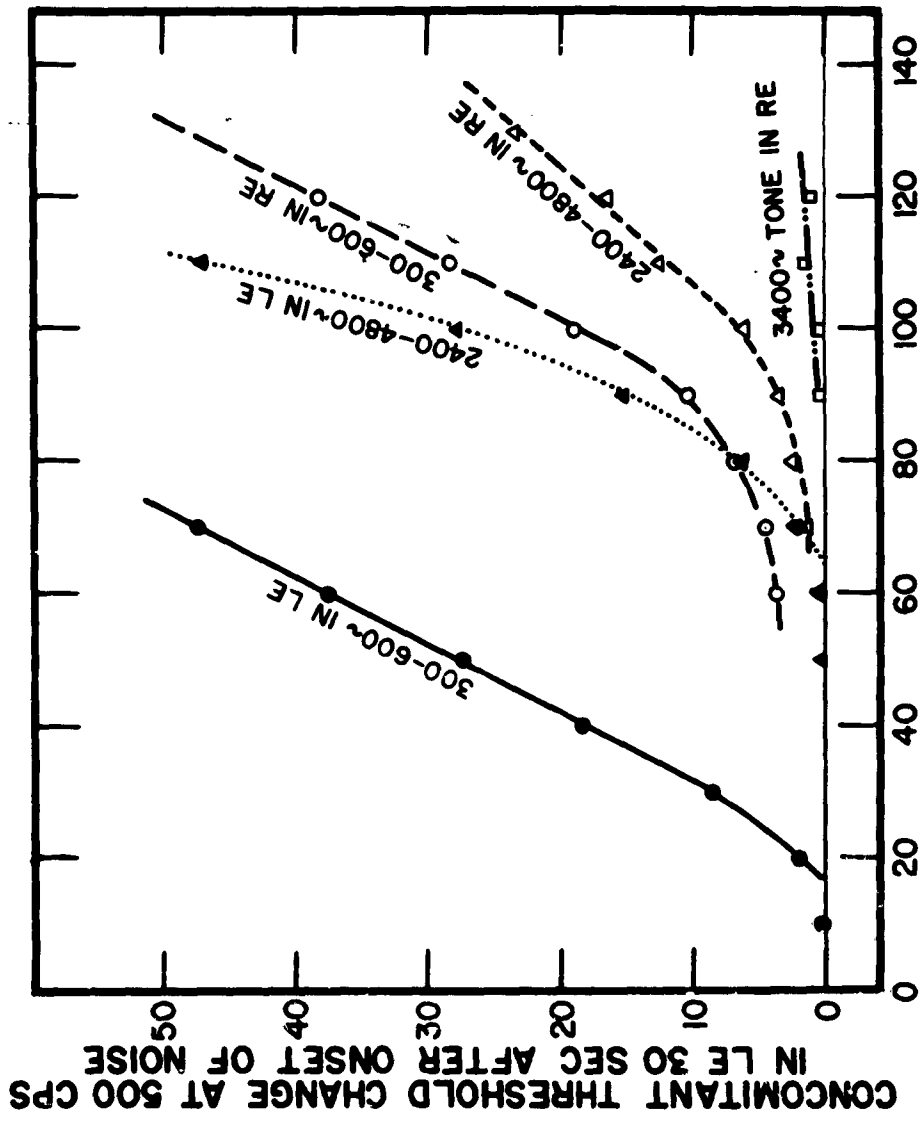

Fig. 1. Masking of a 500 cps tone by high and low frequency bands of noise in the same and in the opposite ear.

