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INSIGHTS INTO HAZARD FROM INTENSE IMPULSES FROM A MATHEMATICAL MODEL OF THE EAR

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U.S. ARMY HUMAN ENGINEERING LABORATORY
Aberdeen Proving Ground, Maryland
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Insights Into Hazard From Intense Impulses From a Mathematical Model of the Ear

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February 1992

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Insights into hazard from intense impulses from a mathematical model of the ear

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In order to provide insight into the mechanisms that operate in the ear when it is exposed to intense sounds, time and frequency domain mathematical models of the ear including significant nonlinearities in the middle ear were developed to trace energy flow from the free field to the inner ear and ultimately allow the calculation of basilar membrane displacement and a consequent hazard function. These models match the ear's behavior at low intensities and also reproduce many of the features of the data on hearing hazard from intense impulses. They provide critical insights into the loss mechanisms, suggest new strategies for protecting hearing as well as reducing hazard at the source and could also serve as a framework for a new, accurate, theoretically based method for rating hazard from intense sounds.

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INTRODUCTION

A. History

As knowledge of the ear's response to intense sounds has developed over the years, the desire for a good theoretical understanding of the processes involved has been amplified by a practical concern for predicting or ameliorating the effect of intense noise exposures. A substantial portion of the world's population is exposed to intense impulses in a military setting; but sport shooting and other less bellicose civilian activities also result in intense noise exposures to an even larger number. And there is no question that even brief exposure to intense impulses can result in permanent loss of hearing. Therefore, the need for valid information with respect to impulse noise hazard is critical because the population at risk is large and the possibility of damage is real.

There are a number of criteria for impulse noise exposure in use in the world (China, France, Germany, Great Britain, The Netherlands, and the United States of America all have their own criteria [CHABA, 1968; Cheng et al., 1987; MIL STD-1474 (B), 1979; Ministry of Defense (British), 1982; Ministry of Defense (French), 1982; Pfander, 1975; Smoorenburg, 1980]). However, all the criteria have been derived essentially empirically, with little theoretical basis. So long as we have only a rudimentary understanding of the mechanisms acting in the ear at high sound pressures, attempts to find the right combination of measures to use in rating hazard will be severely hampered. Furthermore, the latest data on hazard suggest that the applicability of all the criteria is seriously in question in that they over rate the hazard from low-frequency impulses (Dancer et al., 1983; Price et al., 1989).

B. Applied Issues

In order for a criterion for noise exposure to be useful, it should rate hazard accurately. In addition, it is an advantage if it is formulated on sound theoretical bases in acoustics, physiology, and psychophysics. The issues of accuracy and form are separable. An empirically derived criterion's predictions could correlate highly with hazard for the specific type of sound used in its derivation (and therefore be accurate for that type of impulse); but it may not be generalizable to other types of stimulation and may provide no physical insight into the processes that are responsible for damage. On the other hand, if a criterion was theoretically based, then in addition to rating hazard for specific sounds, it would possess the important benefit of generalizability to the effects of sounds not used in its derivation. Furthermore, it could have heuristic value in suggesting designs or procedures that would reduce or eliminate hazard. Clearly, a theoretically based criterion would possess real advantages.

An adequate understanding of the ear's response to impulse noise should provide answers to three perplexing questions that challenge our current concepts. First, why are low-frequency impulses (Friedlander waveforms with long A durations) not as dangerous as impulses in the midrange? For equal peak pressures, the low-frequency impulses contain much more total energy, and they both contain the same energy in the midrange; yet as we have seen earlier, the low-frequency impulses are less damaging. Second, why is it that audiometrically measured or histologically established losses occur in the midrange, even for impulses that have spectral peaks near 100 Hz? Third, why are losses not much greater than they actually are in practice? That may seem like a perverse question; but consider the great energy present in some impulse exposures. Just one impulse from a large cannon could contain as much A-weighted energy as a full year of exposure to a hazardous industrial noise. Often fi res wear no hearing protection while many rounds are fired. Why then are the losses not much greater than they actually are?

C. Operating assumptions

Given the requirement for an accurate understanding of the basis of the problem as well as the apparent inadequacy
of existing methods for rating hazard, some years ago we initiated a research program intended to address the situation. Presently, our working hypotheses are that within the organ of Corti there is a critical displacement or stress at which the mechanism of loss becomes essentially mechanical in nature. Once that level is reached, additional stimulation results in a rapid growth of damage. Furthermore, the free-field sound pressure producing the critical stress is largely a function of the spectral tuning of the external and middle ears, which effectively form a bandpass filter (Price, 1986).

