Assessment of Central Auditory Dysfunction

FOUNDATIONS AND CLINICAL CORRELATES

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CHAPTER 7

The Acoustic Reflex in Central Auditory Dysfunction

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This chapter by Dr. Hall discusses the principles and methodology of acoustic reflex measurements and the factors that influence them. He then describes the acoustic reflex in nerve VIII and intraaxial and extraaxial brainstem pathologies with case examples.

Clinical application of acoustic impedance (immittance) audiometry was first comprehensively described by Metz in 1946. Subsequently, tympanometry and measurement of static compliance and the acoustic reflex became integrated into an effective test battery for objective assessment of middle ear function (Hall and Jerger, 1982; Jerger, 1970; Jerger et al, 1974a). Measurement of the **acoustic stapedial reflex**, however, provided a sensitive index of auditory function extending far beyond the middle ear, contributing significantly to the versatility of immittance audiometry. The neurodiagnostic value of acoustic reflex measurements have, for over a decade, been repeatedly documented. They are clinically useful in the prediction of sensorineural hearing impairment (Hall, 1980; Hall and Koval, 1982; Jerger et al, 1974b; Margolis and Fox, 1977; Niemeyer and Sesterhenn, 1974), the differentiation of cochlear vs retrocochlear auditory dysfunction (Anderson et al, 1969; Jerger et al, 1974c; Sanders et al, 1974; and others), and the identification and localization of brainstem auditory pathology (Borg, 1973; Griesen and Rasmussen, 1970; Jerger and Jerger, 1977; and others).

This final diagnostic application—the acoustic reflex in central auditory dysfunction—is the topic of the present chapter. In contrast to the substantial published clinical experiences with acoustic reflexes in peripheral (middle ear and sensorineural) impairment, there is only a modest number of reports documenting acoustic reflex abnormalities in patients with confirmed **central auditory nervous system** lesions. The neuroanatomy, neurophysiology, and neuropharmacology of the acoustic reflex in man is not adequately described. Also, there are only preliminary studies of the distinctive influence of central auditory system pathology on the major **acoustic reflex** parameters—**threshold, latency, decay, and amplitude.** Furthermore, effects of subject characteristics, such as age and sex, remain poorly understood and rather controversial, and yet they seem to be important in the clinical interpretation of acoustic reflex findings. Finally, there is only a limited number of comprehensive clinical reports of acoustic reflex measurements in patients with neoplastic, vascular, or demyelinating central neuropathies. The purpose of this chapter is to review current knowledge of the acoustic reflex in central auditory dysfunction, illustrating the above noted issues with case studies and original data. Recent detailed descriptions of acoustic reflex measurement procedures are available elsewhere (Hall and Jerger, 1982; Jerger and Northern, 1980; Lilly, 1973). The chapter concludes with a discussion of promising new research directions.

PRINCIPLES OF ACOUSTIC REFLEX MEASUREMENT

Anatomy

Fifty years ago, Lorente de Nó (1933a, b, and 1935) conducted comprehensive neu-

roanatomical investigations of the acoustic stapedial reflex. Yet, the *anatomy* of the acoustic reflex arc is still not completely known. Available information is based on experimental studies in animals and clinical correlation of acoustic reflex abnormalities with sites of neuropathology (Borg, 1973; Brask, 1978). The peripheral components of the arc, both afferent and efferent, are reasonably well-described. Structure and function of the afferent portion—the *middle ear system*, the *cochlea*, and the *eighth (acoustic) cranial nerve*—are presented elsewhere in this volume (Chapter 2). The efferent portion consists of the *seventh (facial) cranial nerve*, the *stapedius muscle and tendon*, and *the stapes*. The motor division of the facial nerve courses from its caudal pontine brainstem nucleus to the muscles of the face. After exiting the internal auditory canal at the stylomastoid foramen, it gives off a short branch of largely myelinated fibers (perhaps 10% of the entire motor division) to the stapedius muscle (Foley and DuBois, 1953). Interestingly, a greater number of fibers appear to innervate the right middle ear muscles than the left, at least in cat (Foley and DuBois, 1953; Lyon, 1975). The stapedius is the smallest skeletal muscle in mammals. Animal studies in cat, rat, rabbit, guinea pig, and chicken have shown that it consists of two types of fast, twitch fibers, and a morphologically slow fiber component (Anniko and Wroblewski, 1981; Borg et al, 1979; Burgener and Mayr, 1980; Erulkar et al, 1964; Fernand and Hess, 1969; Hirayama and Daly, 1974). Also, there are both multiply and singly innervated muscle fibers (Fernand and Hess, 1969; Tieg and Dahl, 1972). The stapedius muscle is largely enclosed within a bony canal in the posterior tympanic wall and runs parallel but anterior/interior to facial nerve. In some persons, the muscle and nerve are in the same bony compartment. The stapedius muscle tendon emerges at the pyramidal eminence in the tympanic cavity (middle ear) to attach on the posterior aspect of the neck of the stapes.

The central components of the acoustic reflex arc—mediating activity between the afferent portion on one side and both left and right efferent portions—are not completely described in animal, and poorly understood in man. Borg's (1973) often cited report of a comprehensive anatomic and physiologic investigation of the reflex pathways in rabbit is suggested for further reading. (A discussion of brainstem anatomy in general is presented in Chapter 2 of this volume.) There are direct and indirect arcs. The direct pathways are most studied and appear to consist of a 3- or 4 neuron chain, including (1) sensory neurons of the eighth cranial nerve, i.e., primary afferents, (2) neurons from the *ventral cochlear nucleus* terminating in the vicinity of the ipsilateral motor facial nerve nucleus and in the ipsilateral and contralateral *medial superior olive* (MSO), (3) neurons from in or around the MSO and terminating on the ipsilateral or contralateral *region near the motor nucleus of the facial nerve*, and (4) motor neurons of the facial nerve, i.e., the final efferents, arising from the area of the facial nerve motor nucleus and terminating on fiber(s) of the ipsilateral stapedius muscle. Thus, there are ipsilateral and contralateral acoustic reflex pathways, with the former involving afferent, brainstem intermediate and efferent structures on the same side, and the latter involving afferents on one side, neurons which cross the caudal pontine brainstem midline, presumably via the trapezoid body, and efferent neurons on the opposite side. These pathways and synapses are often depicted schematically in the literature (Brask, 1978; Clemis and Sarno, 1980; Djupesland, 1980; Jerger, 1980; Jerger et al, 1980; Møller, 1983). However, the components of the central reflex arc, especially in man, are not clearly defined, and such diagrams are probably a gross, and not entirely accurate, oversimplification of a complex temporal and spatial interaction of brainstem auditory structures.

One of the least understood features of the *direct* central acoustic reflex arc is the origin of the facial nerve efferents that innervate the stapedius muscle. Borg (1973) and others (Papez, 1927) offered evidence from lesioning and retrograde degeneration experiments suggesting that the motor neurons (third or fourth neuron of the reflex pathway) originate from the medial and/or lateral region of the nucleus of the seventh cranial nerve. In contrast, studies with horseradish peroxidase (HRP) retrograde trans-

port techniques in monkey and cat (Lyon, 1978; Stach et al, 1983; Strominger et al, 1980) showed motor neurons predominately in an area that was clearly outside of the medial or lateral neuronal groups of facial nerve nucleus, and in close proximity to the lateral or medial superior olive. Further investigation on this component of the acoustic reflex with current neuroanatomic methodology is continuing.

An *indirect* acoustic reflex pathway clearly exists, although very little is known of its components in man or experimental animal. Borg (1973) speculated that this slower, polysynaptic pathway might involve the extrapyramidal motor system or the *reticular formation* (RF). The extreme sensitivity of the acoustic reflex to the influence of barbiturates, discussed and illustrated later in this chapter, supports the concept of an alternate, RF-mediated, pathway which may, in fact, predominate in man. Also, there are other, documented interactions between the traditional auditory structures and the RF (Brodal, 1981; Rasmussen, 1946). Furthermore, the short and long latency components of the acoustically elicited stapedius muscle EMG show differential sensitivity to state of arousal and higher CNS influences (Baust and Berlucchi, 1964; Salomon, 1966), much like the two temporal components of the corneal or blink response (Brodal, 1981), a cranial nerve five-to-seven reflex arc. The possible contribution of auditory cortical areas to acoustic reflex activity has been suggested in recent clinical reports (Downs and Crum, 1980; Jerger, 1980) and is the topic of ongoing experimental study (B Stach, personal communication, 1983).

Physiology

There are numerous recently published accounts detailing the physical and functional basis of the acoustic reflex (Jerger and Northern, 1980), and only a brief description will be presented here. Contraction of the stapedius muscle in response to high intensity sound has been repeatedly demonstrated in man for over 50 yr (Jepson, 1951; Lüscher, 1929; Metz, 1946). The reflex is bilateral or consensual; that is, a sound presented to one ear produces contraction of the stapedius muscle on the same side (ipsilaterally) *and* on the opposite side (contralaterally). Clinically, contraction of the stapedius muscle can easily be detected by measuring the reflex-related change in middle ear acoustic immittance (primarily reduced mobility) close to the plane of the tympanic membrane (Jerger and Northern, 1980; Lilly, 1973; Møller, 1972). Other methods of acoustic reflex measurement, suitable in experimental preparations, include mechanical transduction of ossicular movements or direct EMG of the stapedius muscle (Møller, 1972).

Normal stapedial reflex activity in response to acoustic stimulation requires integrity of the afferent, brainstem, and efferent pathways. Clinical correlations among neurotologic pathophysiologies and acoustic reflex patterns will be discussed and illustrated later in this chapter. It is important to keep in mind, however, that although the diagnostic objective may be evaluation of neurotologic status, it is first necessary to rule out the influence of obvious middle ear and cochlear involvement in acoustic reflex abnormalities (Hall and Jerger, 1982; Jerger et al, 1974a and b). That is, to elicit normal acoustic reflex activity, a sound of 70 to 90 dB sound pressure level (SPL) must first reach the cochlea, then be transduced bioelectrically, and adequately transmitted via the eighth cranial nerve to the ventral cochlear nucleus. The contralateral or ipsilateral acoustic reflex cannot be measured, even with the probe in a normal ear, if the sound intensity reaching the cochlea of the stimulus ear is attenuated to less than 70 to 90 dB SPL by conductive impairment. And an ipsilaterally elicited acoustic reflex is rarely observed even in an ear with a minimal (5 to 10 dB) conductive component (Jerger et al, 1974a). Likewise, acoustic stimulation of an ear with severe cochlear impairment (60 to 70 dB) or mild eighth nerve auditory impairment (0 to 40 dB) is unlikely to produce measurable acoustic reflex activity, although middle ear functioning is normal (Jerger and Jerger, 1974c).

The actual *physiologic basis* of the stapedial acoustic reflex remains unclear. The role of middle ear function in acoustic reflex measurements, at both the afferent and efferent extremes of arc, is well-described (Møller, 1972). However, the sensorineural

and brainstem physiologic substrate of the acoustic reflex is largely conjecture. In normal hearers, at least, the acoustic reflex appears to be dependent in some way on loudness summation, i.e., the perception of a sufficiently loud sound. This conclusion is supported, indirectly, by documented relationships between critical bandwidth estimations made by both acoustic reflex and psychophysical methods (Block and Wiley, 1979; Djupesland and Zwislocki, 1973; Flottorp et al, 1971; Popelka et al, 1976), although there are serious discrepancies in findings among studies (Margolis and Popelka, 1975). Acoustic reflex activity also seems to be closely related to temporal summation of acoustic energy, an interaction of stimulus duration and intensity (Djupesland et al, 1973; Gelfand et al, 1981; Jerger et al, 1977; Woodford et al, 1975; and others). These acoustic reflex phenomena have particular relevance to the present chapter as they are, presumably, mediated in the central auditory nervous system (Boudreau, 1965; Zwislocki, 1969).