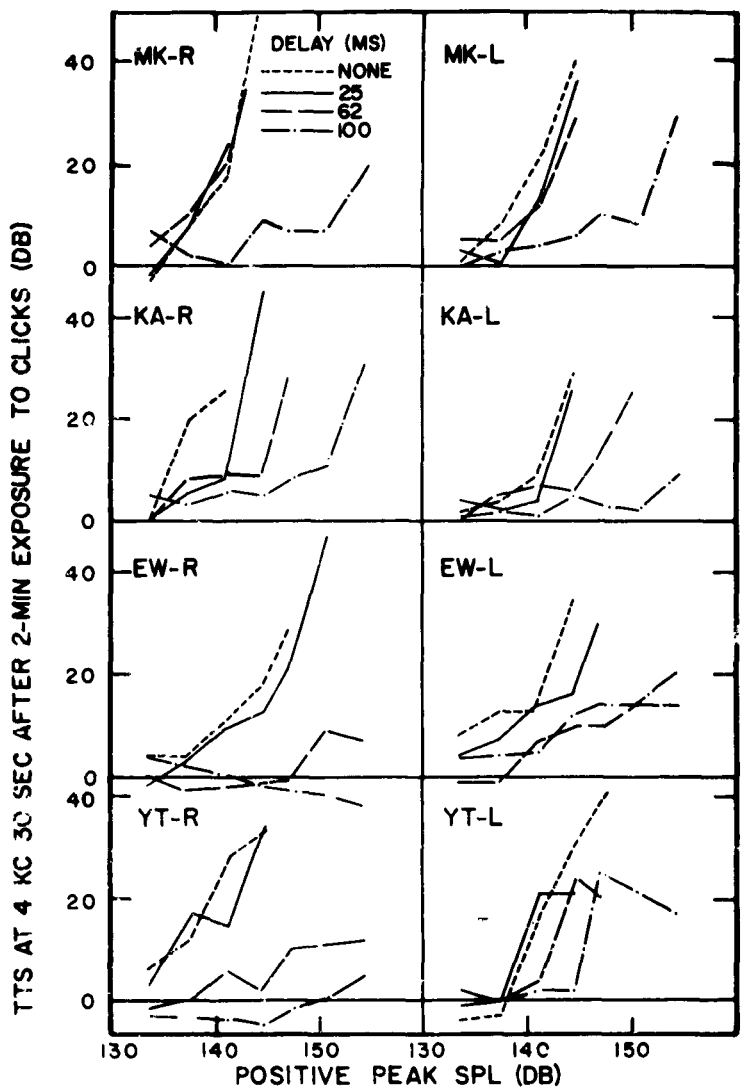

Fig. 2. Typical examples of the growth of $TTS_{0.5}$ at 4 kc as the level of successive 2 min exposures to pulses at 25 pulses per min is raised from 133 db SPL to a level sufficient to produce 20 db or more of TTS. The short-dashed line shows the TTS produced when no reflex-arousal tone is present. The other curves illustrate the changes that occur when a 1000 cps arousal tone at 100 db SPL is presented to the contralateral ear. The parameter is the delay, in msec, between onset of the arousal tone and presentation of the pulse.

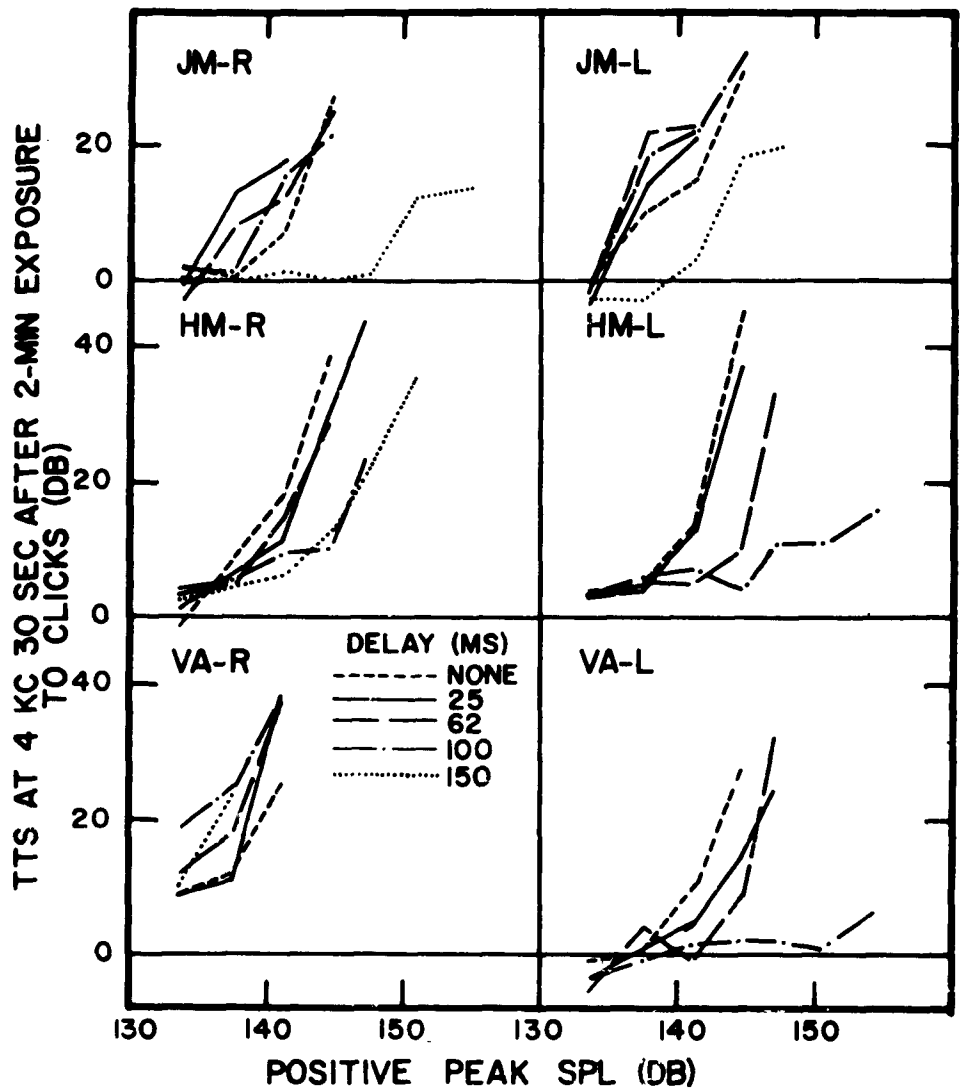

Fig. 3. Growth of $TTS_{0.5}$ at 4 kc in 3 observers who showed little protective effect of the reflex at delays of 100 msec and less between arousal tone and impulse, and who were therefore tested with a 150-msec delay.