We hasten to point out that the models to be presented in this paper are not definitive; but are still in active development. However, they do provide answers to the foregoing questions, and also provide a ready basis for the development of research hypotheses.

I. THE MODELS

A. General considerations

For the purposes of this paper, we will try to present enough technical detail with respect to the modeling to make the implications of a model intelligible. An earlier preliminary report on the model has been published (Kalb and Price, 1987) and full account of the modeling is presently in preparation.

Several general considerations guided the modeling. First, we wanted to be able to relate the various elements of a model to specific physiological entities and to give them physically realistic values, to allow for maximum physical insight. Second, to insure maximum utility, we wanted the model to be calculable with modest computer resources (a PC-level computer). Third, the main focus of the model was on the behavior of the ear at high sound intensities where loss was thought to be a function of mechanical stress (higher than 120 or 130 dB SPL). Last, the model had the goal of beginning with a pressure history in the free field and passing the energy through the outer, middle, and inner ears and allow for a feasible estimate of displacement within the inner ear. In order to meet these last goals, we had to integrate models of the head, outer ear, middle ear, and inner ear and allow for nonlinear elements in them. We gratefully acknowledge the efforts of many investigators who have produced models of different sections or components in this chain. As a consequence of their efforts, we have primarily been concerned with the selection and integration of components into a unified system.

B. The models

The details of the models presented are appropriate for the cat ear. This choice was made because the physiological and acoustic values were best known for the cat and also because much of the hearing loss data to be explained were produced with the cat ear. The ears of other mammals are highly similar in structure and the principles used in the modeling should transfer with only modest adaptations to fit their anatomic details.

The ear was modeled with electroacoustic elements. Mathematically, development proceeded in two alternate forms, one in the time domain and one in the frequency domain (Kalb and Price, 1987). The time domain model can be characterized as equations of motion formulated as a system of coupled ordinary differential equations that allow both linear and nonlinear calculations in the time domain. The frequency domain version can of course only be applied to linear processes. Where the nonlinearity of the stapes was treated (discussed later), we performed a nonlinear transformation of the data and then proceeded with the remaining elements of the model. Both models share the same set of variables and values so that their performance is comparable. The two models are complimentary in that they each are structured to allow efficient computation of particular parameters. The circuit diagram of the external and middle ear portions of the models appears in Fig. 1.

1. From the free field to the cochlea

The head was modeled as a spherical baffle around which the sound wave diffracts and in which the ear canal is located. The acoustic field up to the ear canal entrance was approximated by a simple network proposed by Bauer (1967) and the Wiener et al. (1965) two tube model of the ear canal follows it. The middle ear elements are based on a model by Zwislocki (1962) with additional impedance elements given by Lynch et al. (1982). These elements account for the fact that the ear drum does not move as a unit, for losses in the ligaments joining the ossicles, for the areal ratios of the ear drum and stapes and the lever ratios of the ossicles, and for the resonances of the volumes included in the middle ear.

We can gain confidence in the performance of the integrated model by comparing values calculated from the model with acoustical quantities that have in fact been measured. If for the moment we allow the cochlea and its loads to be represented as linear mass and resistance elements, then the

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**FIG. 1. Circuit diagram of model.**
model's calculations can be compared with the available data. The various transfer functions for the ear appear in Figs. 2-5. Figure 2 is the transfer function from free-field pressure to ear drum pressure (data points from Wiener et al., 1965); Fig. 3 is the transfer function of the middle ear (data from Guinan and Peake, 1967); Fig. 4 shows the impedance of the ear (data from Tonndorf and Khanna, 1967, and Moller, 1963); and Fig. 5 shows intracochlear pressure as a function of pressure at the drum (data from Nedzel-nitzky, 1980). The agreement between the models and the data is good except for frequencies above 10.0 kHz in Fig. 2 and above 5.0 kHz in Fig. 4. We believe the lack of agreement in these areas is due primarily to measurement difficulties inherent at high frequencies (noted by the original authors). In general, we believe that the integrated model does a creditable job of reproducing the quantities that can in fact be measured at low intensities.

2. The nonlinear stapes

The stapes is commonly modeled as a linear element. However, at very high intensities (above 130 dB), the middle ear becomes observably nonlinear (Guinan and Peake, 1967; von Bekesy, 1936). We hypothesize that the primary reason for the nonlinearity is the annular ligament, which holds the stapes in place at the entrance to the cochlea, and because of its mechanical structure can exert a controlling influence on cochlear input (Price, 1974). A preliminary report has been given (Price and Kalb, 1986), and a detailed description of the mathematical model of the stapes suspension and a full consideration of its effects are being written for publication.