More precise understanding of the neurophysiology of the acoustic reflex would significantly enhance its clinical exploitation. There are a host of unanswered questions: for example, aside from the vaguely defined role of loudness sensation, what neurophysiologic processes underly acoustic reflex threshold, latency, and amplitude parameters? Which types of auditory brainstem neurons mediate the acoustic reflex vs, for example, the auditory brainstem response or complex speech signals? Is there binaural neural interaction in acoustic reflex measurement, as suggested by the larger magnitude response to simultaneous diotic stimulation than to typical monaural stimulation (Møller, 1962; Reker, 1977)? Do central auditory structures rostral to the pons modulate (either excite or inhibit) acoustic reflex activity? What are the differences in the neural bases of the acoustic reflex among man and experimental animal materials (e.g., cat, rabbit, guinea pig, monkey)? In view of the now proven clinical value of acoustic reflex measurements, it is likely that these basic questions, and many others, will be the subject of further investigations.

Methodology

Twenty-five years ago, Møller (1958, 1961, 1962) described measurement of the acoustic reflex with specially constructed *experimental* instrumentation. In recent years, a number of investigators have also designed reflex-measuring systems that offer some distinct advantages over commercially available equipment, especially in the assessment of central auditory functioning. A popular approach is the use of laboratory devices for stimulus generation and manipulation (eg., gating, shaping, timing) with reliance on commercially available meters for measurement of immittance (Gelfand et al, 1981; Popelka et al, 1976; Thompson et al, 1980; Wilson, 1979). This arrangement permits virtually unlimited selection of stimulus parameters and assures that immittance changes will be validly measured and reported in acceptable units, such as acoustic admittance (millimhos) or as equivalent compliance in cubic centimeters. There are, however, four main limitations to this approach: (1) only one or two probe tone frequencies (220 and 660 Hz) are usually available; (2) accurate measurement of reflex temporal characteristics, such as latency, is confounded by time constraints inherent in the instruments (Mangham et al, 1982; Niswander and Ruth, 1979); (3) the wide variety of stimuli are presented via a standard contralateral earphone/cushion. Ipsilateral reflexes must be activated with the limited stimuli available on the commercial immittance meter; and (4) the type of acoustic transducer, and its coupling to the ear, differ substantially for contra- vs ipsilateral acoustic reflexes. This final limitation is extremely important in the assessment of central auditory functioning with acoustic reflexes.

Møller's original studies employed apparatus that circumvented the above noted drawbacks. The main concepts of this instrumentation have, within the past 5 yr, been revived, updated, and clinically documented by Jerger and colleagues (Hall, 1982a, b and, c; Hayes and Jerger, 1983; Jerger and Hayes, 1983; Jerger et al, 1978b). As illustrated in Figure 7.1, this system has unique and clinically attractive features: (1) Ipsiláteral and contralateral reflex stimuli are presented by

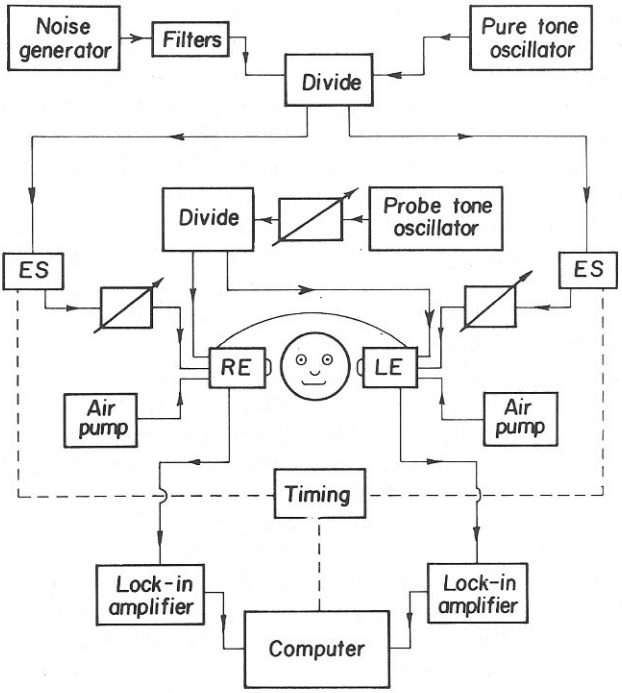

Figure 7.1. Specially constructed apparatus for equivalent measurement of ipsilateral and contralateral acoustic reflexes. Components include matched probe assemblies for presenting acoustic stimulus and detecting reflex-related changes in acoustic immittance, and minicomputer for averaging acoustic reflex activity.

identical, acoustically matched probe assemblies. Stimuli for the ipsilateral and contralateral modes can be calibrated equivalently and presented simultaneously under identical conditions. Reflex findings for the two stimulus modes, therefore, can be directly and confidently compared. (2) A wide variety of stimuli characteristics and probe tone frequencies can be selected with laboratory stimulus generators. (3) Changes in probe tone intensity are detected with the aid of a lock-in amplifier, eliminating the need for the filter components of standard immittance bridges which introduce, in part, the troublesome time constants. (4) Acoustic reflex activity for a sequence of stimuli is computer-averaged under each presentation condition (right and left, contra- and ipsilateral).

Factors Influencing Acoustic Reflex Measurements

Response Parameters. Four main acoustic reflex parameters—threshold, amplitude, latency, and decay are described in the literature.

The acoustic reflex threshold is commonly defined as the lowest sound intensity level in dB (hearing level relative to audiometric dB (HL)) or SPL producing a reliable change in middle ear immittance as measured near the plane of the tympanic membrane. Clinically, acoustic reflex activity is usually detected by visual monitoring of a meter, although most commercial instruments offer an X-Y plotter or strip chart for graphically recording reflex-elicited changes in immittance. Using 5 dB increments, reflex threshold levels of 90 to 95 dB SPL for tonal stimuli and 70 to 80 dB for broadband noise are expected in normal hearers, with standard deviations of approximately 5 dB (tones) and 10 dB (noise), respectively (Jerger et al, 1972; Wilson and McBride, 1978). Adherence to the preferred threshold determination procedures used in pure tone audiometry is not necessary in reflex threshold measurement, and there are no differences in

reflex thresholds obtained with ascending vs descending intensity techniques (Lilly and Franzen, 1970; Peterson and Lidén, 1972).

Metz (1951) first reported that the magnitude (amplitude) of acoustic immittance change increased directly as the intensity of the stimulus was increased. His observation, made on a single subject with a relatively primitive immittance measuring device, introduced a reflex parameter which, it now appears, has considerable clinical value in assessment of peripheral and central auditory functioning (Borg, 1973; Brask, 1978; Hall, 1982a, b, and c: Hayes and Jerger, 1983; Jerger et al, 1978b; Mangham and Miller, 1979). Acoustic reflex amplitude, often referred to as reflex growth, is calculated by comparison of the acoustic immittance of the middle ear (measured as described above) before acoustic stimulation (at rest) with the acoustic immittance during acoustic stimulation (reflexive). Reflex amplitude has been reported in a variety of units, including decibels (Jerger et al, 1978b; Peterson and Lidén, 1972; Silman and Gelfand, 1981), percent of maximum amplitude (Borg, 1973; Møller, 1961, 1962); acoustic ohms (Hung and Dallos, 1972), microvolts (Bosatra et al, 1975), millimhos (Thompson et al, 1980; Wilson and McBride, 1978), absolute cubic centimeters (milliliters) (Beedle and Harford, 1973), and mathematically derived cubic centimeters (Hall, 1982a, b, and c). These differences in the reflex recording units have contributed to discrepancies in amplitude findings among investigations and the variability often attributed to reflex amplitude measures (Wilson, 1979). In clinical studies of patients varying in age and middle ear status, it is important, when recording and interpreting reflex amplitude in any unit, to take into account the pronounced influences of static or resting immittance (Wilson, 1979) in the probe ear.

Latency is the time interval between stimulus presentation and some index of reflex-related immittance change. This statement is necessarily vague, since a major problem in calculating latency is difficulty in consistently defining the actual onset of reflex activity. The three acoustic reflex temporal characteristics most often cited as criteria for onset are: (1) any change from baseline in acoustic immittance (increase or decrease); (2) the points at which immittance first increases from baseline; and (3) some predefined percentage of the maximum reflex amplitude (Borg, 1982b; Clemis and Sarno, 1980; Hall and Jerger, 1976; Jerger and Hayes, 1983; Mangham et al, 1980; Norris et al, 1974). Precise myographic measurements of reflex latency in man, made by Perlman and Case (1939), showed a time interval of approximately 10 msec between tonal stimulation of one ear and action potentials in the contralateral stapedius muscle. The latency of stapedius muscle contraction, when detected with laboratory instrumentation as changes in acoustic immittance, is on the order of 25 to 35 msec at high stimulus intensities and 150 msec near threshold (Metz, 1951; Møller, 1958). Latency values obtained with commercially available immittance measuring devices are, in fact, only relative, as they are invariably affected by the temporal distortion produced by filter components (Clemis and Sarno, 1980; Hall and Jerger, 1976; Mangham et al, 1980, 1982; McPherson and Thompson, 1977; Niswander and Ruth, 1979). The clinical utility of acoustic reflex latency is further compromised by its confounding relationship with stimulus intensity and absolute reflex amplitude (Borg, 1982b; Hung and Dallos, 1972; Jerger and Hayes, 1983). Nonetheless, temporal reflex characteristics have been successfully correlated with sensorineural and brainstem lesions experimentally (Borg, 1982a), and, with careful application, may yet have clinical value (see Borg, 1976 for review of this topic).

Acoustic reflex *decay* has long held an important place in the audiologic test battery as a screening procedure for retrocochlear dysfunction (Anderson et al, 1969). The test is generally administered by presenting the acoustic stimulus for 10 sec at an intensity level 10 dB greater than the reflex threshold level (10 dB SL). The reflex-related immittance change is recorded and then analyzed for evidence of a decrease in amplitude of 50% or more over the time course. Slight decay is not uncommon in cochlea-impaired ears, and, infrequently, decay is also found in normal ears (Alberti, 1978; Habener and Snyder, 1974), but, as illustrated in a subsequent section, significant decay is a strong audiometric sign of retrocochlear (eighth

nerve or brainstem) pathology (Anderson et al, 1969; Jerger et al, 1974c; Olsen et al, 1981).

Stimulus Parameters. Meaningful interpretation of acoustic reflex findings in central auditory pathology requires an awareness of a broad range of stimulus effects on threshold, amplitude, latency, and decay parameters. (The following review is by no means exhaustive, and the reader is encouraged to consult original sources for details). These four reflex parameters are differentially influenced by the *mode of stimulus presentation—ipsilateral vs contralateral.* Investigations with clinical instrumentation have shown no significant difference between threshold levels for ipsilaterally vs contralaterally stimulated reflexes (Laukli and Mair, 1980). Equivalence in calibration of the standard earphone (contralateral transducer) and miniature (ipsilateral) transducer, which is sealed within the ear canal, is, of course, a problem in such studies and in clinical practice. Also troublesome are acoustic and eardrum artifacts with reflex measurements, especially in the ipsilateral mode (Danaher and Pickett, 1974; Kunoz, 1977; Leis and Lutman, 1979). Investigations with experimental, dual probe systems suggest that ipsilateral measurements yield more sensitive threshold levels than contralateral measurements, although the differences, on the average, do not exceed 5 dB, vary as a function of stimulus frequency, and are not consistently observed for all subjects (Hall et al, 1982a and b; Jerger et al, 1978b; Møller, 1962). Reflex amplitude, in contrast, is almost always larger for ipsilateral than contralateral stimulation, even with matched, dual probe acoustic transducers (Hall, 1982a, b, and c; Jerger et al, 1978b; Møller, 1962), and it is larger still for binaural stimulation (Møller, 1962; Reker and Baumgarten, 1981). Likewise, latency is relatively shorter in the ipsilateral mode. There are no comprehensive studies of the interaction of stimulus presentation mode with reflex decay. Alberti's (1978) findings suggest that, in normal ears at least, excessive decay is less common with ipsilateral stimulation. Borg and Ødman (1979), however, reported no ipsi- vs contralateral differences in decay for normal hearers.

