

Disturbances of Loudness Perception

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Abstract

This article reviews information on some auditory disorders that have in common a disturbance in loudness perception. The perceptual disturbances in these disorders have interchangeably been labeled "hyperacusis," "dysacusis," or "phonophobia." Our question concerns whether the loudness disturbances associated with these auditory disorders are sufficiently different as not to justify the equivalence implied by the labelling. Emphasis is placed on those articles that have given clear accounts of the phenomenology of the disturbed perceptual experience and have offered testable hypotheses about the mechanisms underlying it. Hypotheses about the origins of disturbed loudness perception are compared with independent experimental and clinical evidence on those mechanisms. The disturbances of loudness perception that occur in cochlear hearing loss, facial nerve paralysis and stapedectomy, and in more "central" disorders are phenomenologically different, have different underlying mechanisms, and merit different labels that most of them do not currently receive.

Key Words: Facial nerve paralysis, hyperacusis, loudness perception, phonophobia, recruitment, stapedectomy

Abbreviations: ABLB = alternate binaural loudness, LDL = loudness discomfort level

Disorders of loudness perception have received little attention, and this is surprising for a number of reasons. First, the basic sciences of cochlear physiology and auditory perception have advanced to a stage where the neurophysiologic underpinnings of loudness perception are relatively well understood. Second, although there has been a wealth of evidence and opinion reported relevant to the genesis of uncomfortable loudness in facial nerve paralysis, there has been little attempt to evaluate it critically. Third, there has been a proliferation of reports of apparent loudness discomfort in conditions not known to affect the auditory periphery (see esp. Marriage and Barnes, 1995). In these and a number of other disorders, the same labels have been assigned interchangeably to the perceptual phenomena, viz., "hyperacusis," "phonophobia," and "dysacusis" (e.g., Perlman, 1938;

Tschiassny, 1949; Mathison, 1969; Carman, 1973; Citron and Adour, 1978; McCandless and Schumacher, 1979; Gordon, 1991; Jastreboff and Hazell, 1993; Nigam and Samuel, 1994; Bettison, 1996). This interchangeability implies some form of commonality to the perceptual disorders, and does so when it is far from clear that those disorders actually have similar phenomenologies or physiologic underpinnings.

The purpose of the present article is simply to survey the major evidence on these disorders of loudness perception, drawing together, where possible, experimental and clinical observations into coherent descriptions of the perceptual phenomena themselves and of their origins. We begin with an overview of loudness recruitment that occurs in cochlear hearing loss to introduce the neural basis of perceived loudness and its modification in a well-studied pathology. We turn then to examine loudness discomfort that occurs in facial nerve paralysis and stapedectomy. Finally, we examine the aversion to sounds seen in a variety of "central" neural disorders that have in common a bearing on a central hyperexcitability hypothesis. In what follows, our thesis is that the loudness disorders seen in cochlear hearing loss, facial nerve dysfunction, and the more "central" pathologies are fundamentally

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different phenomena that merit different labels and further investigation in their own rights.

LOUDNESS RECRUITMENT

Loudness recruitment is an abnormally steep loudness growth for given increments in stimulus intensity. It is virtually pathognomic of cochlear (i.e., sensorineural) hearing loss, where it occurs in conjunction with elevated thresholds for sound detection (Hallpike and Hood, 1959; but see Knight and Margolis, 1984, for the case of presbycusis). For recruiting listeners, sounds that have low amplitudes are inaudible; once the stimulus amplitude exceeds the elevated detection threshold, loudness grows disproportionately steeply, and sounds that have high amplitudes are perceived as appropriately loud (Hood, 1983). The abnormality in the loudness function is then twofold: an elevated threshold and a narrowed dynamic range. When the pathology is unilateral, one can see the relative rates of loudness growth in the two ears using alternate binaural loudness (ABLB) procedures (Carver, 1978; Knight and Margolis, 1984).