Strictly speaking, the assertion that the annular ligament is responsible for the nonlinearity goes somewhat beyond the available data. However, the ligament is a likely candidate for the distinction. In comparison with the rest of the middle ear, the ligament is capable of withstanding immense mechanical forces. Of course, it is possible for other parts of the middle ear to become nonlinear. For example, in some animals, the malleo-incudal joint is capable of slippage (even if it is almost never observed), the incudostapedial joint could pull apart or slide, and the ear drum itself is clearly capable of greater outward than inward movement. The list could go on; but we believe that these nonlinearities are likely to be preceded by the nonlinearity at the stapes, and as a consequence, their effect, so far as the cochlea is concerned, is overridden by the limitation imposed by the annular ligament. In any event, whatever the source of the nonlinearity, the result is reduced efficiency in energy transmission into the cochlea.

The annular ligament model conception in mechanical form is shown in Fig. 6. The stapes is viewed as suspended at the edges of its footplate by hinged springs, a configuration that naturally allows for high compliance when displacements are small (as is true for the normal range of sounds to which ears are exposed); but which, because of the geometry, quickly becomes stiffer as displacement increases. The values for the spring suspension were chosen to fit data from

FIG. 2. Comparison of calculated values (solid lines) with measurement of the transfer function of the outer ear by Wiener et al. (1965).

FIG. 3. Comparison of calculated values (solid lines) with measurement of the transfer function of the middle ear by Guinan and Peake (1967).

FIG. 4. Comparison of calculated values (solid lines) with measurement of the impedance of the ear. Data points from Tonndorf and Khanna (1967) and Moller (1963).
Lynch et al. (1982) on static and dynamic stiffnesses for the stapes. In addition, as displacements rise we make the stiffness and damping increase proportionally. The increases in damping are just sufficient to keep the middle ear from 'ringing' when it is driven hard. The full effect of the suspension geometry and spring characteristics can be seen in Fig. 7, which portrays the restoring force for any given displacement. As indicated in the figure, the stapes displacement reaches an asymptote at 20 μm ("breaking displacement").

In Fig. 8, the model's output is compared with actual measurements of middle ear displacement made by Guinan and Peake (1967) in the anesthetized cat. It can be seen in this figure that the model fits the available data and limits maximum displacement to about 20 μm in either direction.

3. The cochlea

The cochlea is modeled as a two-chambered, fluid-filled box with rigid side walls. The partition between the chambers is rigid, except for a tapered basilar membrane (BM) that becomes progressively more compliant toward the apex (mechanical stiffness, mass, and viscous losses changing exponentially). In addition, the cross-sectional area of the cochlea decreases exponentially from base to apex.

For rapid computation, we used a WKB approximation to calculate transfer functions to each of 50 points along the BM. By using forward and inverse FFTs, we calculated a history of BM motion along its length. In the differential equation model, the cochlea is discretized into 512-coupled resonant elements whose characteristics change with location. The values chosen for the mechanical properties produce the envelopes of traveling wave displacements that show a changing Q and an increase in amplitude with increasing frequency as seen by von Bekesy (1949). We believe that these envelopes are characteristic of the BM when driven at high amplitudes where the sharp tuning produced by active processes that operate at low intensities cannot have an appreciable effect.
4. Hazard calculation

The mathematical models were first used to calculate the envelopes of BM displacement (Kalb and Price, 1987) as a means of assessing relative hazard of different impulses. But the BM undergoes many oscillations in response to any but the simplest stimulus; therefore to account for the effects of the repeated flexing of the BM during an exposure, we added an additional calculation. As a first approximation, we assume that loss processes operating within the cochlea are akin to those that are generally operative when materials are repeatedly stressed and finally fatigue and fail (Price, 1983a). As Broch (1979) suggested, for materials in general, the number of cycles to failure is a function of the strain raised to a power, usually greater than two, i.e., $N \times S^b = K$, where $K$ is a constant and $b$ is the power to which the strain is raised. It is apparent that this formula could be integrated with the model's calculation of BM displacement to yield an estimate of hazard. Therefore, we calculated a value (strain) for the peak of the upward displacement of each of the 50 segments of the BM model as it was exposed to a given stimulus, the value was raised to some appropriate power (2.5 was used in the calculations reported here) and the effect summed.