Stimulus *frequency* is an important factor in all reflex measurements, and the result of basic investigations offer valuable guidelines for selection of test frequency in clinical assessments. First, reflex threshold and amplitude measures vary as a function of stimulus *bandwidth.* The effect of bandwidth on reflex threshold is particularly well-studied and, in fact, forms the basis for a variety of hearing loss prediction methods (see Hall, 1980 for details). There is universal agreement that acoustic reflex threshold levels improve when the activating stimulus exceeds a critical bandwidth, resulting in a difference of up to 20 dB for pure tones vs Gaussian noise stimuli (Djupesland and Zwislocki, 1973; Flottorp et al, 1971; Popelka et al, 1976). Acoustic reflex amplitude is greater for noise than tonal stimuli (Hall, 1982b; Møller, 1962; Silman et al, 1978; Wilson and McBride, 1978). The prevalence of spuriously abnormal acoustic reflex decay findings increases directly with frequency (Alberti, 1978; Jerger et al, 1974c), with up to 25% of a normal population yielding a false-positive (for retrocochlear lesion) outcome at 4000 Hz. Reflex latency appears to be comparable for very brief duration (25 msec) stimuli of differing frequencies (Møller, 1962), but may be longer for low than high frequencies for durations of 200 msec or more (Hung and Dallos, 1972; Møller, 1958; Ruth and Niswander, 1976). Increased variability of reflex threshold and latency parameters for higher frequency stimuli can confuse this relationship (Djupesland et al, 1973).

As noted earlier in this chapter, stimulus *duration* affects acoustic reflex parameters. Threshold levels increase as the stimulus is shortened to less than 200 msec but are independent of changes in stimulus durations exceeding 200 msec (Djupesland and Zwislocki, 1973). Rate of acoustic reflex amplitude growth also varies with stimulus duration when studied with specially constructed apparatus having minimal time constant characteristics (Jerger et al, 1977). As an aside, slope and maximum reflex growth also seem to be dependent on probe tone frequency (e.g., 220 vs 660 Hz) in normal hearers. This relationship, not unexpected (Møller, 1983), requires further study (Sprague et al, 1981). In summary, then, basic studies suggest that the clinical value,

validity, and feasibility of combined ipsilateral *and* contralateral reflex measurements (threshold, amplitude, latency, and decay) are generally enhanced with the use of pure tone stimuli of adequate duration (200 msec) in the midaudiometric frequency region (500 through 2000 Hz).

Subject Characteristics. The influences of *age* and *sex* pervade virtually all aspects of auditory function, including pure tone sensitivity (Bunch, 1929), speech understanding (Pestalozza and Shore, 1955; Hall, 1983), auditory brainstem response (Jerger and Hall, 1980), and measures of static middle ear immittance (Hall, 1979). Pronounced age and sex effects on acoustic reflex parameters, then, are not surprising. Age differentially influences acoustic reflex thresholds for tonal vs noise stimuli in children and adults (Jerger et al, 1972; Jerger et al, 1978a; Hall and Weaver, 1979). In general, reflex thresholds for tonal stimuli appear to remain stable or to improve slightly, with advancing age, while noise-elicited threshold levels may show an increase. Sex is apparently not a factor. The exact interactions among age, reflex thresholds, and stimulus bandwidth, however, are not clear and, indeed, continue to be a rather controversial issue (Handler and Margolis, 1977; Jerger et al, 1978a; Jerger, 1979; Silman, 1979a and b; Thompson et al, 1980). Aging profoundly influences acoustic reflex amplitude (Hall, 1982a; Silman and Gelfand, 1981; Wilson, 1981). Hall (1982a) found that over the range of 20 through 80 yr, maximum reflex amplitude decreased by an average of 56%. These age-related amplitude changes are usually more pronounced for uncrossed (ipsilateral) than crossed acoustic reflexes (Gersdorff, 1978; Hall, 1982a). The basis for the age effect is unknown, although reasonable neurophysiologic explanations have been offered (Hall, 1982a; Wilson, 1981). There is, in addition, an interaction between age and sex. In younger subjects (20 to 30 yr), males and females show equivalent acoustic reflex amplitude values, while in subjects aged 60 to 80 yr, average maximum amplitude in males is less than 75% of female amplitude (Hall, 1982a). Again, no definitive cause for the age-sex interaction has been demonstrated. There are no comprehensive published investigations of acoustic reflex latency on decay measures as a function of age and sex.

Peripheral Auditory Status. Middle ear and sensorineural status are very important factors in the interpretation of acoustic reflex findings in central auditory dysfunction. As noted earlier in this chapter, the acoustic reflex is characteristically not observed by immittance measuring techniques with the probe in an ear with clinically significant *middle ear pathology* (Jerger et al, 1974a; and many others). However, even relatively minor middle ear dysfunction, such as high static compliance or slight (−50 to −100 mm water) negative middle ear pressure, may contribute to abnormally raised acoustic reflex threshold levels (Hall and Weaver, 1979). The clinical implications of middle ear status and acoustic reflex amplitude measurements are profound (Hall, 1982b; Wilson, 1979). For example, in patients with clinically minor negative pressure or static compliance aberrations reflex amplitude is, on the average, only one-third of the amplitude in control subjects (Hall, 1982b; Jerger et al, 1978b), even when there is no evidence of middle ear impairment by pure tone audiometry. Successful clinical application of reflex amplitude measurements in patients with such subtle acoustic immittance aberrations requires careful documentation of middle ear status by history, otologic examination, and immittance audiometry. Also, the pattern of uncrossed and crossed reflex amplitude findings must be examined closely. Normal and bilaterally equal uncrossed amplitudes argue against a spurious middle ear effect. However, reflex amplitude abnormalities with a common probe (measurement) ear suggest confounding middle ear pathology. Again, the possible deleterious influence of middle ear abnormalities on reflex latency and decay has, to my knowledge, not been studied.

There are demonstrated interactions among acoustic reflex findings, stimulus bandwidth, and sensorineural hearing sensitivity loss. For *cochlear hearing impairments*, reflex threshold levels for tonal activators may show slight improvement in patients with mild to moderate (up to 45 dB HTL) deficits, in comparison to normal hearers, and then gradually worsen with increased loss (Hall and Weaver, 1979). Reflex

activity often cannot be elicited with maximum acoustic tonal stimulus intensity levels (110 to 125 dB) in hearing impairment exceeding 70 dB HTL (Jerger and Jerger, 1974). Reflex threshold levels for noise stimuli, on the other hand, increase rather systematically with cochlear hearing impairment (Hall and Weaver, 1979). Eighth nerve auditory dysfunction, even with mild sensitivity loss, is associated with grossly elevated or unmeasurable, acoustic reflex threshold levels for tone or noise stimuli (Anderson et al, 1969; Jerger et al, 1974c; Jerger and Jerger, 1974).

Reports of acoustic reflex amplitude in sensorineural hearing loss are conflicting. Some investigators found decreased amplitude for contralateral stimuli in sensorineural (cochlear) hearing loss (Beedle and Harford, 1973; Peterson and Lidén, 1972; Sprague et al, 1981; Thompson et al, 1980; Wilson, 1981) but did not assess ipsilaterally stimulated reflex amplitude. Others reported varied findings for contralateral amplitude measures (Silman et al, 1978; Silman and Gelfand, 1981). Complicating interpretation of the outcome of these studies are the combined influences of age and stimulus frequency. In systematic investigations of simultaneously stimulated crossed and uncrossed reflex amplitude, Hall (1982b and c) and Jerger et al (1978b) described the relationship of amplitude, stimulus, and age. In young adult subjects (20 to 30 yr), amplitude was generally equivalent in normal vs sensorineurally impaired ears. In older subjects (60 to 80 yr), in contrast, reflex amplitude was significantly depressed for a noise band stimulus, but not a tonal stimulus (4000 Hz), when compared with a normal hearing age-matched control group (Hall, 1982b). As illustrated in the next section and demonstrated by others (Hayes and Jerger, 1983; Mangham and Miller, 1979), *neural auditory dysfunction* characteristically produces greatly reduced acoustic reflex amplitude values. Latency is apparently prolonged abnormally in retrocochlear (vs cochlear or normal) ears (Mangham and Miller, 1979; Mangham et al, 1980; Clemis and Sarno, 1980), although the validity of these conclusions has recently been questioned (Jerger and Hayes, 1983). Of all the reflex parameters, decay has been most extensively studied and clinically applied in sensorineural hearing loss. A number of studies have shown that excessive (greater than 50%) reflex decay provides early and yet strong evidence of retrocochlear auditory pathology (Anderson et al, 1969; Jerger et al, 1974c; Johnson, 1977; and others), even in patients with normal reflex threshold findings.

Drugs. CNS-acting drugs may influence acoustic reflex activity. Elevated threshold levels and decreased amplitudes were found with blood *alcohol* (*ethanol*) concentrations of 0.07 to 0.13% (Borg and Møller, 1967; Cohill and Greenberg, 1977; Robinette and Brey, 1978). Ipsi- and contralateral reflexes were influenced similarly, although the effects varied substantially among subjects. CNS depressants, particularly barbiturates, also suppress acoustic reflex activity (Borg and Møller, 1967, 1975; Bosatra et al, 1975; Giacomelli and Mozzo, 1964; Richards et al, 1975; Robinette et al, 1974; Thompson et al, 1984; Wersäll, 1958). The magnitude of the effect is dependent on the type of drug (e.g., pento-, pheno-, or secobarbital), the dosage, and the subject material (man vs experimental animals, such as cat, rabbit, and monkey). There is evidence that the effect is greater for the contralaterally stimulated reflex (vs ipsilateral) (Borg and Møller, 1967). Ketamine, an anesthetic agent acting on the CNS in a different manner than the barbiturates, also exerts a strong suppressive influence on reflex activity, even at low, preanesthetic doses (e.g., 5 mg/kg) (Thompson et al, 1984).

The pronounced influence of barbiturates on clinical measurements of acoustic reflex activity is illustrated in Figure 7.2, and related to neurologic status and auditory evoked response findings in Table 7.1. Data were obtained serially, by the author, from a 38-yr-old male during withdrawal from induced barbiturate coma. Barbiturates (pentobarbital, Nembutal) were administered therapeutically in management of intracranial pressure due to brain swelling secondary to an acute hypertensive cerebral insult. Acoustic reflex activity was not observed with barbiturate blood levels of 3.62 down to 1.50 mg/dl (Fig. 7.2) and was not consistently normal until there were no detectable barbiturates in the blood. Emergence of measurable acoustic reflex activity

followed the reappearance of neurologic signs (pupillary response and corneal reflex) and the auditory middle latency evoked response. The auditory brainstem response, in contrast, was reliably recorded and consistently normal appearing throughout barbiturate coma. The acoustic reflex, then, is highly sensitive to the CNS depressant effect of barbiturates, more so than other electrophysiologic auditory measures and clinical neurologic brainstem responses. These data provide further evidence that the acoustic reflex arc in man includes important indirect pathways and perhaps, as suggested by Borg (Borg, 1973; Borg and Møller, 1967, 1975), includes reticular formation components.