The pathophysiologic processes that give rise to loudness recruitment have in common that they compromise the outer hair cell system of the cochlea (e.g., Harrison, 1985; Phillips, 1987). Recall that the transducing elements of the cochlea are the inner hair cells, whose receptor potentials are driven by basilar membrane motion. That motion at any given site is itself driven by two factors: the passive mechanics of the basilar membrane at that site and the active contribution of the outer hair cells. It is the outer hair cell system that is responsible for the great absolute sensitivity, narrow frequency tuning, and dynamic range of basilar membrane motion, and thus cochlear output, at that site (Pickles, 1988; Ruggero and Rich, 1991; Dallos, 1992). Damage to the outer hair cell system results in elevated thresholds and broad ("passive") tuning of cochlear output at the affected site (Harrison and Evans, 1977; Wightman et al, 1977; Harrison, 1985).

There are two further consequences of outer hair cell loss, and both have been used to explain perceptual loudness recruitment. First, as Evans (1975; see also Kiang et al, 1970) pointed out, the frequency tuning curves of cochlear nerve cells at the pathological site are insensitive, broad, and highly overlapping. This means that once a tonal stimulus exceeds the (elevated) threshold for detection by any one of them, then further increments in stimulus intensity very

rapidly incorporate new nerve cells into the discharging population. Evans (1975) suggests that we can assume that perceived loudness is related to the number of active neurons. By doing so, we see that in the pathological cochlea, low-amplitude sounds activate fewer neurons than normal; once the stimulus level exceeds threshold, new neurons are incorporated into the discharging population more rapidly than normal, and high-amplitude stimuli activate near-normal numbers of cells. The phenomenological similarity between this neurophysiologic model and the description of perceptual loudness recruitment is striking, perhaps accounting for the popularity of the model as an explanation of recruitment (e.g., Kiang et al, 1970; Evans, 1975; Pickles, 1988).

The Evans model is essentially a "spread of activation" one (i.e., the neurologic correlate of loudness is a spreading of activation from cells tuned to the test frequency to adjacent neurons). What distinguishes the pathological case from the normal one is the ease with which small changes in stimulus amplitude do this, and this in turn reflects the insensitive and broad frequency tuning caused by outer hair cell loss. There is probably little doubt that the events described in the model actually occur, but the model has a number of problems if it is intended to explain the perceptual loudness problem in recruiting ears. First, wide-band noise of only modest amplitudes (e.g., 20-30 dB SL) probably excites the majority of cochlear neurons yet is not particularly loud; moreover, both the loudness function itself and difference limens for amplitude are similar for tones and noise (see Scharf, 1978; Phillips, 1987 for review). This means that perceived loudness cannot simply depend on the number of active neurons. Second, in normal listeners, difference limens for the intensity of a narrow-band noise signal remain very small even when the signal is presented against a band-reject masker that prevents any effective spread of excitation (Viemeister, 1974, 1983; see also Moore et al, 1985).

These considerations suggest that sound amplitude coding is executed within a cochlear channel, that is, by the neurons serving any specified cochlear site (e.g., that representing the stimulus frequency: Viemeister, 1974, 1983; Phillips, 1987). This brings us to a second consequence of outer hair cell loss relevant to the neural basis of perceived loudness. The intensity profiles of cochlear neurons (i.e., their spike rates plotted as a function of stimulus intensity) are roughly sigmoidal in shape and have

dynamic ranges on the order of 20 to 40 dB (Lieberman, 1978), although, parenthetically, some cells with higher absolute thresholds and "sloping saturation" have wider dynamic ranges. In any event, in ears with outer hair cell loss, intensity dynamic ranges in cochlear nerve cells appear to be both shifted toward higher sound amplitudes and significantly narrowed (Harrison and Evans, 1979; but see Kiang et al, 1970, for a counter-example, and Phillips, 1987, for review), and this set of findings may extend to man (Harrison, 1981). We thus have a neurophysiologic correlate of perceived loudness, operating within frequency-specific processing channels (as does probably most low-level auditory sensory processing; e.g., Phillips, 1995; Phillips et al, 1997), and whose response to outer hair cell loss maps onto human perceptual performance.