An additional factor needs to be noted regarding the calculation of strain. The BM's width changes about 3 fold from base to apex; therefore it follows that the amount of strain represented by any given displacement must also vary as a function of the linear dimension of the member being displaced. To account for this, in calculating hazard we weighted displacements of the BM progressively less with more apical locations in accordance with the change in BM width. The end result of this exercise was a number for each of the 50 segments of the BM that integrated both the peak amplitude of its displacement with the number of occurrences. As this is written, we have only begun to explore the hazard calculation. The units of hazard are arbitrary, although we presume that the calculated value represents at least an ordinal scale of hazard.

In calculating hazard, only the upward deflections of the BM were included. This was done because such displacements had earlier appeared to correlate best with the damage (Kalb and Price, 1987) and have the additional advantage of making physiological sense as possible damage producers. Briefly, upward movements of the basilar membrane produce predominantly stretching forces at the level of the tectorial membrane and reticular lamina. Given that tissue tends to fail in tension, upward displacements could well be especially hazardous. Conversely, compressive movements (downward displacements), would be expected to be less damaging. A recent conception of the micromechanics of transduction in hair cells (Pickles et al., 1984) fits this line of argument. Upward displacement of the basilar membrane is associated with forces that cause the stereocilia to deflect toward the longer stereocilia, stretching the tip links, decreasing the membrane resistance and exciting the hair cell. Movements in the opposite direction simply allow the links to go slack. If this is in fact the case, then stretching movements (upward displacements) could easily result in damage and compressive movements (downward displacements) could be relatively less hazardous.

II. APPLICATION OF THE MODELS
A. Effect of the middle and external ear

The influence of the conductive properties of the middle ear on an impulse is immense. The bandpass filtering effect is well known and the proposed peak limiting add to it. The effect of peak limiting can be appreciated by considering the displacement that would have taken place, had the middle ear been linear. In response to impulses in the crew areas of large caliber weapons, calculations indicate that the stapes would try to move 2000 or more microns. With peak limiting present (as is the case in the real ear), the displacements are limited to less than 20 μm, a reduction of more than two orders of magnitude!

The effect of the middle and external ears on the energy that enters the cochlea is portrayed in Fig. 9. In this somewhat complicated figure the energy in 1/3 octave bands entering the cochlea has been calculated for Friedlander waveforms with A-durations characteristic of a rifle and a howitzer, each at three intensities. These waveforms are close simulations of weapons impulses; but do not have the "hash" on them associated with measures of real impulses. Two major points can be made from this calculation. First, even though the spectral peaks of these impulses in air would have been at 140 and 800 Hz, respectively, at the input to the cochlea they all have their greatest energy at about 3000 Hz. The ear acts like a fairly sharply tuned bandpass filter; therefore all impulses naturally tend to have their greatest effects in the ear's mid-range. Second, note that with increases in intensity, the energy increases nonlinearly for both impulses. However, because of the peak limiting, the howitzer type impulse at 180-dB peak pressure actually has less energy in the midrange than the 160-dB rifle-type impulse. This process could explain much of why the low-frequency impulses are less hazardous than previously thought.

![FIG. 9. Calculated 1/3 octave energy entering the cochlea for Friedlander waveforms simulating rifle and howitzer impulses at three intensities.](image-url)
B. Effects for the intact ear

Given that we are interested in modeling the ear's response to intense impulses and the attendant hazard, it is instructive to explore what happens when the model is driven with acoustic waveforms that have been used in experiments with real ears. These calculations can be seen in Figs. 10-15. In figures where the hazard function is displayed, the maximum on the vertical scale for the hazard function was arbitrarily set at 10,000 “units” in order to allow at least relative comparisons between impulses.

1. The 166-dB howitzer impulse

In Fig. 10, we see the response of the ear to a 166-dB howitzer impulse used in a previous study (Price, 1986a). The top panel is the pressure history of the impulse, the middle panel is the calculated stapes displacement, and the bottom panel is the calculated BM displacement envelope and the hazard function. It is apparent that while the calculated stapes displacement preserves the general form of the impulse, the effect of the clipping is visible. All the extremes of pressure appear compressed in the stapes displacement and the oscillations near ambient pressure are proportionately larger, as would be expected.