CENTRAL AUDITORY DYSFUNCTION

Acoustic reflex measurements have clinically proven value in the identification and

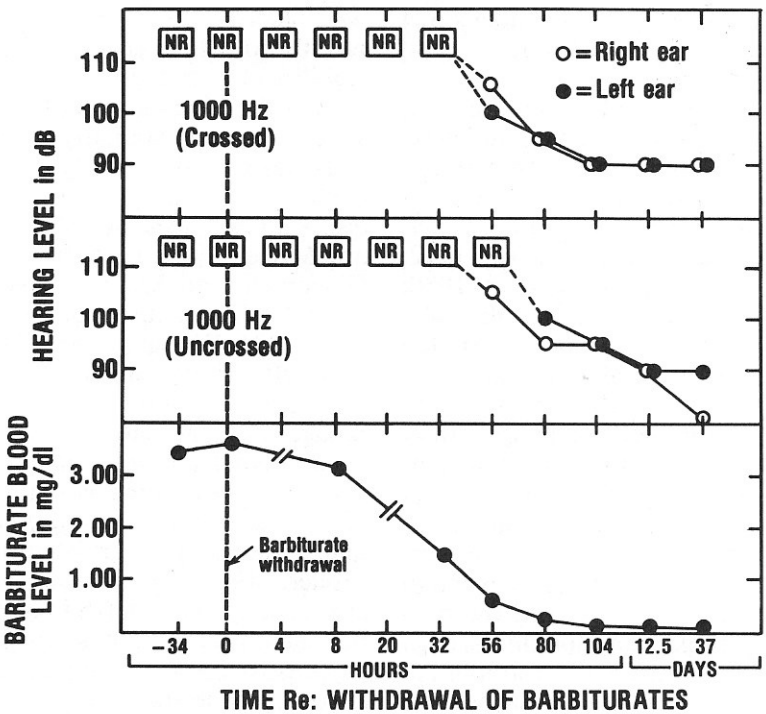

Figure 7.2. Acoustic reflex threshold levels in relation to barbiturate blood levels in a 38-yr-old male subject. Neurologic data and other auditory findings are provided in Table 7.1.

Table 7.1
Correlation of Acoustic Reflex Findings with Neurologic Signs and Auditory Evoked Responses for a 38-yr-old Male during Emergence from Barbiturate Coma[a]

Time postbarbiturate withdrawal (hr)	−34	0	4	8	20	32	56	80	104	300
Barbiturate blood level (mg/dl)	3.34	3.62	NA[b]	3.09	NA	1.50	0.57	0.18	0	0
Pupil reactivity	−	−	+/−	−	+/−	+	+	+	+	+
Corneal reflex	−	−	−	−	+/−	+/−	+/−	+	+	+
Auditory evoked response										
Brainstem	+	+	+	+	+	+	+	+	+	+
Middle latency	−	−	−	+/−	+/−	+/−	+/−	+	+	+
Acoustic reflex	−	−	−	−	−	−	+/−	+/−	+	+

[a] +, normal response; +/−, abnormal response; −, no response.
[b] Data not available.

localization of central auditory pathology. At least three factors contribute to their usefulness in diagnostic audiology. They are: (1) recognition of the patterns of acoustic reflex findings associated with different sites of lesions; (2) an understanding of the relationship among outcomes for acoustic reflex measurements, diagnostic speech audiometry, and the auditory brainstem response; and (3) routine use of reflex measures as a part of a basic audiology test battery. In this section, literature on the clinical application of acoustic reflexes in central auditory nervous system pathology is reviewed. Characteristic patterns of findings are illustrated and correlated with other diagnostic audiometric results in a series of case studies.

A dominant diagnostic role of acoustic reflexes is in the differentiation of cochlear vs retrocochlear lesions. As a result of their neuroaudiologic sensitivity and time effectiveness (Jerger and Jerger, 1983), acoustic reflex threshold and decay are widely used as primary measures of eighth nerve functional integrity (Anderson et al, 1969; Freyss and Casteran, 1979; Hirsch and Anderson, 1980a and b; Jerger and Jerger, 1974a; Jerger et al, 1974c; Jerger et al, 1975; Johnson, 1977; Lidén and Korsan-Bengtsen, 1973; Olsen et al, 1975, 1981; Sanders et al, 1974; Sheehy and Inzer, 1976; Sterkers et al, 1980; Thomsen and Terkildsen, 1975). Analysis of latency and amplitude parameters has further contributed to this diagnostic application of the acoustic reflex (Clemis and Sarno, 1980; Jerger and Hayes, 1983; McPherson and Thompson, 1977; Mangham et al, 1980).

Auditory abnormalities in intracanalicular eighth nerve lesions are ipsilateral, affecting peripheral afferent function exclusively, and are, therefore, not within the scope of this chapter. Lesions arising from within the region of the cerebellopontine (CP) angle or extending into this region from the internal auditory canal are termed extrinsic, or extraaxial, brainstem lesions. This dichotomy is, in part, artificial, distinguished in some patients only by millimeters in lesion location and size or months in time of progression. In clinical audiometry, it is usually not possible to differentiate prospectively between pure eighth nerve lesions and extrinsic brainstem lesions. Unfortunately, many clinical reports on auditory findings, including acoustic reflexes, in brainstem pathology do not even clearly define their patients on the basis of **intrinsic brainstem** vs. **extrinsic brainstem**/retrocochlear **pathology** (see Jerger and Jerger, 1975a). The following section, then, is a discussion of acoustic reflex findings in patients with brainstem level central auditory dysfunction in general. Every attempt, however, will be made to distinguish patterns of findings associated with intrinsic vs extrinsic brainstem sites of lesions.

Intrinsic (Intraaxial) vs Extrinsic (Extraaxial) Brainstem Pathology

Griesen and Rasmussen (1970) first reported clinical evidence of **contralateral (crossed) acoustic reflex** abnormalities in brainstem pathology. Their observations were subsequently confirmed repeatedly with clinical and experimental investigations (Alberti et al, 1977; Borg, 1971, 1973, 1977, 1982a; Bosatra, 1977; Bosatra and Russolo, 1976; Bosatra et al, 1975; Brask, 1978; Colletti, 1975; Hannley et al, 1983; Hayes and Jerger, 1983; Jerger and Jerger, 1974b; Jerger and Jerger, 1981; Jerger et al, 1979; Jerger, 1980; Lehnhardt, 1973, Lehnhardt et al, 1982; Lidén and Korsan-Bengtsen, 1973; Nagafuchi, 1982; Russolo and Poli, 1983; Steinberger and Lehnhardt, 1972; Stephens and Thornton, 1976). Three factors have contributed importantly to the continued interest and success in this application of acoustic reflexes.

First, Borg's (1973, 1982a) correlations of acoustic reflex abnormalities with carefully defined experimental lesions of brainstem auditory nuclei and pathways in rabbits provided much needed basic neuroanatomic information for meaningful interpretation of clinical reflex findings. For example, lesions in the ventral cochlear nucleus (but not the dorsal nucleus) in the vicinity of the eighth nerve root produced elevated reflex threshold levels and excessive reflex decay with ipsilateral stimulation. These findings would seem to be indistinguishable from pure eighth nerve dysfunction. In contrast, trapezoid body lesions spare ipsilateral reflex activity. They may cause only modest increases in crossed reflex thresholds, with no decay, yet produce pronounced latency and amplitude abnormalities. Subsequent total transection of the intermediate and dorsal

stria, on the other hand, created no additional deficits. The medial portion of the superior olivary complex (SOC) is a major structure on both crossed and uncrossed reflex anatomy. Further experimental study of the neuropathologic correlates of acoustic reflex abnormalities will, no doubt, significantly enhance their clinical value.

Second, since the mid-1970s, it has been clinically feasible to measure contralateral *and* **ipsilateral (uncrossed) acoustic reflexes**, with commercially available instrumentation. Analysis and interpretation of the pattern of reflex findings for the four possible measurement conditions (ipsi- and contrastimulus X right and left ears) permits confident differentiation among afferent (sensorineural), efferent (middle ear or facial nerve), and central (brainstem) auditory dysfunction and substantially increases the specificity of reflex data. Clinical correlates of acoustic reflex patterns are described in detail in recent publications (Jerger, 1980; Jerger and Jerger, 1977; Jerger and Jerger, 1981; Jerger et al, 1980), and are illustrated in the following case studies.

Third, measurement of acoustic reflex amplitude, decay, and latency parameters has supplemented threshold information and has heightened sensitivity of acoustic reflexes to brainstem pathology. Amplitude decrements for crossed acoustic reflexes may be found in experimental and clinical brainstem auditory lesions, which do not influence reflex threshold levels (Borg, 1973; Bosatra et al, 1975; Brask, 1978; Hayes and Jerger, 1983). Similiarly, there are clinical reports of excessive decay of acoustic reflex amplitude in intraaxial brainstem pathology, in the absence of eighth nerve involvement and reflex threshold abnormalities (Borg, 1982a; Hannley et al, 1983; Jerger and Jerger, 1977; Stephens and Thornton, 1976), although this is certainly not an invariable sign of brainstem dysfunction (Russolo and Poli, 1983). Abnormally prolonged acoustic reflex latency also appears to be associated with brainstem pathology (Borg, 1973, 1982a; Bosatra et al, 1975; Colletti, 1975; Jerger and Jerger, 1977; McCandless and Harmer, 1975). Recent study of this phenomenon, however, suggests that reduced reflex amplitude may account for the apparently increased latency values (Jerger and Hayes, 1983). In short, suprathreshol measures have added a new dimension t acoustic reflex application in the identifica tion and localization of brainstem auditor pathology.

CASE STUDIES

Case 1

Cerebellopontine angle tumor. The patient wa a 7-yr-old female whose chief complaint was a awkward and stumbling gait and difficulty in self feeding. Physical examination indicated cerebel lar ataxia and 6th and 7th cranial nerve paresi on the left. CT scan showed a mass in the poste rior fossa on the left, with questionable compres sion of the anterior-lateral aspect of the 4th ven tricle on the left. Electroencephalography wa normal. Clinical impression was a left cerebello pontine angle tumor. This diagnosis was late confirmed surgically.

As shown in Figure 7.3, pure tone sensitivit was generally within normal limits and symmet ric, with the exception of thresholds at 8000 H on the right and 250 Hz on the left. Performanc intensity (PI) functions for kindergarten phonet ically balanced word lists showed good maximun scores bilaterally, but borderline-normal rollove (20%) on the right ear, and a dramatic decreas in performance with increased intensity above 4 dB on the left. Tympanometry and static compli ance values were normal. Acoustic reflex meas urement yielded the "inverted L" pattern of ab normalities (see Jerger and Jerger, 1977, 1981) with only the right uncrossed reflexes observed ABR findings are also illustrated in Figure 7.3 Each wave form is the averaged response to 200 clicks at 85 dB HL. With right ear stimulation there is a distinct wave I, II, and III, and no late waves. Only wave I was observed with left ea stimulation.

