The growth of and recovery from TTS in human subjects exposed to impact noise

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It is now recognized that temporary threshold shift (TTS) grows to an asymptotic level (ATS) after a prolonged exposure to steady-state and intermittent noise. Few studies have been conducted to verify this process of acquisition for impact noise. However, results obtained on animals have demonstrated that an asymptote is reached after 1–2 h of exposure. These results have been confirmed in the present study on four human subjects using a 470-ms (B duration) impact noise at a rate of 1 pps. The above pattern of acquisition has been retested on two subjects exposed to the same impact for periods of 15–150 min. An ATS of 15–20 dB was measured ($L_p = 102–104$ dB) after 30–60 min of exposure. Recovery curves were determined for each exposure duration. They appeared to follow a logarithmic function of post-exposure time. It took from 30–50 min for one subject and from 4–5 h for the other to completely recover. The time for total recovery did not seem to vary as a function of exposure time. Implications are drawn for damage risk criteria and for the study of the effects of impact noise on hearing.

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INTRODUCTION

Temporary threshold shift (TTS) studies are of interest in evaluating the risk of damage to hearing. One damage risk criterion assumes that the asymptotic value of TTS (ATS) is closely related to the amount of permanent hearing loss resulting from several years of exposure to the same noise (Mills et al., 1970; Bohne and Clark, 1982). Rules to predict the growth of and recovery from such an ATS are the result of many studies done on both humans (Melnick and Maves, 1974; Ward, 1975; Melnick, 1976; Mills et al., 1979; Shaw, 1983; Takagi et al., 1987) and animals (Carder and Miller, 1972; Mills and Talo, 1972; Mills et al., 1973; Mills, 1976; Saunders, 1977; Burdick et al., 1978) exposed to steady-state or intermittent noise. Few such studies have been done with respect to impulse noise. There are indications that ATS could be attained much more rapidly than with steady-state noise. The present investigation had two distinct objectives: (1) to summarize the data available to date on the growth of and recovery from TTS resulting from exposures to impulse noise, and (2) to present and discuss the results of recent studies conducted on human subjects exposed for periods of 8–150 min to long-duration reverberant impact noise presented at a rate of 1 pps.

I. REVIEW OF AVAILABLE DATA

A. Growth of TTS for impulse noise

It is generally stated that TTS grows linearly as a function of time or of the number of impulses (Melnick, 1978). In fact, to our knowledge, only three relevant studies have been conducted with human subjects (Ward et al., 1961; Loeb et al., 1965; Walker, 1970).

A close analysis of these studies has demonstrated that many functions prevailed: linear, exponential, logarithmic, and asymptotic. The differences noted between the result of these studies (Table I) could be attributed to many factors in terms of the exposure parameters used and the method used to analyze the data.

In this context, these do not allow the definition of a simple rule that relates the exposure time to TTS for impulsive noises.

Recently, however, studies conducted on chinchillas by Blakeslee et al. (1978), Henderson et al. (1979), and Henderson and Hamernik (1982, 1986) suggest a process that is neither linear or logarithmic, but asymptotic.

In one study on human subjects (Hétu et al., 1984), the results suggested that, after 60–90 min of exposure to impacts presented at a rate of 1 pps, TTS approached asymptotic level. Based on these results, a study has been carried out to confirm the conclusions of Henderson and Hamernik (1986). Before presenting these results, we will examine the data available in the literature on the recovery from TTS following an exposure to impulse noise.

B. Recovery from TTS for impulse noise

Luz and Hodge (1971) had observed that, following an exposure to impulse noise, the thresholds did not recover...
linearly as a function of the logarithm of time. To explain this phenomenon, they have postulated a model based on two mechanisms of fatigue: a metabolic process \( (M) \) and a mechanical process \( (S) \), the first of which recovers before the other. From these two processes, they had suggested four types of recovery that had been observed experimentally: "log time" (Cohen, 1961; Smith and Goldston, 1961; Rice and Coles, 1965; Fletcher, 1969; Blakeslee et al., 1978), "rebound" (Romba and Gates, 1964; Clack, 1966; Henderson et al., 1974), "diphasic" (Ward, 1970), and "delayed recovery" (Ward, 1960, 1970; Rice and Coles, 1965). When the TTS due to the process \( S \) is absent, the simple logarithmic recovery \( M \) is seen. If there is some slight \( S \)-type TTS, the curve shows a slight tail. If \( M \) recovers before \( S \) has grown to its full magnitude, a rebound will appear. Part of the recovery of \( M \) may be masked out by the growth of \( S \), which gives a diphasic recovery. Finally, if the entire \( M \) recovery curve is masked by an \( S \) type of large magnitude, there is delayed recovery. Mechanical fatigue would become predominant when the subject is exposed to a certain level of noise exceeding a critical level, which differs from one individual to another (Hétu and BoudreauIt, 1978; Price, 1979).