The envelope of BM displacements is relatively broad and shows one peak in midcochlea and a second one near the apex. When the evolution of the BM displacements resulting from the stapes motion was examined, then a surprising effect was observed. The initial peak pressure was not responsible for the maximum displacement within the envelope (the BM only went a little over half the envelope height in midcochlea), rather the largest displacement was a result of the relatively insignificant peak marked with an arrow in the

![166-dB howitzer impulse](image1.png)

FIG. 10. 166-dB howitzer impulse (Price, 1986a). The upper panel is the pressure history, the middle panel is the calculated stapes displacement, and the lower panel is the envelope of basilar membrane displacements and the hazard function (explained in the text). The arrow in the upper panel indicates the peak in the pressure history responsible for the peak BM displacement.

![160-dB howitzer impulse](image2.png)

FIG. 11. 160-dB howitzer impulse (Price, 1986a). The upper panel is the pressure history, the middle panel is the calculated stapes displacement, and the lower panel is the envelope of basilar membrane displacements and the hazard function (explained in the text).

![154-dB howitzer impulse](image3.png)

FIG. 12. 154-dB howitzer impulse (Price, 1986a). The upper panel is the pressure history, the middle panel is the calculated stapes displacement, and the lower panel is the envelope of basilar membrane displacements and the hazard function (explained in the text).
pressure waveform. We presume that it was a particularly effective driver because it occurred near ambient pressure (when the stapes was not clipping) and its duration was such that it put its energy where the ear was most easily driven.

The evolution of the hazard function was also instructive. First, the maximum hazard was calculated to occur in the middle of the cochlea. This was true for all the impulses tested, regardless of their spectral peak, and was apparently due to the fact that the largest oscillations often occurred there as well as a greater number of the smaller oscillations. The apical portion of the BM would typically go through a large single displacement when significant low-frequency energy was present (reflected in the envelope of displacement). However, this apical displacement added less than might be expected to the hazard calculation because it occurred less often and the hazard calculation weighted it less because it occurred in a wide portion of the BM and therefore it represented a smaller strain. The peak of the hazard calculation was in the middle of the cochlea and for this impulse reached more than 60,000 "units," mostly during the latter portions of the impulse (after 10 or 15 ms).

The foregoing calculation compares favorably in several respects with the measured hazard (Price et al., 1989). Severe losses did result from exposure to this impulse, and the electrophysiological and histological loss measures indicated damage primarily in the middle of the cochlea.

2. The 160-dB howitzer impulse

Figure 11 is a parallel to the previous figure. The clipping is a little less visible in the stapes displacement as would be expected, given the lower peak pressure of this impulse. The displacement envelope for the BM shows a large peak apically and a smaller one in midcochlea. The hazard function, on the other hand, shows the reverse. The reason for this is apparent when the evolution of the pattern is ob-
served. The apical regions of the cochlea oscillate less often and the oscillations are weighted less. The hazard function grew to about half scale based on the response to the first 5–10 ms of the impulse and the remainder of the wave roughly doubled the height.

Again, certain of the model calculations match the loss data. This impulse was indeed less hazardous than the 166-dB impulse, and losses tended to occur in midcochlea.

3. The 154-dB howitzer impulse

The model’s response to the 154-dB howitzer impulse is portrayed in Fig. 12, where we see a continuation of the trends established in the two higher level impulses. For this impulse, the hazard function reached essentially its full height after the first phase of the impulse had passed, the latter portions of the wave contributing little to its growth.

The match between the hearing loss data and the hazard calculation for this impulse raises questions of a more general sort. The greatest losses were in fact in the midrange, which does match the hazard calculation. However, this impulse and the 160-dB impulse produced essentially the same losses in the cat ear. That in itself is somewhat surprising [noted in the original article (Price et al., 1989)]; however, if the hazard calculation is examined after the first 10 ms, then the two impulses are rated about equal. We tentatively speculate that if the middle ear muscles begin to act at about this point, then the hazard may be reduced for the latter portions of the wave. This, of course, would be true for the 166-dB impulse as well.

4. 155-dB rifle impulse

In Fig. 13, the data for a rifle impulse are depicted. The pressure and time scales were expanded to show the waveform better; but the cochlear scales were unchanged. Some clipping can be seen in the step displacement. The envelope of BM displacement shows less activity at the apex, as one would expect, and the hazard function shows its peak in midcochlea. The rifle impulse is short, therefore the hazard calculation grows only in the first few milliseconds.

The match between calculated and measured hazard (Price, 1983b) is relatively good. The losses were in fact primarily in the middle of the cochlea, and this impulse was about as hazardous as the 160-dB cannon impulse. If we only consider the first 5–10 ms of the cannon impulse, then this relationship also holds for the calculated hazard. If the entire waveform is considered, then the 160-dB howitzer impulse would be rated as much more hazardous by the model.