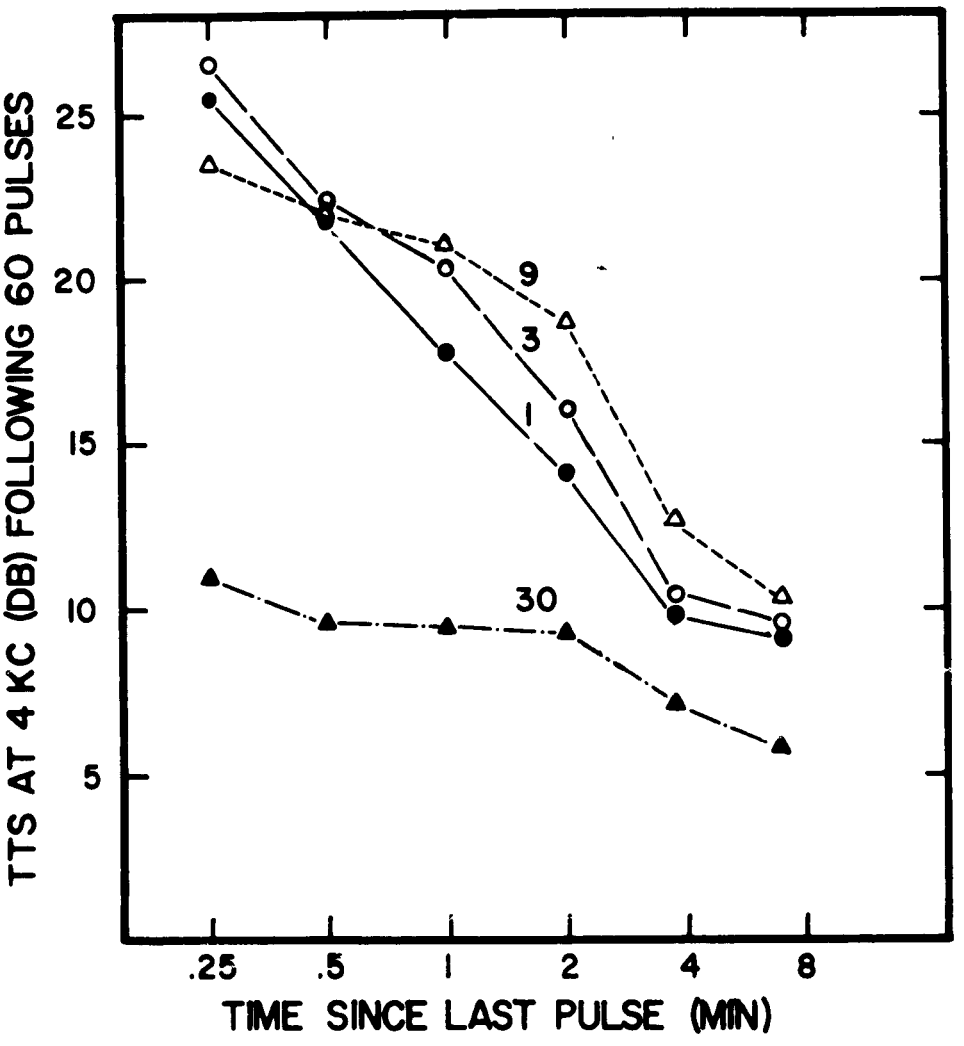

Fig. 4. Recovery in time of the TTS at 4 kc produced by 60 high-intensity pulses. Interpulse interval, in seconds, is the parameter.