The studies done with chinchillas by Blakeslee et al. (1978) and Henderson et al. (1982) that reported reaching ATS after an hour of exposure would not involve mechanical fatigue since the recovery curves were linear with respect to the logarithm of time. Furthermore, they have noted that, at the higher frequencies where more TTS was observed, recovery was more rapid than at the lower frequencies. Moreover, a comparison between the results of the Blakeslee et al. (1978) and Mills and Talo (1972) reveals that, for equal amount of ATS, the risk of permanent hearing loss will be more pronounced if the exposure is to impulse rather than to steady-state noise.

In light of this analysis, one must conclude that no data are available to describe the process of recovery following exposure to impulse noise for human subjects, except for those from Luz and Hodge (1971), which focused on very high exposure levels (155-dB peak) for unspecified durations. It is also unknown whether ATSs were reached during these exposures.

To clarify the relationship between the growth of and recovery from TTS, experiments were conducted on TTS from impulse noise with human subjects. Experiment I aimed at describing the growth of TTS as a function of exposure duration to a reverberant impulse having a long decay time presented at a repetition rate of 1 pps. This type of signal was chosen because the range of exposure levels that would induce specified amounts of TTS was known from the results of previous experiments (Trémolières and Hétu, 1980; Hétu and Lazure, 1982; Hétu et al., 1984). The signal corresponded to noise I in the study by Trémolières and Hétu (1980). Figure 1(a) and (b) shows the pressure–time profile and the third-octave-band energy spectrum of the signal. The \( B \) duration (see Coles et al., 1968) of the noise was 470 ms. It had maximum energy in the third-octave band centered at 3150 Hz.

### II. GENERAL METHOD

#### A. Noise generation and calibration

The recorded noise of a hammer beating against a metal plate was played back through a loudspeaker. The subject was seated facing the speaker at a distance of 1.5 m; the height of the seat was adjusted to insure that the subjects always remained in the horizontal plane of the diffraction horn to which the compression driver was coupled. The noise generation system and calibration procedure were identical to those employed in previous studies (Trémolières and Hétu, 1980; Hétu and Lazure, 1982; Hétu et al., 1984). The signal corresponded to noise I in the study by Trémolières and Hétu (1980).

Experiments were conducted to confirm the results of Experiment I and to study the recovery process following this kind of exposure.

#### B. Hearing threshold measurements

The subjects were selected for having hearing threshold levels better than 15 dB (ANSI-1969) from 500–6000 Hz. Thresholds were measured with TDH-50 earphones in MX-41/AR cushions and a Grason-Stadler audiometer (1703-B) on which an attenuator was installed to measure threshold down to \(-20\) dB HL.

The measurements of TTS\(_2\) was performed at one frequency, namely at the most sensitive ear and at the frequency most affected by the particular noise under study. In fact, post-exposure audiograms were initiated 75 s after the end of the exposure at the frequency preceding the one most affected. Each interrupted test tone (200 ms on–off) was presented for 30 s. The most affected frequency was identified...
through successive exposures to series of 60 impacts; the peak level was progressively increased until a 15-dB threshold shift could be observed at one frequency between 3 and 8 kHz at one ear. This last exposure was repeated in a separate session in order to verify the identification of the target frequency.

III. EXPERIMENT I: TTS GROWTH CURVES AS A FUNCTION OF THE PEAK LEVEL AND THE DURATION OF EXPOSURE

A. Exposure conditions

Four peak levels were tested, namely, 102, 108, 114, and 120 dBA (i.e., 101, 107, 113, and 119 dB SPL). The repetition rate was 1 pps in all exposure conditions.

The effect of exposure durations between 0.5 and 60 min was tested in separate sessions on four subjects. A minimum of 24 h separated two successive sessions for a given subject. For the higher peak levels, the exposure was limited to durations that induced amounts of TTS less than 20 dB.

B. Data reduction

The progression of TTS against time was expected to follow an asymptotic function as suggested by the results of Walker (1970) and the ones obtained on chinchillas. Accordingly, individual and median data relating TTS to exposure duration were fitted with a very high degree of accuracy using the Gompertz function (Hunt, 1982). This function is defined by Eq. (1):

\[ \text{TTS}(D) = V_g e^{-h_1} - V_g, \]

where TTS(D) is the amount of TTS after an exposure duration of D at a repetition rate of 1 pps. Here, \( V_g \) and h are parameters.

C. Results

The individual results for the four subjects exposed to the four levels are presented in Fig. 2. As in most TTS studies, results show an interindividual variability. In our study, however, subject 1 is the most sensitive in each case. The median results, as depicted in Fig. 3, give a straightforward picture of the combined influence of the peak level and of the exposure duration. It can be seen that an asymptote of 4–18 dB appears to be reached within 30 min at 102 and 108 dBA, and possibly within 20 min or less at higher peak levels. Serious reservations concerning the estimated asymptote at 120 dBA should be made. In that particular case, the exposures were carefully controlled to limit the TTS to 20 dB. Because of that criterion, no exposure exceeding 12 min was allowed. The asymptote was then predicted by Eq. (1) without knowing TTS values for longer exposure durations. These results should be tested on animals to clarify whether the TTS reaches an asymptote or increases linearly with time.