What makes this view of special interest is that comparable conjunctions of threshold elevations and dynamic range compressions for preferred-frequency signals are seen in cortical auditory neurons under conditions of wide-band noise masking (Phillips, 1990). This is a stimulus configuration whose effects on the psychophysical loudness function are similar to those incurred by peripheral outer hair cell loss, that is, signal detection threshold elevations and steepened loudness growth functions (Stevens and Guirao, 1967). The steepening effect of wideband noise masking on the intensity profiles of cortical auditory neurons is not also seen in the cochlear nerve (Costalupes et al, 1984). This suggests that the effect seen in the cortex is a purely central phenomenon, and that noise masking should not be construed as equivalent to or a model for outer hair cell pathology. Nevertheless, if one understands the neurophysiologic substrate of perceived loudness to be related to the firing rates of the relevant neurons, then that substrate is now twice seen to have properties that map on to those of the perceptual phenomena, and does not have the problem of the spread-of-excitation models.

FACIAL NERVE PARALYSIS

Nature of the Problem

There is a long history of concern over the discomfort experienced by facial palsy patients presented with moderately intense sounds (Perlman, 1938; Tschiasny, 1949; Adour, 1975; McCandless and Schumacher, 1979; Wormald et

al, 1995). The problem, variously labeled "hyperacusis," "dysacusis," or "phonophobia," may occur in the absence of a patent hearing loss, which thus distinguishes it from loudness recruitment (above). It has further expression in poor speech discrimination at high stimulus intensities ("rollover" seen in speech audiometry: McCandless and Goering, 1974; Borg and Zakrisson, 1975; McCandless and Schumacher, 1979; Wormald et al, 1995).

The most obvious interpretation of these observations lies in paralysis of the stapedius muscle. In normal listeners, contractions of this muscle are elicited only by moderate to loud signals (Borg, 1968; Jerger et al, 1972); in sensorineural ears, the reflex threshold may be increased, although this effect can be partially offset by recruitment (Jerger et al, 1972; Silman and Gelfand, 1981). The stapedius contractions serve to dampen the transmission of low-frequency vibrations to the cochlea (Borg, 1968). Note that this also means that the normal, seamless loudness function is generated by a set of mechanisms that includes any dampening function of the stapedius only at high stimulus levels (see also Morgan and Dirks, 1975). Undampened, the low-frequency vibrations might impair speech discrimination at high intensities through masking of high-frequency signal components (Borg and Zakrisson, 1975; Borg and Counter, 1989).

The hypothesis that a paralyzed stapedius underlies the discomfort aroused by intense sounds in facial palsy derives support from a number of sources. First, there are reports of the absence of an acoustic reflex in such patients (Perlman, 1938; McCandless and Goering, 1974; McCandless and Schumacher, 1979). Second, there is a correlation between the sound amplitude normally evoking stapedius contraction and those evoking the uncomfortable sensations (Perlman, 1938; McCandless and Goering, 1974). Third, McCandless and Schumacher (1979) used ABLB methods to show that loudness growth at the affected ear was normal up to about 70 dB HL, at which there was a discontinuity, followed by a resumption of normal loudness growth, but at a subjectively greater level. Moreover, apparently comparable patterns of discomfort are seen in stapedectomy patients, especially in the months immediately following surgery (Mathison, 1969; McCandless and Goering, 1974).