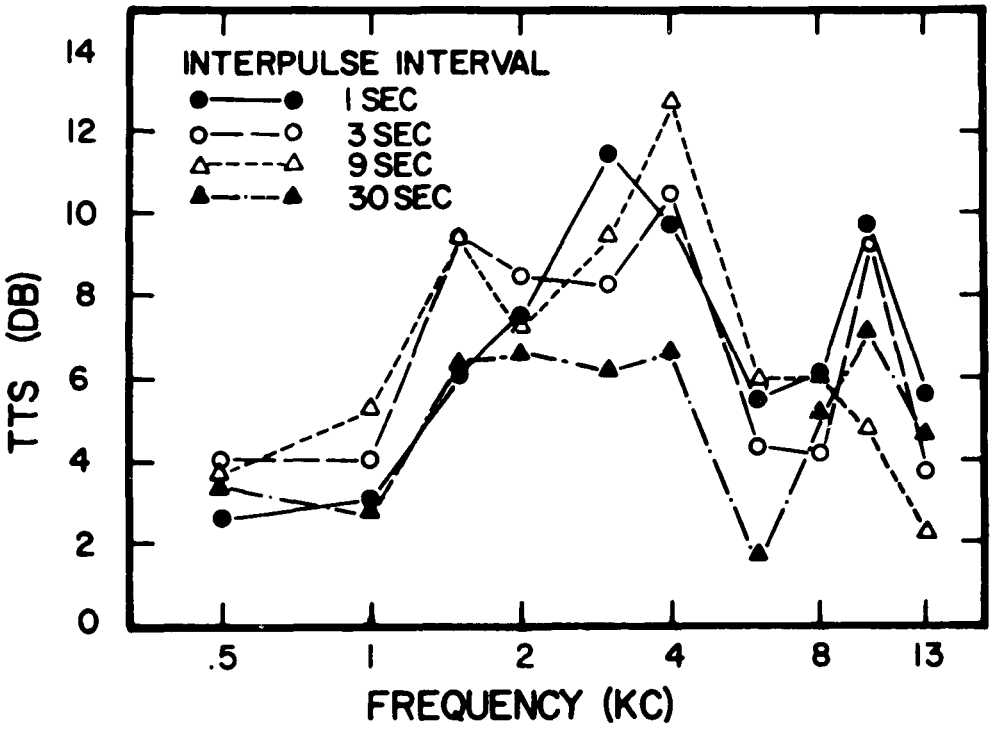

Fig. 5. Temporary threshold shift 5 min after exposure to 60 high-intensity pulses as a function of test frequency. Interpulse interval is the parameter.

PROTECTION AFFORDED AGAINST IMPULSIVE NOISE BY VOLUNTARY CONTRACTION OF THE MIDDLE EAR MUSCLES

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Los Angeles, California

INTRODUCTION

A study by Fletcher and Riopelle (1959) was the first to clearly show that prior activation of the middle ear muscles results in reduction of temporary threshold shift (TTS) from intense impulsive noise. TTS is defined as the difference between the resting threshold and a post noise-exposure threshold. Fletcher and Riopelle exposed a group of soldiers to gunfire with and later without a reflex-activating (1,000 cps at 98 db SPL) and found a much reduced TTS with reflex activation. Ward, Selters and Glorig (1961) using pulses generated by a high intensity speaker demonstrated that a 1,000 cps tone at 103 db SPL, whose onset preceeded each pulse by 105 msec, attenuated the TTS-producing pulses by an average of 10 db.

Elicitation of the middle ear muscle contraction in these studies has been by loud tones only, although other studies have suggested that voluntary contraction of the middle ear muscles also results in sound attenuation. Smith (1943) reported on a case in which voluntary contraction resulted in an increase in the auditory threshold, most notable for frequencies below 2,000 cps. Reger (1960) reported a very similar pattern of threshold increase in four voluntary contractors.

No studies have, as yet, been concerned with demonstrating the attenuating effect of voluntary contraction on TTS-producing noise. The purpose of the present investigation was to determine if voluntary contraction of the middle ear muscles has a similar ability to attenuate intense impulsive noise as has been demonstrated for its involuntary counterpart, the acoustic reflex. It was hypothesized that less TTS would develop when voluntary contraction preceeded noise exposure than when it did not. The investigation was also designed to suggest the amount of such protection.

METHOD

Pre-experimental Procedure: Six subjects were selected from a panel of 15 listeners previously engaged in an impulsive-noise

experiment. Selection was based upon their claim, in response to questioning, that they were able (apparently by voluntary contraction of the middle ear muscles) to attenuate intense noise. These subjects also reported that during voluntary contraction they hear a low pitched roar, presumably noise produced by the muscle contraction itself. Results of audiograms administered to these subjects, with and without voluntary contraction, revealed a pattern of threshold elevation during voluntary contraction very similar to those obtained by Smith and by Reger.

Experimental Procedure: Acoustic clicks served as TTS-producing noise. A procedure, previously employed by Ward, Selters, and Glorig (1961), was used which consisted of eight 1-minute exposures (25 clicks at regular 2.4 second intervals) separated by 1-minute TTS measurements. The intensity of the first exposure was approximately 132 db SPL and each successive exposure was increased by approximately 3.5 db, reaching a maximum of 155 db on the eighth exposure. The exposures were terminated after the eighth exposure or at any exposure after which the measurement showed a TTS of 30 db or more at 4,000 cps.

Each subject participated in two sessions (one week apart), one in which he was instructed to anticipate each click with sound-attenuating contraction, and another in which he was instructed to make no such contraction. Three of the six subjects participated in replications for the purpose of increasing reliability.

Only ears showing at least 30 db of TTS, in either treatment, were considered in testing the experimental hypothesis. When both ears met this criterion, the ear meeting the criterion at the lower exposure level was chosen.

RESULTS

To test the hypotheses that less TTS is developed in the voluntary contraction condition, a comparison was made of the TTS developed under the two conditions for each subject. The click level at which the comparison was made was always the highest level common to both conditions.

A t-test for differences of paired observations showed that the difference in TTS between the two treatments was significant beyond the .01 level of probability. Some idea of the amount of attenuation afforded by the voluntary contractions can be gained from Figure 2.

It can be seen that voluntary contraction produced an average of about 10 db of attenuation of click intensity. This amount of attenuation is the same as that produced by a reflex activating tone of 103 db (Ward, Selters, and Glorig, 1961). Figure 1 shows that all five subjects reaching the criterion of 30 db TTS did so with fewer exposures (consequently at lower intensity levels) under the uncontracted condition than under the voluntary contraction condition.