Acoustic tumors in young children are no common. Yet, even basic audiologic find ings in case 1 provided powerful evidence o retrocochlear dysfunction, thus confirming the neurologic impression. Furthermore, the pattern of audiologic findings suggested that the tumor was large and not within a pri marily eighth nerve site. That is, the left ea results of normal hearing sensitivity and PB max and a well-formed ABR wave I com ponent with normal latency argued for rel atively preserved eighth nerve function. The "L"-shaped acoustic reflex pattern, the con tralateral (right-sided) abnormalities fo ABR, and, to a lesser extent, PI-PB, coupled

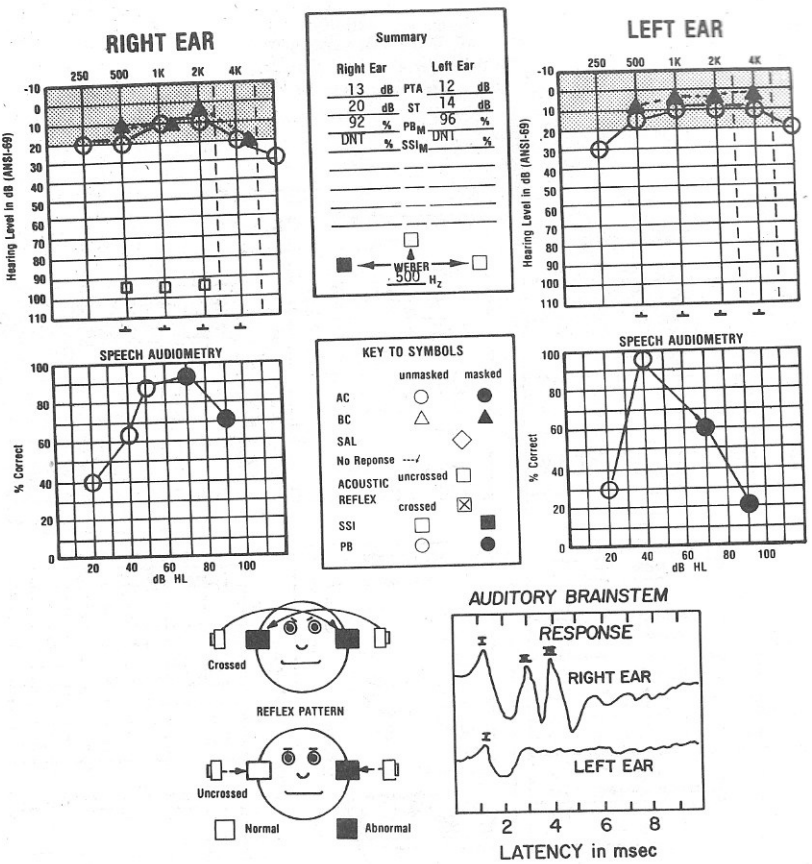

Figure 7.3. Audiometric findings for case 1, a 7-yr-old female with a left cerebellopontine angle tumor. Note evidence of bilateral brainstem dysfunction by acoustic reflex and auditory brainstem response measures.

with the highly significant PI-PB rollover and the absence of an ABR, except wave I on the left, are consistent with a tumor producing a direct effect on pontine auditory brainstem structures, plus contralateral brainstem compression.

Case 2

Vertebral basilar artery transient ischemic attacks. A 49-yr-old female was admitted to the hospital with a 26-yr history of hypertension, treated with multiple drugs, and recent difficulty in hearing with the telephone at the left ear and in understanding speech in crowds. Chief complaints included vertigo, nausea, and tinnitus. On admission in the emergency room blood pressure was 220/140. Rotatory and lateral nystagmus on right lateral gaze was noted. Initial medical impression was vertebral basilar transient ischemic attacks, although peripheral vestibular disease was also considered.

Audiometric findings, obtained 5 days after hospital admission, are shown in Figure 7.4. Hearing sensitivity was within normal limits (*shaded region*), with the exception of a mild notching deficit in the 4000 Hz region on the left. A slight rising configuration, however, was apparent. There was no air-bone difference in pure tone thresholds. Speech audiometry performance for word (PB) recognition and synthetic sentence identification (SSI) tasks was excellent bilaterally. Immittance audiometry yielded normally shaped tympanograms (type A) in each ear, with static compliance values in the low-normal range. Acoustic reflexes were observed under all stimulus conditions, but crossed reflex thresholds for lower frequencies were elevated relative to uncrossed. There was no reflex decay. This horizonal pattern of reflex threshold aberrations is displayed pictorially in the lower portion of the record form.

The single audiometric abnormality for case 2 was a discrepancy between crossed

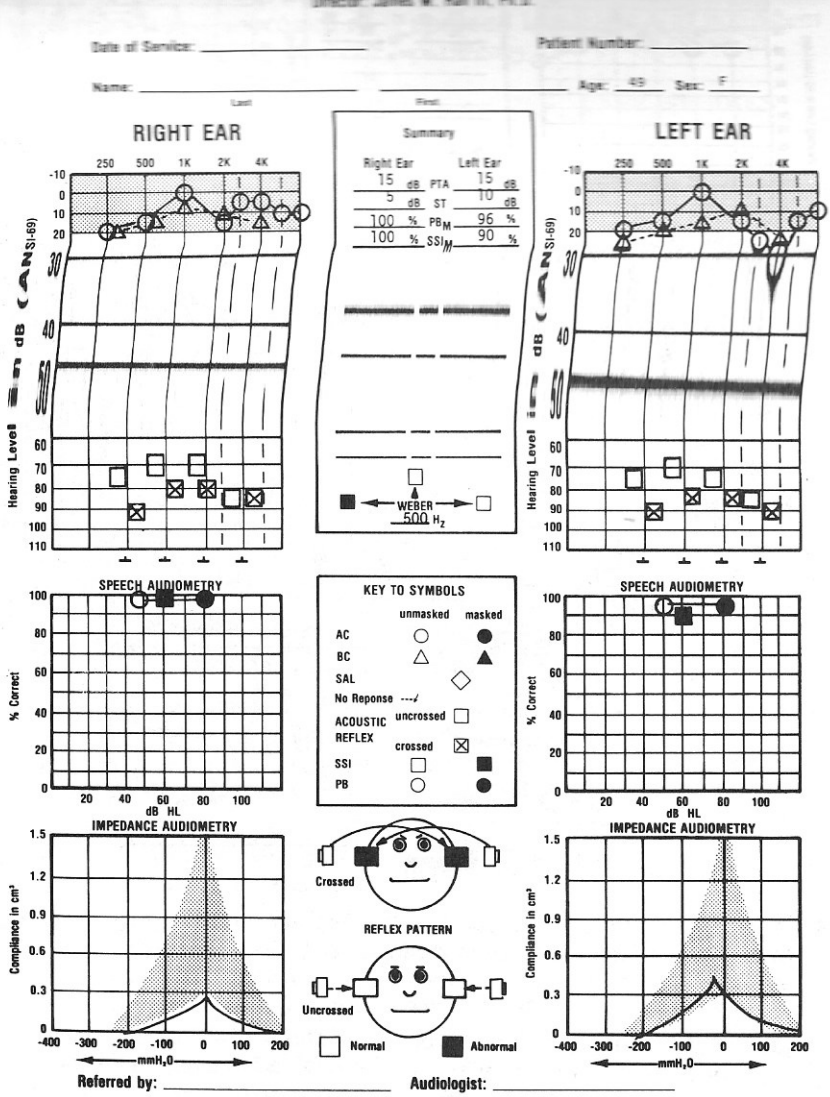

Figure 7.4. Audiometric findings for case 2, a 49-yr-old female with acute hypertension and vertebral basilar transient ischemic attacks. Note discrepancy in crossed vs uncrossed reflex thresholds for 500 and 1000 Hz stimuli.

and uncrossed reflex thresholds. Differential medical diagnosis was not clear-cut, with symptoms characteristic of Ménière's disease, but also not inconsistent with brain stem vascular insufficiency. Crossed acoustic reflex abnormalities in CNS vascular disease at the brainstem level were observed by Bosatra and colleagues (1975). Their patients also presented with complaints of vertigo and both clinical and electronystagmogram (ENG) evidence of spontaneous nystagmus. The reflex pattern in case 2 argues against a purely peripheral labyrinthine disease. The slightly rising configuration is, of course, consistent with the diagnosis of Ménière's disease but not unexpected in central auditory system involvement (Hayes and Jerger, 1979a). This case illustrates the possible utility of acoustic reflexes as a functional correlate of vascular status. Additionally, it illustrates the frequent independence in outcome for different measures of auditory functioning, such as the acoustic reflex vs SSI, and the clinical value of *routinely* administering multiple tests of peripheral and central auditory status.

Case 3

Caudal brainstem stroke. A 57-yr-old male was admitted to the hospital with a history of diabetes mellitus, glaucoma, and hypertension, status postcerebrovascular accident in 1963 with slight residual deficit. Chief complaint was loss of balance and falling to the right, worsening over the past 3 days. Associated symptoms were tinnitus and decreased hearing on the left, hoarseness, and numbness of the right face (fifth cranial nerve). Blood pressure was 190/106. Physical examination on admission was significant for decreased tone and slightly decreased strength on the right, decreased pin prick sensation on left leg, cerebellar signs of dysmetria right, with a wide-based gait, upbeat nystagmus with lateral and rotatary components on lateral gaze, and right vocal cord paralysis. There was a sluggish corneal reflex and central facial deficit on the right and a spastic gag reflex. Diagnosis was right posterior inferior cerebellar artery (PICA) infarction, with medullary and pontine brainstem involvement.

Auditory findings are displayed in Figure 7.5. There was a mild sensorineural hearing impairment in both ears, with a greater deficit for frequencies above 1000 Hz, particularly on the left. Audiometric Weber at 500 Hz was lateralized to the right ear. Maximum speech audiometry performance for word (PB) recognition and synthetic sentence identification (SSI) was excellent on the right and good on the left ear. Excessive rollover (Jerger and Hayes, 1977) was present on the SSI task for the right ear (30%) and left ear (70%). Immittance audiometry showed normal tympanograms and static compliance bilaterally. Acous-

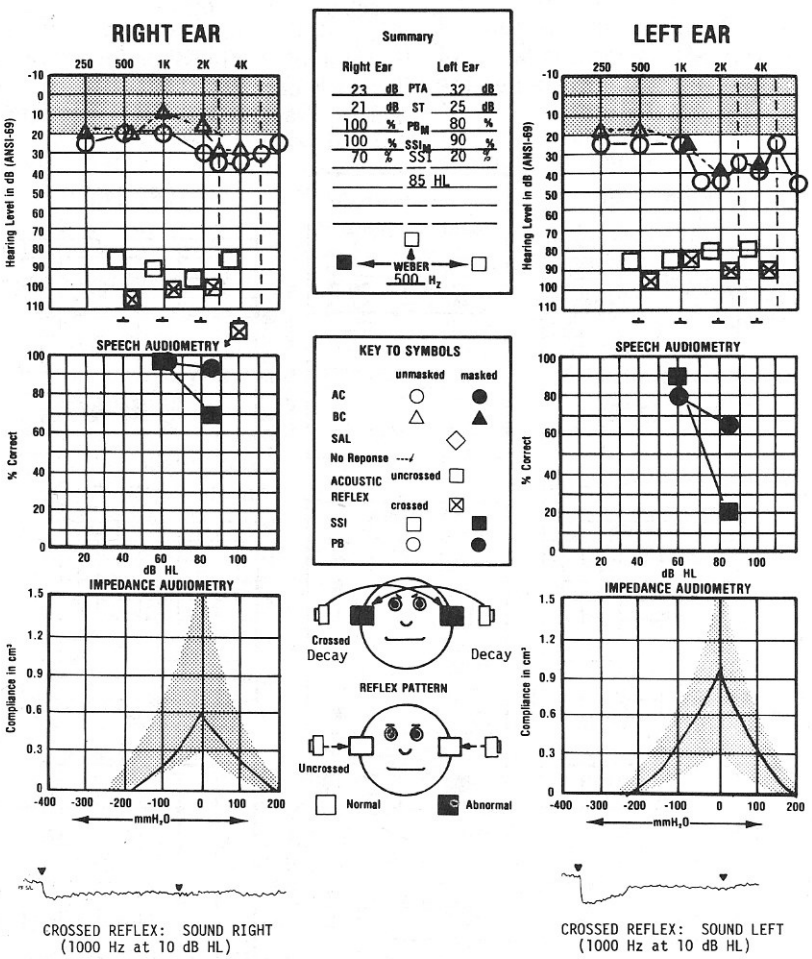

Figure 7.5. Audiometric findings for case 3, a 57-yr-old male with documented history of caudal brainstem stroke. Note excessive decay of crossed acoustic reflex amplitude bilaterally and rollover in speech audiometry for synthetic sentence identification.

tic reflexes were generally observed. There was, however, no measurable reflex for the contralateral condition at 4000 Hz on the right and somewhat elevated low-frequency threshold levels. Notably, abnormal crossed acoustic reflex decay was recorded in both ears (see tracings in bottom portion of Fig. 7.5).