This general account, however, is not without its problems. First, there is one report that the "phonophobia" in unilateral Bell's palsy was eliminated by insufflation of the middle ear with

10 percent cocaine (Tschiasny, 1949), raising the question of whether the loudness "discomfort" in facial palsy is a somatic sensation rather than an auditory one (see also Melnick, 1968, for a similar questioning of loudness sensitivity in stapedectomy patients). Certainly, the middle ear mucosa receives its own sensory innervation (Jacobson's nerve, a branch of the glossopharyngeal: Duckert, 1993; Guffin and Lucente, 1993), and the tympanic membrane itself receives sensory innervation through cranial nerves V, VII, IX, and X (Wilson-Pauwels et al, 1988). The extent to which these nerve endings are irritated by large motions of the ossicles is not known. Nevertheless, these observations make it very important to distinguish auditory from somatic or other discomfort. This point may also be relevant to the interpretation of anecdotal observations such as that of Nigam and Samuel (1994), who reported that of 100 children undergoing bilateral insertion of grommets for otitis media with effusion, 47 percent reported "hyperacusis" of varying severity.

Second, Adour and Citron and Adour (Adour, 1975, 1982; Citron and Adour, 1978) have argued (a) that acute facial palsy is, in practice, probably a polyneuropathy since the same patients often show signs of Vth and IXth nerve involvement and (b) that there is a very poor correlation between preservation of the acoustic reflex and loudness discomfort levels (LDL: sound level at which the stimulus first becomes annoying) in the same patients (see esp. Citron and Adour, 1978). On the basis of these two premises, Citron and Adour assert that the "hyperacusis" in facial palsy must be due to involvement of the efferent component of the cochlear nerve.

We are thus left with two apparently conflicting reports about abnormal loudness perception and preservation of the stapedius reflex in facial palsy: Citron and Adour (1978), who report no correlation of LDLs with the reflex threshold, and McCandless and Schumacher (1979), who, using ABLB methods, find systematic effects on the loudness function at the affected (areflexic) ear. The present authors favor the view that the conflict between these reports is more apparent than real, because it is quite possible that the two studies were measuring different things: discomfort, in the case of Citron and Adour, and loudness, in the case of McCandless and Schumacher. According to this view, stapedial paralysis may well cause a systematic and reproducible discontinuity in the loudness function (at an intensity normally associated with reflex threshold). The LDL effect

is less consistent because the presence or level of "discomfort" may arise from factors other than perceived loudness, such as a somatic component (see above) or the listener's anxiety level (Stephens, 1970), and these may be present only variably.

Role of the Efferent Component of the VIIIth Nerve

The preceding section leaves unaddressed the issue of whether any involvement of the efferent component of the cochlear nerve could, even in principle, account for disturbances in loudness perception seen in facial palsy (after Adour, 1975, 1982; Citron and Adour, 1978). There is long-standing evidence that section of the crossed olivocochlear bundle in rabbits jointly displaces the input-output function of the acoustic reflex toward lower sound pressures and reduces its slope (Borg, 1971). This suggests that the efferent system exerts a tonic inhibitory influence on cochlear output. In cats, isolation of the efferent neurons from descending control is without consequence to cochlear sensitivity or compound action potential intensity functions, suggesting that efferent control of cochlear function can be mediated entirely by brainstem "reflexive" pathways (Rajan, 1990). This function includes protection of the cochlea from neural desensitization caused by loud sound (Rajan, 1995). Guinan and Stankovic (1996) showed in cats that electrical stimulation of efferents to the outer hair cell system displaces the intensity profiles of cochlear afferent neurons toward higher sound pressures and changes their slopes such that the greatest effect is on responses to middle and high sound pressures, especially for cells with preferred frequencies in the most sensitive portion of the audible range. Although somewhat circumstantial, all of these observations are compatible with a role of the efferent system in modulating responses to moderate and high-intensity sounds.

In man, the evidence is somewhat mixed. Cohen et al (1988) described a multiple sclerosis patient with MRI-confirmed lesion of the left pons that almost certainly interrupted the olivocochlear neurons on that side. This patient's pure-tone audiograms were normal, but the patient had LDLs 20 dB lower than normal on the right and 40 dB lower on the left, in the absence of any commensurate changes in reflex threshold. These data are again consistent with a role of the cochlear efferent system in regulating the encoding of intense sounds.