DISCUSSION

In order for proper inferences to be drawn concerning the actual role of the middle ear muscles in producing the experimental results, some other relevant observations will be reported. First, subjects (DK and DL) were observed grimacing and blinking strongly before each pulse. The results of these subjects might be explained by attenuation produced through activation of the tensor tympani associated with blinking of the eyelids (Klockhoff, 1961). In support of this explanation it was noted that DK could not produce loudness changes of sustained (more than a second) suprathreshold sounds. It is probable, in his case, that threshold shifts noted during contraction were the result of direct masking by noise from contracting face and neck muscles. However, in the case of another "grimacer" (DL) voluntary contraction was observed using a monaural balancing procedure to reduce the loudness of a supra-threshold tone (500 cps at 40 db SL) by 15 db. It is concluded for this observation that sustained voluntary contraction of the middle ear muscles may be accompanied by unnecessary facial movement.

CONCLUSIONS

It may be concluded that persons classified as "voluntary contractors" on the basis of their ability to raise their auditory threshold, also are able to attenuate intense TTS-producing sounds. The results may have military and industrial significance. There is no doubt that progressive hearing loss develops from repeated exposure to impulsive noise. Of added significance is the finding by Ward, Selters, and Glorig (1961) that, unlike noise-induced TTS, which increases in proportion to the logarithm of time, pulse TTS increases linearly with time.

Fletcher and Riopelle (1959) suggest the use of a sound-induced contraction as a protection in impulsive-noise situations. A voluntary contraction method of protection may be contrasted with one based on sound-induction: 1) contraction may be activated by any stimulus of

signal value (light, weak tone, time interval, etc.); 2) it may serve as additional protection in conjunction with the wearing of an earplug or cushion. However, the practicality of any voluntary contraction method requires that this ability be possessed or readily acquired by a large percentage of the population. Reger (1961) estimates that only 2% of the population have this ability. Results of the present investigation suggest that this proportion is much higher (6 out of 14 subjects). This discrepancy may be explained if it is assumed that our subjects learned by trial and error to make middle ear muscle response under voluntary control, perhaps through a conditioning procedure, merits some consideration.

REFERENCES

Fletcher, J. L. and Riopelle, A. J. Protective effect of the acoustic reflex for impulsive noise. J. Acoust. Soc. Am., 1960, 32, 401-404.

Klockhoff, I. Middle ear muscle reflexes in man. Acta Oto-Laryngol., Suppl. 164, 1961, 1-92.

Reger, S. Effect of middle ear muscle action on certain psychophysical measurements. Ann. Otol. Rhin. and Laryngol., 1960, 69, 1179-1198.

Smith, H. D. Audiometric effects of voluntary contraction of the tensor tympani muscle. A.M.A. Arch. Otolaryngol., 1943, 38, 369-372.

Ward, W. D., Selters, W. and Glorig, A. Exploratory studies on temporary threshold shift from pulses. J. Acoust. Soc. Am., 1961, 33, 781-793.

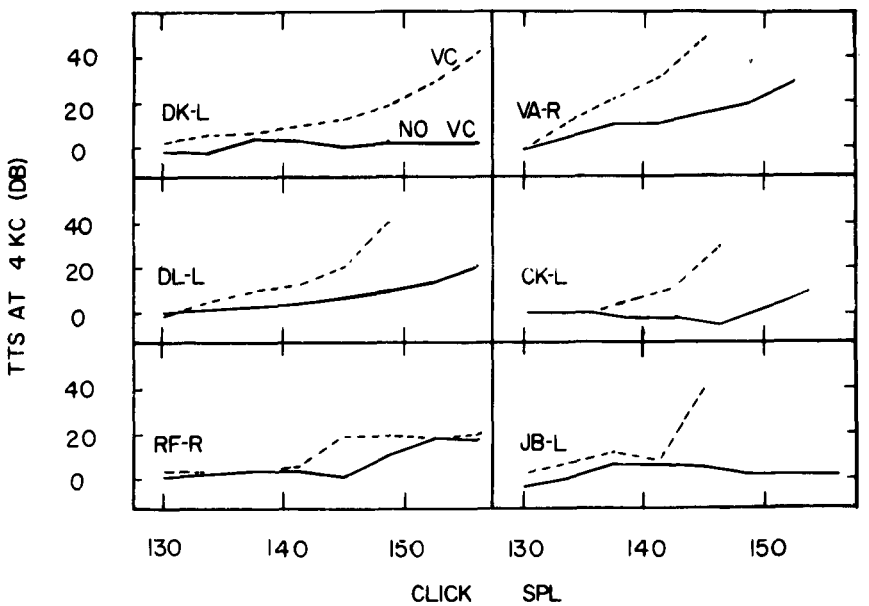

Fig. 1. Individual records comparing TTS produced with and without voluntary contraction (VC) of the middle ear muscles.