5. Patterson’s hi-peak and low-peak impulses

Patterson and his colleagues (1986) did an interesting experiment in which two impulses were used for an exposure. The impulses were digitally processed so that at the acoustic output of a speaker, the impulses had identical magnitude spectra (peak energy at 1.4 kHz); but because the phase relationships of the components were different, the peak pressures differed by 8 dB (producing what was referred to as a hi-peak and a low-peak impulse). Both histological analyses and behaviorally measured audiograms were available for these ears. These data provided an interesting challenge for the model; although, strictly speaking, the data were gathered on the chinchilla, and the model values were for the cat.

The model’s response to the hi-peak impulse at 147 dB is shown in Fig. 14. At this pressure, there is little clipping. The envelope of BM displacement and the hazard function both show maxima in midcochlea, as one would expect. The model’s response to the low-peak impulse at 139 dB is presented in Fig. 15. The maximum of the envelope of the BM displacement and the hazard function are both somewhat smaller than for the high-peak impulse and are located somewhat more apically. This corresponds with their effects on the ear. The hi-peak impulse produced somewhat more PTS and hair cell loss, and the histological damage for the low-peak impulse was located somewhat more apically than the damage for the high-peak impulse (their Figs. 15 and 16). Admittedly, these relatively subtle correspondences may be fortuitous; but we note them with interest.

III. INSIGHTS

A. Function of the ear

The observation that the ear is spectrally tuned, primarily because of the external and middle ears, is hardly new. Nevertheless, it is a concept that has utility in explaining effect of noise on the ear. Certainly, the fact that the middle of the cochlea is most susceptible to damage is largely due to the fact that the transmission path works most efficiently in the midrange of frequencies.

The effect of a nonlinear stapes has not been so thoroughly explored, however. A limit to displacement does explain why it is that damage is less than we might otherwise expect from intense impulses. At a somewhat more subtle level, a displacement limit means that we need to reexamine traditional methods of rating waveforms for hazard. The initial positive peak has received almost all the attention in damage risk criteria (DRCs) (it contains most of the energy); yet once the stapes has reached its limit, the ear is functionally disconnected from the acoustic field. Thus the feature that controls the rating of hazard may be a minor part of the stimulus. On the other hand, when the acoustic pressure is near ambient, the stapes can drive the ear very effectively. This means that the apparently insignificant “tails” of intense impulses may actually be the real sources of cochlear input. Furthermore, when the initial positive pressure arrives, the stapes usually starts from a rest position and can displace 20 μm or so. However, it is capable of displacing twice that distance, if driven from extreme to extreme, which presumably would be a worst case condition.

Although not presently a part of the model, the middle ear muscles also act to change the transmission path. They are often viewed as contracting reflexively to loud sound and hence, for impulses lasting only a few milliseconds, as being a negligible influence. Alternatively, given their short latency of contraction, they may be able to influence the later portions of longer impulses, even if they only contract reflexively. However, they may be active for other reasons and their influence should perhaps not be ruled out for any noise exposure.
B. Ameliorating hazard

Several of the interesting implications from these calculations relate to novel methods of ameliorating hazard. The presence of low frequencies at the input to the ear might be more of a help than a hazard, if they push the stapes into clipping. In fact, enough pressure in the outer ear or middle ear could do the same thing. Small barriers between the impulse source and the ear could create a sound shadow, especially for the mid and high frequencies and offer significant protection to the ear. In any event, because the model is theoretically based, it does offer the possibility for evaluating and developing schemes for reducing hazard.

C. Methods of assessing hazard

The foregoing analysis suggests that because of the non-linearity in the middle ear, virtually all conventional approaches to the rating of hazard from intense sounds (above 140 dB) are likely to have difficulties that may be insuperable. On the other hand, we are encouraged with the successes the model has had and the insights it is providing. Should additional development of the model and the hazard function continue to match hearing losses already measured and predict hazard for different situations, then it could serve as a basis for a new DRC for impulse noise. The use of a calculational procedure such as this in a standard is not traditional; but given the modest computational requirements and the persuasiveness of the PC-level machines, it may succeed. Alternatively, it is conceivable that a circuit or chip could be integrated into a "hazard meter," much like the current sound-level meters.

The foregoing discussion has shown briefly that the mathematical models are reproducing the essential elements of hearing hazard and are doing it in a theoretically consistent manner. At this time, no other method of rating hazard comes close to being accurate. We are very much encouraged that these models are on the right track and will prove to be sound basis for a new design criterion for impulse producing weapons.