Fisch (1970) described a history of experience with vestibular neurectomy for treatment of Menière's disease; the surgery almost certainly sectioned the efferent fibers to the cochlea. The hearing of 51 percent of these patients improved following surgery, and in all of these patients, Weber's sign was lateralized toward the operated ear in the months immediately following surgery. In the absence of evidence for a conductive loss on the operated side, the lateralized Weber sign reflects an imbalance in the outputs of the two cochleae. The fact that the sign was lateralized to the operated ear suggests a greater cochlear output on that side, a situation that might well be achieved given the removal of a tonic inhibitory influence.

On the other hand, Scharf et al (1994, 1997), in very detailed studies of vestibular neurectomy patients, found no compelling evidence for abnormalities in loudness perception. In the patient described in the greatest detail, Scharf et al (1994) showed that this was true whether studied using ABLB, adaptation, or magnitude estimation methods, despite the absence of vestibular responses after surgery, and in the same (operated) ear through which the patient showed impaired "frequency focusing" (selective attention in the frequency domain) following surgery.

Taken together, these observations offer only somewhat inconclusive support for the notion that the cochlear efferent system might be capable of exerting an inhibitory influence on cochlear output, perhaps especially for stimuli at moderate and high sound pressures. Whether it is necessary to invoke pathology of this system to explain loudness or other auditory disturbance in facial palsy is a separate issue. One approach to this question accepts Adour's (1982) suggestion that Bell's palsy is in fact a polyneuropathy, but instead of postulating a role of the efferent component of cranial nerve VIII to explain the hyperacusis, ascribes the pathological sensation to the somatic sensory innervation of the middle ear mucosa or tympanum. Recall that this innervation is through cranial nerves V, VII, IX, and X, and the argument that Bell's palsy is a polyneuropathy was based on independently demonstrable clinical signs of V and IX involvement in palsy of the VIIth nerve. Following Occam's Razor, then, there is no need additionally to postulate an involvement of the efferent component of cranial nerve VIII.

Using a quite different approach to the whole issue, Wormald et al (1995) attempted to simulate stapedial paralysis in normal listeners

by acoustically pre-emphasizing the low-frequency content of speech materials used in speech audiometry. They found only 31-percent rollover at 100 dB for the augmented speech in these normal listeners, compared to 49-percent rollover for unaugmented speech in facial palsy patients lacking acoustic reflexes. They concluded from this that, in Bell's palsy, the absent reflex alone could not account for the rollover, thus supporting the view that Bell's palsy is a polyneuropathy involving the cochlear nerve. Those authors, however, did not take into account that the speech audiograms for the augmented signals were 10 dB less sensitive than controls (their Figure 5); when this factor is considered, rollover for the modified speech in the normal listeners appears to be at least as great as that seen in the facial palsy patients. Whether a paralyzed stapedius is alone sufficient to produce a speech discrimination rollover at high stimulus levels is perhaps an open question.

CENTRAL HYPEREXCITABILITY

Above, we reviewed data on some pathophysiologic processes affecting the auditory periphery in ways that might reasonably be expected to have consequences for loudness perception. There are anecdotal reports of others, including closed head injury (Waddell and Gronwald, 1984), perilymphatic fistula (Fukaya and Nomura, 1988), endolymphatic hydrops (Gordon, 1991), and Ramsay-Hunt syndrome (Wayman et al, 1990). It awaits the presentation of systematic observations to determine whether these conditions exert their effects on loudness through the processes described in the preceding pages.