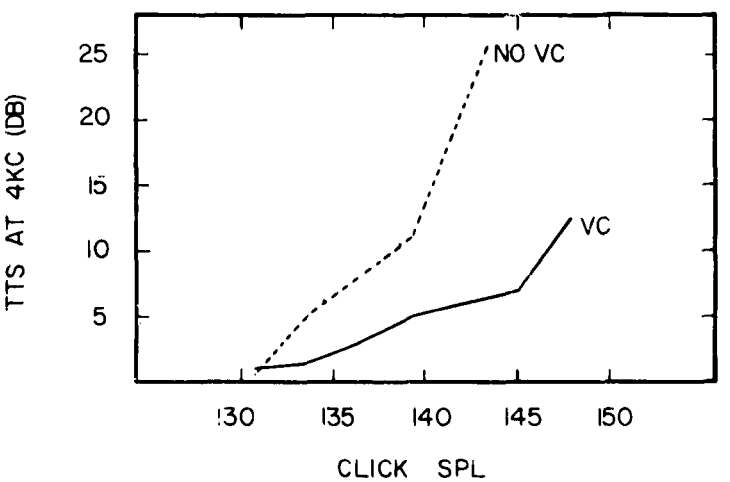

Fig. 2. Average growth of TTS (6 records of Fig. 1) with successive exposures to increasing pulse levels with and without voluntary contraction (VC) of the middle ear muscles. The attenuation produced by VC is given by the amount by which the function generated with VC is shifted to the right.

SUMMARY OF SYMPOSIUM ON MIDDLE EAR FUNCTION

William D. Neff

Bolt Beranek and Newman Inc.
Cambridge, Massachusetts

In the course of our two-day symposium, we have had the privilege of listening to and discussing reports of research on many different aspects of the structure and function of the middle ear and especially of the middle ear muscles: studies of the anatomy of the middle ear muscles and their nerve supply; investigations of factors affecting reflex responses of the middle ear muscles in lower animals and in man; experiments done to develop better techniques of recording middle ear muscle responses in man; measurements of the protection provided against loud noise by sound induced reflex response and by voluntary contraction of the middle ear muscles; and finally, attempts to construct a theory of middle ear function which takes into account known facts about anatomical structures involved and the actions of the middle ear muscles.

Dr. Henson has described middle ear structures of a number of species of the bat and has made some interesting suggestions as to the possible functional significance of certain differences in structure. In particular, he has pointed out differences between bats which make great use of echolocation and others which do not. He has sought to explain the problem of how bats hear faint echoes at the same time that they are emitting a series of intense sound pulses; the mechanical isolation of the cochlea appears to be one important factor. The middle ear muscles, he feels, may aid in reception of faint high frequency signals by "tuning" the ear and attenuating low frequency background noise.

After so many years since the first anatomical investigations of the innervation of the middle ear muscles, it is surprising how many questions remain to be answered. Dr. Blevins and Dr. Lawrence have shown how some of these questions can be answered through the careful application of modern neuroanatomical methods. Dr. Blevins has emphasized the finding of a rich supply of motor endings in the middle ear muscles (a low ratio of muscle fibers to nerve fibers). His failure to discover any sensory endings in either the stapedius or tensor tympani is of special importance to those of us interested in the neural mechanisms controlling responses of the middle ear muscles.

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The evidence presented by Dr. Lawrence from his study of the temporal bones of two 15 week (105 mm) human fetuses has confirmed in part the results of earlier studies, namely, that the stapedius muscle receives its innervation from the facial nerve. But, he has shown that the nerve supply to the tensor tympani is more complex than that reported by earlier investigators. The tensor tympani receives a dual innervation, from the mandibular branch of the trigeminal nerve via the otic ganglion and from tympanic plexus nerve fibers as they pass over the promontory to join the lesser superficial petrosal nerve. Dr. Lawrence has noted that this dual innervation is probably related to the known complex physiological response of the tensor tympani.

In his experiments, Dr. Simmons has undertaken a most difficult task, that of recording the responses of the middle ear muscles in the unanesthetized animal. Through the use of electrodes inserted into the muscles and an electrode placed near the round window, he has been able to record and identify electrical activity, apparently generated by each of the muscles separately, and to measure the reduction in cochlear microphonic response when both muscles are active. The results of the many experiments described by Dr. Simmons cannot be summarized readily or briefly. To illustrate the nature of the experiments, a few of the findings will be mentioned. Stimulus parameters affecting the responses of the middle ear muscles were examined. Tones in the lower part of the range of frequencies to which the experimental animal (cat) was sensitive were more effective in producing sustained muscle contractions than were higher frequencies. On the other hand, higher frequencies were more effective in producing an "on" (not sustained) response. Other stimulus variables studied included rise-time, duration, and manner of presention--i.e., to the ipsilateral ear, to the contralateral, or to both. On the response side, attention was given to type of response ("on" and sustained) and to such variables as strength and latency of contraction. Very short latencies (time between onset of cochlear microphonic response and start of response recorded from electrode implanted in muscle) were measured. For stimuli applied to the ipsilateral ear, latencies as short as 3.7 msec were recorded for the stapedius and 5.4 msec for the tensor tympani. Maximum latencies of 12 msec for the stapedius and 20 msec for the tensor tympani were observed.