There was abnormal crossed acoustic reflex decay bilaterally and also grossly elevated right to left crossed reflex threshold levels. SSI performance, however, was relatively poorer for the left ear. Consistent with the previous two cases, and with expectations, findings for an incomplete audiologic assessment (pure tone audiometry and word recognition at 40 dB SL) showed no signs of central auditory dysfunction. Yet, the patient clearly had neurologic signs and hearing complaints. Routine application of acoustic reflex and SSI measures were of value in confirming and localizing the source of these clinical deficits.

Case 4

Rostral brainstem stroke. A 58-yr-old fema was admitted to the hospital with the chief com plaint of a "burning" sensation on the right sic of her face and body of 3 weeks duration. Soo after the onset of this sensation, she noted blurre vision and rapid movements of the right ey There was a longstanding history of hypertensio medically controlled. Physical examination re vealed intermittent lateral (left beating) nystag mus in the right eye, decreased sensation on th right face, and suggestion of right-sided moto weakness. Medical impresson was a hypertensiv vascular insult in the rostral pontine-midbrai region.

Audiometric findings are displayed in Figur 7.6. Pure tone testing showed a mild, symmetri high-frequency sensitivity impairment bilaterall Maximum speech audiometry performance wa excellent (92 to 96%) for word (PB) recognitio but poor bilaterally (50%) for SSI materials. Sig nificant rollover in SSI performance was observe for each ear. Performance deficits on the SS ICM at varying message to competition ratio

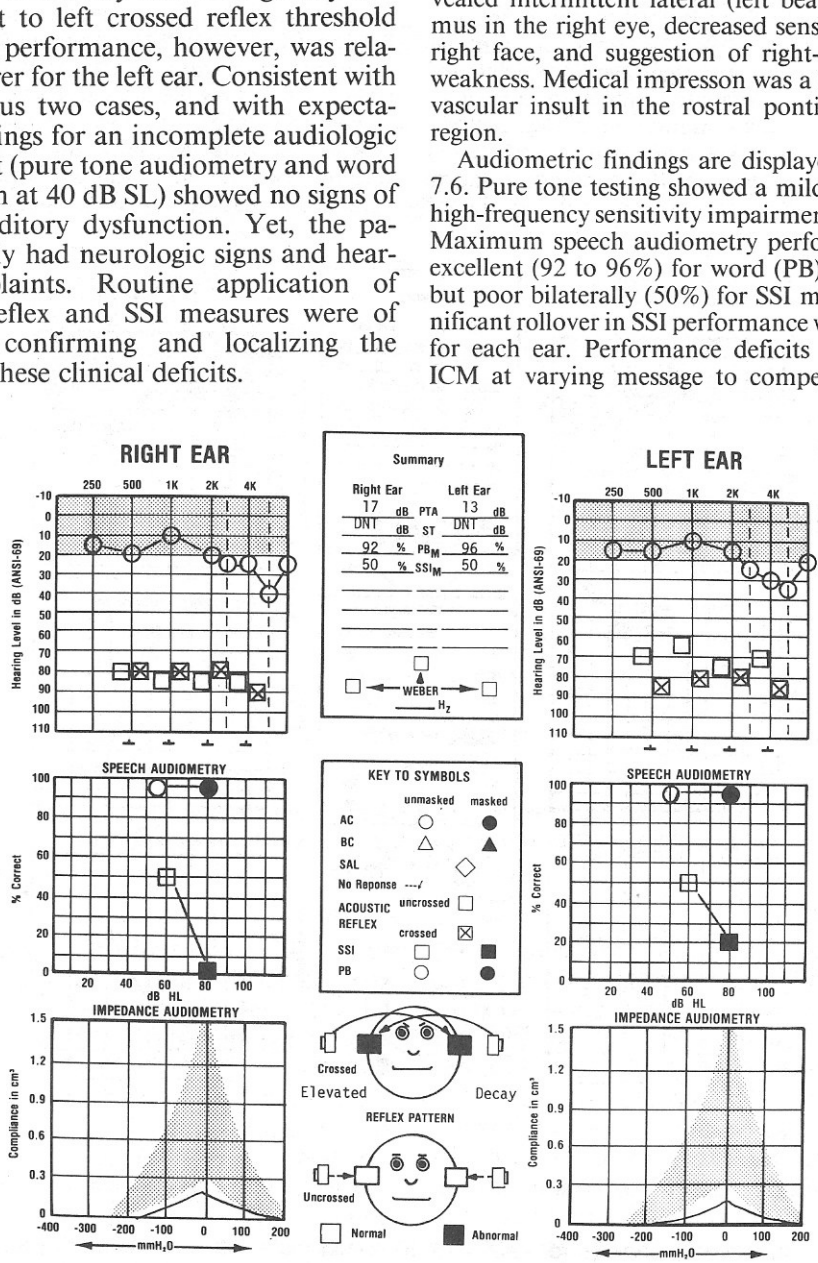

Figure 7.6. Audiometric findings for case 4, a 58-yr-old female with documented history of rostra brainstem stroke. Note "horizontal" pattern of acoustic reflex abnormalities (elevated threshold level and excessive amplitude decay) and depressed performance for synthetic sentence identification.

(not shown) confirmed this finding. Immittance audiometry demonstrated tympanograms that were normally shaped but somewhat shallow. Acoustic reflexes were present in all four measurement conditions. However, crossed reflexes with stimulus-left were elevated relative to the left uncrossed condition, and excessive reflex decay (greater than 50% in 10 sec) was recorded for the crossed stimulus-right condition. In combination, these reflex abnormalities yielded the horizontal pattern, as shown in the lower center portion of Figure 7.6. The ABR was normal for left ear stimulation, but for the right ear the wave I to V latency interval was abnormally prolonged. The auditory middle latency response appeared normal bilaterally.

In this case, neurologic evidence contributed to the diagnosis of a brainstem (pontine-midbrain) vascular insult, and both acoustic reflex and SSI outcome provided evidence of bilateral central auditory system involvement. Reflex abnormalities were in the characteristic horizontal pattern (Jerger and Jerger, 1977), yet were varied for the left (elevated threshold) vs right (excessive decay) ears. Both of these deficits have long been associated with intrinsic brainstem pathology (Bosatra et al, 1975; Griesen and Rasmussen, 1970; Jerger J and Jerger S, 1974a and b, 1975; Jerger S and Jerger J, 1975a, 1977). The differential effect of the brainstem lesion on the two reflex parameters is, however, intriguing.

In combination, cases 2, 3, and 4 illustrate the sensitivity of acoustic reflex measurements to varying degrees of vascular brainstem impairment, ranging from transient ischemic attacks to stroke. This apparent influence of ischemic pathophysiology on reflex activity is clinically exciting and worthy of basic investigation.

Multiple Sclerosis

By definition, neuropathy in multiple sclerosis (MS), a dissemminating demyelinating disease, may have both peripheral and central nervous system components. Consequently, there are clinical reports of eighth nerve and brainstem auditory dysfunction in MS. Studies of acoustic reflex patterns in MS are no exception (Bosatra et al, 1975; Colletti, 1975; Hannley et al, 1983; Hayes and Jerger, 1983; Jerger and Jerger, 1977; Lehnhardt et al, 1982; McCandless and Harmer, 1975; Russolo and Poli, 1983). Indeed, a major clinical contribution of acoustic reflex measurements in this population is the early confirmation of peripheral *and/or* central nervous system involvement, and the rather precise localization of brainstem lesions. In addition to these neuroanatomic variations, MS may produce diverse types of reflex abnormalities, including elevated threshold levels (Colletti, 1975; Bosatra et al, 1975; Hannley et al, 1983; Hess, 1979; Jerger et al, 1979; Lehnhardt et al, 1982; Russolo and Poli, 1983), excessive decay (Hannley et al, 1983; Jerger and Jerger, 1977; McCandless and Harmer, 1975), prolonged latency (Bosatra et al, 1975; Colletti, 1975), and reduced amplitude (Hayes and Jerger, 1983).

Case 5

Multiple Sclerosis. The patient was a 24-yr-old female with a 3 yr history of multiple sclerosis. She was referred by the Neurology Service to Audiology for auditory brainstem response assessment. Symptoms included other sensory deficits and abnormal visual evoked responses.

Auditory findings, including ABR data, are shown in Figure 7.7. There was a mild, unilateral sensorineural hearing impairment for the left ear on this test date. Two years before, a complete audiologic assessment, including diagnostic testing (SISI, tone decay, Békésy audiometry, SSI-ICM, SSI-CCM, time compressed speech) failed to reveal any evidence of eighth nerve or central auditory dysfunction, and hearing sensitivity at that time was normal in each ear (PTAs of 3 dB on right and 5 dB on left). As shown in Figure 7.7 speech audiometry performance at this test date was excellent on the right. On the left, maximum SSI scores were depressed relative to word (PB) recognition scores, and there was abnormal rollover for both speech materials. Tympanograms and static compliance values were normal. Acoustic reflexes were observed bilaterally, but thresholds were elevated for all activating stimuli presented to the left ear. In addition, there was excessive decay of crossed acoustic reflexes for both ears, resulting in the "inverted L" pattern (Jerger and Jerger, 1977, 1981) depicted in the lower portion of Figure 7.7. The auditory brainstem response with left ear stimulation was grossly abnormal, with the wave I component prolonged in latency and poorly formed and an excessive wave I to V latency interval (brainstem transmission time). With right ear stimulation, the ABR was reasonably normal in appearance, although the wave V component was delayed in latency, variable in morphology, and noticeably reduced in amplitude (vs waves I, II, III, and IV).

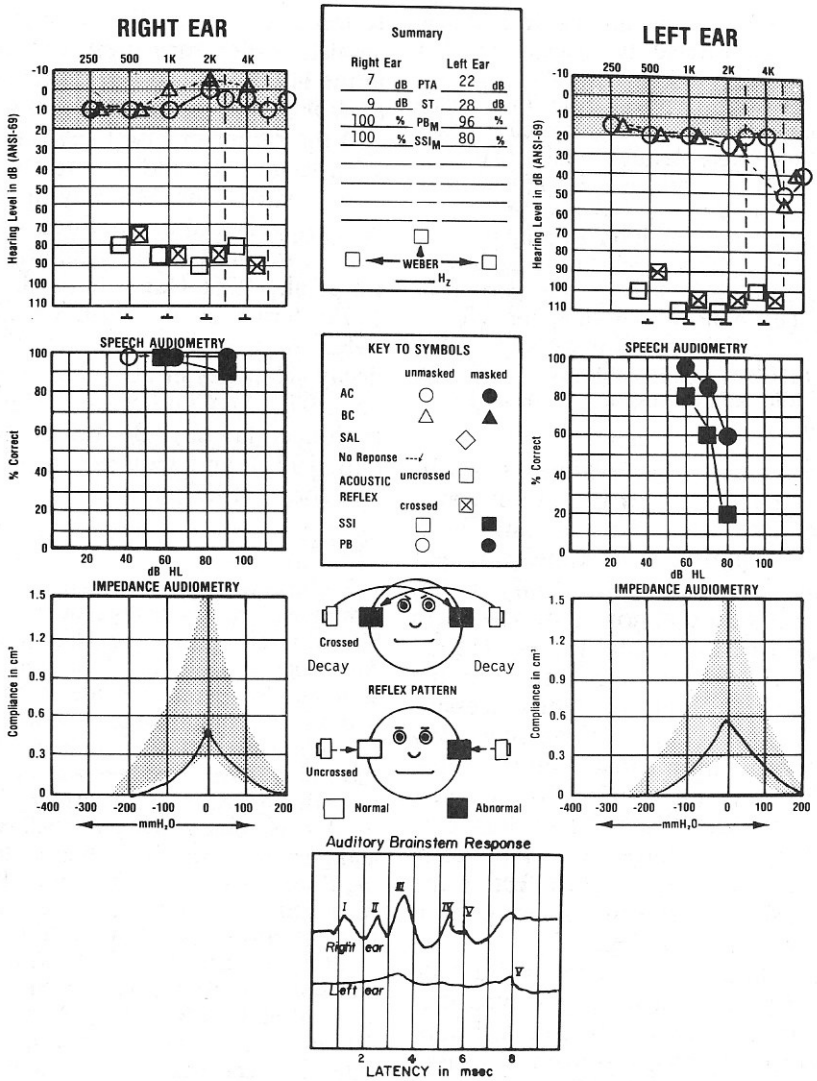

Figure 7.7. Audiometric findings for case 5, a 24-yr-old female with multiple sclerosis. Note "inver L" pattern for acoustic reflex findings, unilateral (left) rollover for speech materials, and primarily ear auditory brainstem response abnormalities.