There is, however, a growing number of reports of apparently disturbed loudness function in a diverse range of conditions whose underlying pathophysiologies, if identifiable at all, are central and without obvious auditory, let alone peripheral, auditory components. These include headache (Solomon et al, 1992), central tinnitus (Jastreboff and Hazell, 1993), depression (Carman, 1973), autism (Marriage and Barnes, 1995), and Williams syndrome (Martin et al, 1984; Klein et al, 1990; Nigam and Samuel, 1994).

Marriage and Barnes (1995) offered the intriguing hypothesis that many of these conditions can be linked to dysfunctions of central 5-hydroxytryptamine (5-HT or serotonin) metabolism. Without repeating their arguments in detail, their general hypothesis is that central 5-HT neural systems exert an inhibitory

modulation of central responses to sensory input (e.g., acoustic startle). In concept, it is not unlike Jastreboff and Hazell's (1993) view that hyperacusis is a "manifestation of increased central gain" that mediates an upward modulation of central responses to peripheral stimulation. Certainly, there is anatomical support for such a modulating role of central 5-HT systems, since although central auditory nuclei do not contain serotonergic neurons, they are often innervated by, and have receptors for, serotonergic inputs (Thompson et al, 1994).

The 5-HT hypothesis is not a simple one. This is because some patient groups displaying hyperacusis have elevated 5-HT levels (e.g., autistics: Schain and Freedman, 1961), while in animal studies, it is depletion of central 5-HT that is associated with increased magnitude, and slower habituation of, auditory startle responses (Conner et al, 1970). On the other hand, there is evidence that the upward or downward regulation of startle responses by the 5-HT system depends on its site of action (Davis et al, 1980). Moreover, without necessarily disputing the 5-HT hypothesis, there is also good evidence for a role of gamma amino butyric acid (GABA) neural systems in modulating central excitability (e.g., Szczepaniak and Møller, 1995). In particular, GABA has been implicated in mediating the enhancement of physiologic response magnitude seen in aging and hearing loss and the increased behavioral sensitivity to electrical stimulation of the central auditory system after peripheral hearing loss (Gerken, 1996). There is also increasing suggestion that GABA systems are involved in determining the properties of central tinnitus (Gerken, 1996; Kral and Majernik, 1996), with which central hyperacusis is often associated (Jastreboff and Hazell, 1993).

From the clinical point of view, there may be advantage in withdrawing from attempts to specify any pathological neurophysiology, in favor of addressing the nature of the perceptual experience. In studies of psychophysical responses to noise exposure, "noise sensitivity" has been postulated as an intervening variable between exposure to the stimulus and the "annoyance" it provokes (Taylor, 1984; Stansfeld, 1992). "Noise sensitivity" refers to the predisposition to perceive noisy events, while the "annoyance" is a more affective or attitudinal dimension referring to the extent to which noisy events are evaluated unfavorably (Taylor, 1984; Stansfeld, 1992). The notion that loudness discomfort is a multidimensional phenomenon, with sensory, attentional, and affective components, is not

fundamentally different from well-established conceptualizations of pain in general (Ward et al, 1982; Melzack and Katz, 1984; Melzack and Wall, 1988).

These distinctions may be useful ones. Meikle et al (1984) studied the subjective reports of the quality and severity of tinnitus. They found that subjective severity was not correlated with perceived loudness (achieved using a loudness balance test with an external sound of comparable pitch), but that severity was highly correlated with the degree of sleep disturbance caused by tinnitus. Similarly, a subset of depressed patients who report "hyperacusia" (which the authors defined as being bothered by sounds otherwise routine) regard those sounds as annoying distractions that intrude on attempts to concentrate or sleep (Carman, 1973). Finally, uncomfortable loudness levels are influenced by the anxiety state of the listener (Stephens, 1970). These examples may illustrate the "annoyance" or affective component.