In addition to studying stimulus and response variables such as mentioned above, Dr. Simmons also explored the effects on middle ear muscle activity of variables that are not related directly or immediately to the sound stimulus nor to the aspects of response measured. The muscle responses were found to be affected by a variety of conditions

such as auditory fatigue, habituation, and arousal. (Definitions of these terms are given in the text of Dr. Simmons' paper.)

In experiments on the anesthetized guinea pig, Dr. Mundie has made measurements of the impedance of the ear. He has shown that the total impedance, measured by his techniques, "is predominantly the elastance of the middle ear space up to 2,000 cps, the resistance of the cochlea from 2,000 cps to 9,000 cps, and is probably the inertance of the ossicular chain above 9,000 cps. In the frequency range where the impedance of the total ear is the least (2,000 - 9,000 cps), that impedance is predominantly cochlear and resistive. This is an optimum situation wherein most of the sound energy absorbed by the ear goes to the cochlea and the frequency response is reasonably flat because there is little reactance in the total ear." Mundie's measurements have also led him to propose hypotheses as to the action of the stapedius and tensor tympani muscles.

The problem of measuring activity of the middle ear muscles in man is one to discourage any but the most persistent investigator. For a number of years Mr. Mendelson has been demonstrating that he possesses this characteristic and, as a result, he has been able to develop a manometric technique by which he can obtain quite reliable measures of middle ear muscle responses. He has shown us the results of numerous interesting experiments. I shall call attention to only a few of his findings. One is the individual differences in the nature of the combined response of stapedius and tensor tympani; for example, some subjects show outward displacement of the eardrum in response to loud sounds presented to the contralateral ear, other subjects have inward displacements, and the direction of displacement for a given subject may change over a period of time. In the course of measurements made during studies of temporary threshold shift from noise exposure, Mendelson found evidence which might be explained by a conditioning of the response of the middle ear muscles. He also noted spontaneous contractions in some subjects who had been exposed to loud noise repeatedly. He suggests that these periodic contractions may in some instances make the ear better able to detect certain signals in a noise background.

Weiss, Mundie, Cashin, and Shinbarger have used a somewhat different manometric technique to examine middle ear muscle responses in normal human subjects. They mention also the results for one patient suspected of having middle ear pathology. In this patient, the manometric tests successfully predicted the absence of an otosclerotic condition. For the normal human subject, one finding of considerable interest is that both the stapedius and tensor tympani contract in response to loud sounds. It has sometimes been suggested

that in man only the stapedius reflex is initiated by sound. The average threshold for the response of the middle ear muscles as measured by the method of Weiss et al was about 92 db ± 4 db above the detection threshold. They did not find that frequency or shape of the signal was particularly critical. Mendelson also reported similar high threshold levels. No appreciable response could be recorded in approximately 5% of the 30 subjects tested by Weiss and his collaborators even at stimulus levels 115 db above detection threshold. The records obtained in those subjects in which middle ear muscle responses could be recorded readily support the view that the tensor tympani muscle contraction pulls the eardrum inward and that stapedius contraction produces an outward movement. Further confirmation of this interpretation can come from examination of patients with one or the other muscle detached. Tests made on patients with only the tensor tympani functioning indicated that it does pull the eardrum inward, producing negative pressure in the external meatus.

Dr. Loeb has presented a concise but comprehensive review of past research on the function of the middle ear muscles. In this review he refers to a number of unanswered questions--some of which are being answered by other papers presented in the symposium. Dr. Loeb concludes his review by mentioning how knowledge of factors controlling responses of middle ear muscles may be applied in order to make use of their action to protect the ear from injurious sound stimuli. The research of Fletcher and his collaborators here at Fort Knox, as well as similar investigations by others, is summarized. Dr. Loeb also points out the need for considering the effects of action of the middle ear muscles in interpreting results of a number of kinds of experiments, for example, studies of sensory interaction, of attention, of masking, and of remote masking.

In his experiments on contralateral remote masking, Dr. Ward has shown that the amount of masking is not a measure only of attenuation produced by responses of the middle ear muscles. Central masking must also be taken into consideration. We have had mentioned several times measures of latencies of response of the middle ear muscles. Dr. Simmons presented a table showing measurements which he has made in the unanesthetized cat. Dr. Ward raises the question of response latency in relation to the practical problem of providing protection against loud sounds by producing contraction of the middle ear muscles in advance by presentation of a loud sound stimulus but one which will produce no damage to the ear and usually no temporary threshold shift. He has found that the reflex arousing signal should precede the "noxious" or

damaging sound by 150 to 200 msec in order to provide maximum protection for nearly all individuals.