Acoustic reflex amplitude data for case 5 are plotted as a function of stimulus (1000 Hz) intensity in Figure 7.8. The right uncrossed reflex alone shows normal, systematic amplitude growth with increasing intensity. For the crossed reflex with stimulus-right, the amplitude function is relatively flattened. Strikingly, both crossed and uncrossed reflex amplitude values with stimulus-left are grossly depressed, even at highest intensity levels. This pattern of reflex amplitude abnormalities is a graphic extension of the threshold aberrations noted above and cannot be explained by middle ear afferent or efferent dysfunction.

The audiometric outcome for case 5 is entirely consistent with previously repor findings in MS. The acoustic reflex patte however, contributes uniquely to the loc zation of the lesions. Pure tone and spe audiometry argue for unilateral (left-sid involvement. Abnormal crossed reflex de and decreased amplitude in both ears evidence of bilateral brainstem lesions. T pattern is confirmed by ABR audiomet Once again, these rather clear-cut clini audiometric deficits would have gone u noticed with a simple, routine hearing ev uation consisting of pure tone audiome

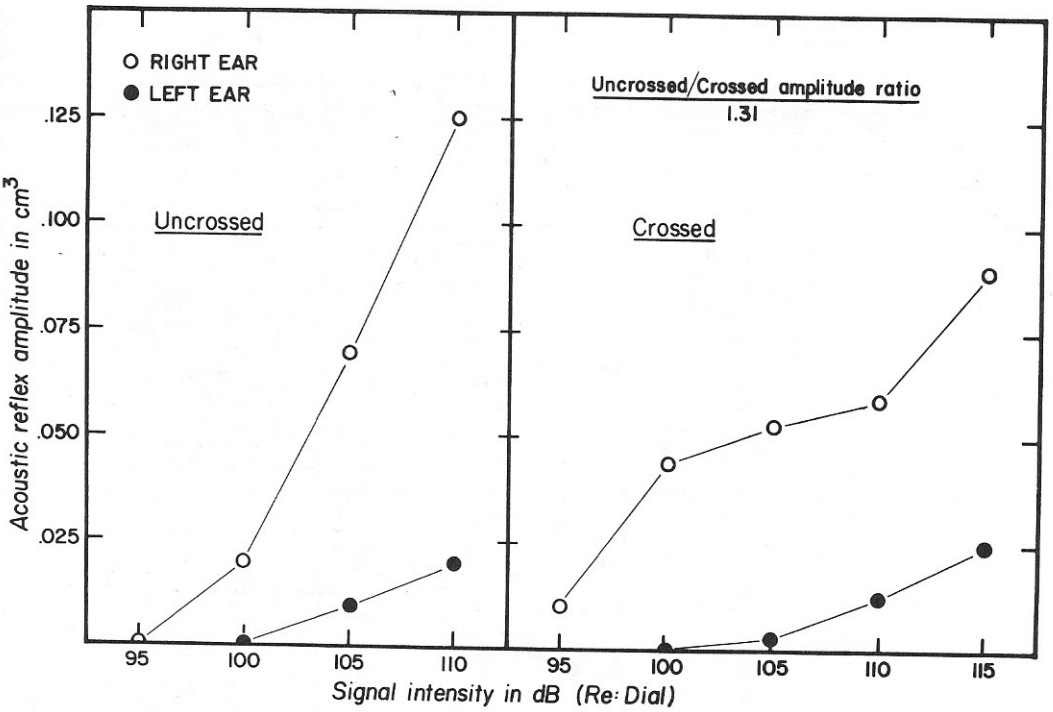

Figure 7.8. Acoustic reflex amplitude as a function of intensity for case 5. Systematic reflex amplitude growth is apparent only in the right ear, uncrossed condition.

and word recognition at a comfortable listening level.

Facial Nerve Dysfunction

By virtue of its efferent anatomy and physiology, the acoustic stapedius reflex is clinically useful in localizing and quantifying seventh (facial) cranial nerve dysfunction (Alford et al, 1973; Citron and Adour, 1978; Draskovich and Szekély, 1984; Koike et al, 1977; Matzui, 1981; Ruth et al, 1978). Unequivocally normal acoustic reflex activity in facial nerve palsy implies that the lesion is distal (peripheral) to the facial nerve branch innervating the stapedius muscle. Absence of measurable acoustic reflexes is a sign of gross facial nerve dysfunction proximal (central) to the stapedial branch. Of primary interest clinically are elevations of acoustic reflex threshold levels, abnormal decay, or a reduced reflex amplitude. These reflex parameters offer an objective, noninvasive means of quantifying and monitoring facial nerve function during progression of or recovery from disease. In any case, the reflexes of interest are with the probe in the ear on the affected side, with contralateral probe reflex measurements serving as control values.

Case 6

Bell's palsy. The patient was a 33-yr-old male with a 1-month history of idiopathic right facial paralysis diagnosed as Bell's palsy. He reported no hearing complaints, no history of otologic disease, tinnitus, or balance disturbances, and was otherwise in good health. Medical therapy consisted of prednisone.

Audiometric findings are illustrated in Figure 7.9. Hearing sensitivity was well within normal limits bilaterally. Audiometric Weber (500 Hz) was referred to midline. There was no consistent difference between air- and bone-conducted pure tone thresholds on the right. Maximum word recognition scores were 100% in each ear, but there was asymmetric PI-PB rollover of 20% on the right. Immittance audiometry yielded normal (type A) tympanograms and static compliance values in the normal range bilaterally. Acoustic reflex thresholds for tonal stimuli were at expected hearing levels (80 to 95 dB) with probe in the left ear, but the crossed and uncrossed reflexes with probe right were relatively elevated (10 to 20 dB). There was no evidence of excessive reflex

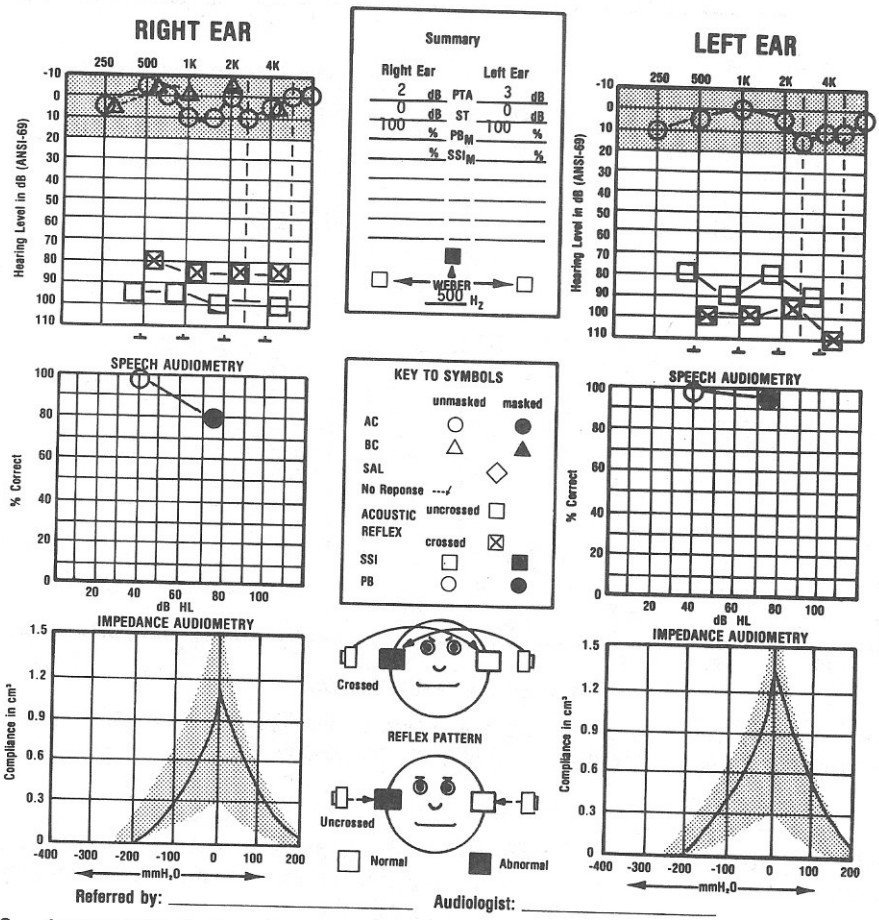

Figure 7.9. Audiometric findings for case 6, a 33-yr-old male with Bell's palsy. Note vertical p of acoustic reflex abnormalities (*right*) and ipsilateral evidence of rollover in word recog performance.

decay. As depicted in the lower center portion of Figure 7.9, these reflex aberrations formed the vertical pattern (right side).

Abnormalities with a common probe (measurement) ear (vertical pattern; Jerger and Jerger, 1977, 1981) reflect disruption of the efferent portion of the acoustic reflex arc. Usually, they result from middle ear pathology, but they may be produced also by facial nerve dysfunction. In case 6, middle ear pathology was effectively ruled out by other immittance findings (symmetrically normal static compliance and tympanograms), by pure tone audiometry (no air-bone gap and midline Weber), and by normal otologic examination. The reflex threshold elevations with probe right were, therefore, secondary to facial nerve dysfunction proximal to the branching to the stapedius muscle, and consistent with the diagno Bell's palsy on the right. The unilatera dence of decreased word recognition ip eral to this dysfunction is probably du the reduced sound attenuation capaci the right stapedial muscle having impa facial nerve innervation. This findir sometimes associated with facial nerve resis (Borg and Zakrisson, 1973).

Age-related Central Auditory Dysf tion. As discussed earlier in this cha age is an important factor in acoustic re measurements. In many cases, age-rel changes in middle ear or sensorineural f tion directly influence activity at the affe or efferent portions of the reflex arc. T is some evidence, however, that integrit the central reflex structures may also involved in the age effect. For exam

Quaranta et al (1980) reported reflex abnormalities in 55% of a presbycusic population and attributed the finding to auditory brainstem aging processes. Supporting this contention was the tendency for abnormalities of crossed, rather than uncrossed, acoustic reflex thresholds. Comparable age effects are documented as well for other measures of central auditory functioning, including the auditory evoked responses (Fujikawa and Weber, 1977; Jerger and Hall, 1980) and complex speech perception tasks (Hall, 1983; Hayes and Jerger, 1979b; Jerger and Hayes, 1977).