Pathological "noise sensitivity," in the sense used by Taylor (1984) and Stansfeld (1992), may be one of the phenomena seen in conditions such as Williams syndrome, where the description of the hyperacusis syndrome includes not only aversive response to sounds but also an abnormal capture of perceptual or attentive resources by sounds (Klein et al, 1990). In this regard, nearly 75 percent of the sounds that bother Williams syndrome patients are loud or sudden—two properties perhaps most likely to capture attention. A second instance may occur in autism. Here, there is also often a hyperacusis in children with otherwise normal hearing (Bettison, 1996); there is also detailed laboratory evidence for impoverished control of attentional processes (Bryson et al, 1997). Parenthetically, attempts to treat the hyperacusis in autism and, indeed, autism itself, led to the development of the controversial "auditory integration training" (Rimland and Edelson, 1994; Amenta, 1996), in which patients listen to filtered music in which the filter settings are randomly altered (see Rimland and Edelson, 1994). While there is some evidence for behavioral improvement following this training, the most well-controlled study to date found that this effect was no greater than that achieved by listening to unfiltered music (Bettison, 1996). This raises the question of whether it was the intervention per se that was the source of the amelioration.

These sensory, attentive, and affective factors appear also to be represented in the subjective descriptions offered by depressed patients

who are bothered by sounds and who participate in Internet support newsgroups (e.g., <alt.support.depression>). These descriptions often include subsets of these components, frequently without modality specificity, again suggesting a physiologic underpinning outside the classical auditory sensory system. The patients point out that the sounds that bother them are neither necessarily intense ("the smallest noise will distract me") nor painful ("It isn't actually painful, just overwhelming"). This is a quite different phenomenology to those seen in the disorders described above. The important implication, from the clinical standpoint, is that the development of measurement tools that tease out the contributions of the different dimensions of the "painful hearing" experience will provide a basis for targeting those dimensions selectively for medical or behavioral interventions.

CONCLUSIONS

Our argument has been that the interchangeability with which the terms hyperacusis, dysacusis, and phonophobia have been applied to a number of loudness disorders implies some sort of equivalence between the disorders, and that this implication is not warranted. Our thesis has been that the disturbances of loudness perception can arise for different reasons, and that differences in underlying pathophysiology are reflected behaviorally. Our goal has thus been to show that there are readily differentiable syndromes associated with different kinds of disturbed mechanisms, and to argue that each of these merits its own label.

Loudness recruitment is the most familiar of the disorders reviewed above. It is differentiated from all of the others by its systematic co-occurrence with elevated sound detection thresholds. Its expression is in the exaggerated slope of the loudness growth function, and this likely has its basis in the foreshortened intensity dynamic ranges of cochlear afferent nerve cells. This, in turn, reflects damage to the outer hair cell system, so that the perceptual disorder likely arises from a pathological transduction mechanism.

Hyperacusis, in the sense of literally meaning abnormally low sound-detection thresholds, has, to our knowledge, only ever been described in a single abstract without follow-up (Gordon, 1991).

The loudness disorder(s) accompanying facial nerve paralysis and stapedectomy differs

from both of the above. A strictly auditory sensory component likely has its origin in impoverished dampening of low-frequency sound transmission to the cochlea due to an ineffective stapedius muscle. This may well lead to a discontinuity in loudness growth at sound pressures that would normally mark the occurrence of stapedial contractions. There is also the real possibility of a strictly somatic component to the perception of loud sounds in facial nerve paralysis or in middle ear disorders in which the sensory nerves of the mucosa may be prone to irritation. We propose that the term dysacusis be used in such cases.

Finally, there is a host of heterogeneous "central" disorders that have in common listener reports of intense behavioral responses to sounds that would not otherwise be expected to arouse intense sensory experiences. Some studies have attempted to tease apart heightened awareness of sounds (as opposed to lowered detection thresholds for them) and emotional responses to those sounds. The mechanisms underlying these phenomena have not yet been worked out in detail, but they seem clearly to lie more centrally than those mediating the other loudness disorders reviewed here, and the subjective experiences themselves are not the same as those in the other disorders. We propose that the term phonophobia be used to describe the aversive responses in such instances.

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