It would be highly desirable in many situations, such as in tanks firing guns, to make full use of the protection provided to the ear by contraction of the middle ear muscles. Drs. Loeb, Ward, and others have described techniques which make this possible by using a non-damaging loud sound to produce reflex contraction of the muscles. We may sometimes question Nature's lack of good design in not providing voluntary control of the middle ear muscles. Whether it be a "hangover" from the long past when voluntary control of the muscles was useful for attending or not attending to certain classes of sounds or a precocious adaptation to our increasingly noisy world, a few individuals do apparently have the ability to contract their middle ear muscles. The papers presented by Fleer and by Reger, Menzel, Ickes and Steiner describe experiments for which subjects were selected because they could contract their middle ear muscles voluntarily.

Fleer measured temporary threshold shift in six subjects under two conditions, exposure to loud noise (series of clicks) with voluntary contraction of the middle ear muscles and the same exposure without contraction. He found that the voluntary contraction produced protection approximately the same as a 10 db decrease in sound pressure level of the noise stimulus.

Reger et al made measurements of air conduction and bone conduction transmission losses (using a pure tone Békésy type audiometer) for the six ears of three subjects who were able to contract their middle ear muscles voluntarily. Shifts in threshold for air conduction were in the range 20 to 30 db for tones below 1,000 cps; reduction was very small or absent for higher frequencies.

Both Fleer and Reger report numerous observations which may be useful to other investigators interested in discovering subjects who have voluntary control of the middle ear muscles. The suggestion is made that it may be possible for subjects to learn voluntary control.

Dr. Zwislocki has made use of known facts about the anatomy of the middle ear of man and indirect evidence of middle ear function such as measurements of input impedance in unanesthetized human subjects to attempt an electro-acoustic analogue of the middle ear. In constructing his model, he found it necessary to check on certain measurements which have been previously reported on the volume of the middle ear cavities. Large individual differences were found and the maximum

volumes measured were greater than those reported by earlier investigators. Dr. Zwislocki discusses ways in which his model may be used in place of experiments which cannot be done at present on the living human subject. Pathological changes in the middle ear can be simulated by the model.

Any scientist interested in hearing and particularly the role of the middle ear will find, I am sure, in reading the papers of this symposium, information of several classes: (1) confirmation of the findings of earlier experiments but, because of improved techniques and equipment, more precise and more reliable measurements; (2) new results that either provide data where there were none or correct mistaken observations and speculations; (3) questions raised to which we can expect answers from future experiments. Some examples of information falling in all three of these categories have been given in the preceding part of this summary. I wish to call attention to several problems which I see as remaining to be solved. My selection will be biased because of my interest in psychophysiology and neurophysiology.

We need more comparative information about both structure and function of the middle ear muscles and their associated neural system so that we know to what extent we are dealing with similar mechanisms as we go from bat to guinea pig to cat to monkey to man. There appear to be considerable differences between the bat and higher mammals, and there may be important differences between the cat or monkey and man. Because we can do experiments on the cat and monkey that we cannot do on man, it behooves us to find out, in so far as possible, how they differ. In this symposium, we have had quite convincing evidence presented that the tensor tympani muscle in man contracts in response to loud sounds. But many researchers in the past have stated that man probably differed from other animals studied in that only his stapedius muscle was reflexly controlled by sound. Inadequate techniques of measurement led to a false conclusion if our new evidence is correct.

At the neurophysiological level, we have only the simplest diagram of the complex plan by which we may represent the control system of the middle ear muscles. It is most critical that we have a conclusive answer about the presence or absence of sensory endings in the muscles. Dr. Blevins has failed to find any, and he has argued that the sensory endings described by others were not in fact true sensory endings. The final solution to this problem of kinesthetic receptors will have to come from physiological experiments.

If the stapedius and tensor tympani muscles have no afferent nerve supply, the neural circuitry involved in their control will be simplified, but it is still a complex system. There have been only the crudest kinds of experiments done on the brain stem part of this system. Progress in the future depends upon improving techniques for recording muscle responses and nerve impulses in the unanesthetized preparation. Electrodes placed in the muscles, for example, can pick up change in muscle fiber potentials and cochlear microphonic response. Movement of the electrode by the muscle may also produce a recordable electrical change.

Several of the speakers have mentioned the possibility that reflex responses of the middle ear muscles may account for some changes in auditory sensation in psychophysical experiments. A related problem is the possible role contraction of the muscles may play in such psychological phenomena as attention.

On the practical side, evidence such as that which has been presented here makes it appear feasible to use acoustically induced reflex responses of the middle ear muscles to provide limited protection against sudden bursts of noise such as gunfire. Research in this area should be continued to explore other means of producing action of the muscles. Some kinds of tactual stimulation can set off reflex contractions. The possibility has also been suggested that contraction of the muscles may be brought under voluntary control through conditioning.

This symposium has served a most useful purpose in bringing together researchers from a number of different fields to present and discuss their studies of the structure and function of the middle ear. I wish to conclude by thanking the speakers and discussants for their participation in the symposium. And, on behalf of all the visiting participants, I wish to thank Colonel Bach, Dr. Odell, Dr. Harker, Captain Fletcher and the other members of the USAMRL staff who have provided this opportunity for us to meet and who have made our visit pleasant as well as productive. We are particularly grateful to Captain Fletcher for the time and effort he has given in planning and arranging our meeting.