Acoustic reflex amplitude-intensity functions are shown for two groups of elderly subjects in Figure 7.10. Subject characteristics are displayed in Table 7.2. Groups were comparable in age, hearing threshold levels, and maximum performance for word recognition. The centrally impaired group, however, demonstrated significant deficits for synthetic sentence identificaton (SSI) with a competing message, both in maximum percent correct score and degree of rollover. Ipsilateral and contralateral acoustic reflexes were simultaneously stimulated and then measured and computer-averaged with specially constructed dual probe apparatus, described in detail elsewhere (Hall, 1982a). Average magnitude of uncrossed and crossed acoustic reflex activity was greater for the control group than the centrally impaired group. The difference is most apparent in the crossed reflex condition. These data suggest that age-related central nervous system changes (Hansen and Reske-Nielsen, 1965) may involve the brainstem auditory structures of the acoustic reflex arc, including, perhaps, the ventral cochlear nucleus region (Borg, 1973; Königsmark, 1969), as evidenced by the ipsilateral reflex magnitude deficit in the central group.

Cerebral Auditory Pathology. Whether or not suprabrainstem regions influence acoustic reflex activity is currently debated. On the one hand, there are case studies documenting normal acoustic reflex thresholds in patients with confirmed, bilateral damage to auditory temporal cortex (Jerger and Jerger, 1975b; Jerger, 1980). Gelfand and Silman (1982) also contend that cerebral damage does not affect reflex thresholds for tonal stimuli. Unfortunately, in this latter, retrospective study there were no control subjects. Furthermore, the site of CNS damage was not specified neuroradiologically, nor was there any documentation of associated neurologic deficits. These serious methodologic deficiencies cast doubt on the stated conclusions of the study. In contrast, Downs and Crum (1980) found abnormally

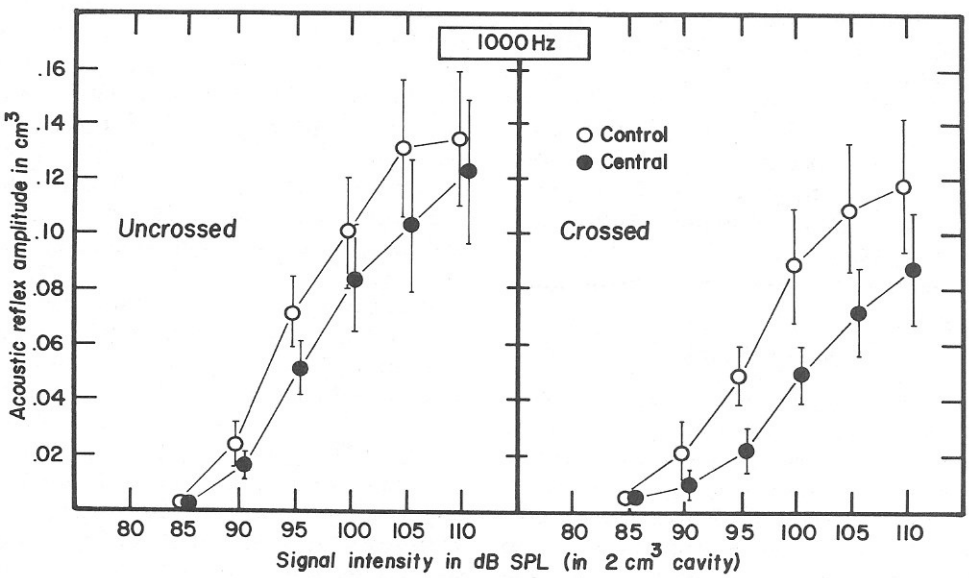

Figure 7.10. Acoustic reflex amplitude intensity functions in elderly subjects with speech audiometry evidence of central auditory impairment and a control group. See Table 7.2 for description of subjects. Note depressed amplitude in centrally impaired group, expecially for crossed acoustic reflexes.

Table 7.2
Characteristics of Seventeen Subjects (Seven Male and Ten Female) with Evidence of Centra Auditory Impairment by Speech Audiometry and Twenty-Three Subjects (Nine Male and Fourteen Female) with No Evidence of Central Auditory Impairment[a]

Group	Index Age (yr)	HTL[b] (dB) (1000 Hz)	Static compliance (cm^3)	PBmax[c] (%)	SSImax[d] (%)	SSI-rollove (%)
No central auditory impairment						
mean	69.2	15.5	0.76	93.0	91.1	2.4
SD	(5.0)	(11.2)	(0.36)	(15.0)	(17.5)	(7.6)
Central auditory impairment						
mean	72.5	15.4	0.64	90.9	65.9	25.0
SD	(5.1)	(9.8)	(0.23)	(9.7)	(20.9)	(22.8)
Difference in mean	3.3	0.2	0.12	2.1	25.2**	22.6**

[a] Audiologic data are averaged for both ears of each subject.
[b] Hearing threshold level (dB) at test signal frequencies of 1000 Hz or 500 to 1500 Hz (noise-banc
[c] Maximum score (%) for Phonetically Balanced (PB) word test.
[d] Maximum score (%) for Synthetic Sentence Identification (SSI) test.
[e] Decrease in SSI performance (%) between maximum score and score at highest speech intensi level.
[f] Difference in mean values between groups is significant at 0.01 level of confidence by *t*-test.

sensitive acoustic reflex threshold activity in four patients with neurophysiologic, neuroradiologic (e.g., computerized tomography), and/or neurologic evidence of cerebral lesions. Also, Peterson and Lidén (1972) described reduced reflex amplitude in a patient with a corpus callosum lesion. Experimental bilateral ablation of auditory cortex reduced middle ear muscle activity in cat (Baust and Berlucchi, 1964), and recent experimental investigations in monkey also provided preliminary evidence of a cortical modulation of suprathreshold acoustic reflex activity (B Stach, personal communication, 1983). The issue requires further experimental study and more carefully conducted clinical exploration to define the potential role of rostral brainstem, midbrain, thalamic, and/or cortical influences on the acoustic reflex.

Other Central Nervous System Dysfunctions

Scattered throughout the literature are reports of acoustic reflex findings in diverse confirmed and suspected central auditory nervous system dysfunctions. The suppressive acute influence of alcohol on acoustic reflex activity was noted previously in this chapter. In a recent study, Spitzer and Ventry (1980) found a significantly higher proportion of inexplicably absent acoustically measured reflexes in a group of *chronic alcoholics* than in an age-matched contr group. Acoustic reflex threshold levels we equivalent for the two groups. The results a diagnostic audiologic test battery, inclu ing these reflex findings, were consiste with brainstem auditory dysfunction.

Acoustic reflex measurements are clin cally useful in patients with *speech-langua and voice problems* of varied CNS etiologie including autism, cerebral palsy, and ment retardation (Keith et al, 1977; Niswand and Ruth, 1977; Suria and Serra-Ravento 1975). The main applications are in predi tion of hearing loss and identification middle ear pathology. There is no conclusiv evidence of reflex abnormalities in these pa tient groups, assuming middle ear functio is unequivocally normal (Keith et al, 1977 Hall and colleagues (Hall and Jerger, 197 1978; Hall, 1981) observed acoustic refle aberrations in two speech-voice disorders unknown etiology. A reflex latency measu (rise time) was longer in a group of si patients with *spastic dysphonia* than an ag and sex-matched control group (Hall an Jerger, 1976). In a subsequent study, u crossed reflex amplitude was curiously larg in a group of 12 spastic dysphonics than i a control group, but crossed reflex amplitu showed no group differences. This devia pattern (greater uncrossed reflex amplitud had previously been associated with brai stem pathology (Bosatra and Russolo, 1976

In addition, reflex amplitude aberrations were reported in *stuttering*, again with reference to control data (Hall and Jerger, 1978). Further investigation of reflex parameters in such patient populations may help to define the etiology and possible neurologic bases of these enigmatic communicative disorders.

In acutely and severely head-injured patients, acoustic reflexes are rarely observed, even in persons with normal middle ear function as determined by otologic examination and other acoustic immittance measures (Hall et al, 1982 a and b, 1983). Only 16% of a series of 25 patients showed evidence of any acoustic reflex activity within 3 days after a head injury defined as severe by the extent of coma. This high proportion of abnormal acoustic reflexes was probably related to their comatose state and neurosurgical management rather than to brainstem structural damage. That is, severe head injury impairing consciousness presumably affects the brainstem reticular activating system. Barbiturate-induced coma, as noted earlier in the chapter, is sometimes employed therapeutically in management of brain pathophysiology secondary to head injury and was used in six of the above patients with no reflex activity. Both findings lend further support to the role of the reticular formation in the central acoustic reflex arc in man.

PROMISING NEW DIRECTIONS FOR ACOUSTIC REFLEX MEASUREMENTS IN CNS PATHOLOGY

In the years since Griesen and Rasmussen (1970) first described reflex abnormalities in brainstem pathology, interest in neurodiagnostic applications has not abated but, rather, increased. This clinical popularity is not surprising. Acoustic reflexes offer a noninvasive, objective, simply administered, and cost-effective measure of eighth (acoustic) nerve, auditory brainstem and seventh (facial) nerve function, available in most audiology facilities (Jerger and Jerger, 1983).

The general diagnostic value of acoustic reflexes is now well-documented, yet the specific neuropathologic correlates of abnormal findings are not adequately defined. Also, pathophysiologic processes affecting acoustic reflex parameters are virtually unknown. Particularly unclear is the influence of brainstem regions not traditionally considered part of the auditory system (e.g., reticular formation) and the suprabrainstem auditory anatomy. These basic and clinically important issues require further study.

The versatility of reflex measurements in central auditory dysfunction has expanded with the clinical application of latency, decay, and amplitude parameters, as well as threshold levels. In the literature, and in this chapter, are clinical cases illustrating the independence of these parameters. That is, the response parameters are characterized by differential sensitivity to brainstem lesions. Put another way, it is likely that different pathologic processes produce distinctive reflex aberrations, some affecting amplitude, others time course (decay), and still others temporal characteristics (rise time or latency). Defining these associations is a worthy objective for future clinical research. Among the response parameters, reflex amplitude especially has shown promise in identification of brainstem dysfunction (Borg, 1973; Brask, 1978). The clinical feasibility of amplitude measures, however, has been questioned on the basis of the troublesome variability of intersubject data, and, consequently, the difficulty in developing meaningful normative values. Recently developed amplitude indices that emphasize the intrasubject crossed vs uncrossed relationship appear to reduce the clinical problem posed by variability (Hall, 1982c; Hayes and Jerger, 1983).

Computer averaging of acoustic reflex activity for a series of stimuli is becoming clinically feasible and probably will contribute to the stability and reliability of measurements in patient populations. Often this is done by interfacing commercially available immittance audiometers with signal averaging evoked response systems. Multipurpose audiometric microprocessors offer another means of averaging acoustic reflexes. In addition to enhancing the sensitivity and reliability of acoustic reflex determinations, signal averaging over a brief time period (less than 500 msec) offers increased accuracy in temporal measurements, and data storage capacity with some microcomputers permits archiving and facilitates intersubject comparisons of reflex findings.

Finally, because it is noninvasive, cost-

effective, and brief, acoustic reflex measurement is well-suited for serially monitoring auditory brainstem status, or eighth and seventh cranial nerve function, over time. This is likely to be a particularly important clinical application in dynamic neurologic pathologies, including multiple sclerosis, Bell's palsy, and even neuromuscular disorders, such as myasthenia gravis (Kramer et al, 1981; Neff et al, 1980). The extreme susceptibility of the acoustic reflex to the influence of many CNS depressants, such as barbiturates, suggests a potentially valuable application in anesthetic monitoring. In summary, the role of acoustic reflex measurements in neuroaudiology appears secure, and, indeed, will probably expand with further basic science and clinical investigation.

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