IV. EXPERIMENT II. GROWTH OF AND RECOVERY FROM TTS

A. Exposure conditions

TTS growth curves were measured for two subjects at peak pressures, which induced a predetermined effect of 10–20 dB. These levels were 102 dBA for subject CL and 104
dB for subject RH. The amount of TTS was measured for exposures lasting from 8–150 min in order to verify if an asymptote was actually reached. A minimum of 24 h separated each exposure to ensure complete recovery.

B. Data reduction

The data for the growth of TTS were fitted to the Gompertz function presented in Eq. (1). In order to plot the recovery curves, the model proposed by Ward et al., 1958, Eq. (6) was chosen. If has the following form:

\[ TTS_t = TTS_0 [1 - k \log(t/2)] \]  

where TTS is the amount of TTS still present at time t and k is a constant representing the rate of recovery per logarithmic units of time.

C. Results

1. Growth of TTS

Figures 4 and 5 present the results for subjects CL and RH, respectively. In Fig. 4, the TTS value reaches an asymptote of 15 dB after 30–45 min of exposure. The amount of TTS of 18 dB after 90 min appears to be attributable to measurement error since longer exposures did produce lower amounts of TTS.

For subject RH (Fig. 5), ATS of 19.5 dB is reached after 60–90 min of exposure. In this case, every point is very close to the curve fitted by the Gompertz function.

The amount of ATS and the time required to reach the asymptote differed between the two subjects. This difference probably reflects interindividual differences, but a procedural factor, such as the exposure level, cannot be ruled out.

2. Recovery from TTS

Table II presents the results as estimated from Eq. (2) for the recovery from TTS for subjects CL and RH. The two subjects differ in the amount of time necessary to recover. In the case of subject CL (Fig. 6), it took from 182–1008 min to recover completely. The difference between these two values is quite large, and we can suspect an effect related to exposure duration. Actually, close inspection of the data suggests that the recovery process was completed to within 1 dB after 300–400 min for the 60- and 90-min exposures [Fig. 6 (c) and (d)]. The 300 to 400-min recovery period for the other exposure durations is close to those observed for 30, 45, and 120 min of exposure. Moreover, it is worth noting that the amount of TTS after 90 min of exposure reaches 18 dB, while it is 13 and 14 dB for the other exposure durations. This may explain the longer recovery time.

In the case of subject RH (Fig. 7), the recovery time does not vary from one exposure duration to the other except for the 120-min condition [Fig. 7 (e)]. It is difficult to explain this particularly long recovery duration (154 min), since all TTS values are similar and the recovery duration for the 150-min exposure is comparable to the others, namely, 54 min as compared to 35–40 min.

In summary, the results obtained with these subjects suggest that increasing the exposure duration beyond the interval necessary to reach an ATS does not substantially affect the recovery time. This holds for exposure durations

<table>
<thead>
<tr>
<th>Exposure duration (min)</th>
<th>Time interval for complete recovery (min)</th>
</tr>
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<tbody>
<tr>
<td>30</td>
<td>334</td>
</tr>
<tr>
<td>45</td>
<td>242</td>
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<tr>
<td>60</td>
<td>856</td>
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<td>90</td>
<td>1008</td>
</tr>
<tr>
<td>120</td>
<td>182</td>
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<td>150</td>
<td>...</td>
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TABLE II. Time required for complete recovery from TTS for subjects CL and RH as estimated from Eq. (2).
between 8 and 150 min (for number of impacts between 480 and 9000) at levels that induce 10–20 dB of TTS. It is currently too risky to generalize these results to other conditions.

V. GENERAL DISCUSSION

From the previous results, it is possible to derive theoretical, methodological, and practical implications.

A. Theoretical Implications

First of all, the Gompertz function accurately reflects the growth of TTS for reverberated impulses when the data set includes points beyond the point of inflexion, as in Experiment II. This has also been the case with nonreverberant impulses (Laroche et al., 1986; Nicolas et al., 1989). In these studies, ATS on the order of 8–12 dB was attained after 30 min of exposure to nonreverberant impulses of different frequencies at peak levels between 124 and 136 dB.

The rapid appearance of the asymptote, therefore, characterizes the exposure to impulse noises of moderate level, whether of the reverberated type or not. Our results are in agreement with the conclusions of Henderson and Hamernik (1986), who exposed chinchillas to impact noise. An asymptote is reached after 1 h or less of exposure, compared to between 8 and 24 h for steady-state noise. In a recent review, Henderson et al. (1987) consider that a distinction between the effects of impulse and continuous noise is more a question of degree than of dichotomy. It seems that a transition region exists between metabolic exhaustion and mechanical damage for the impulse as well as for the continuous noise. This transition region is in the vicinity of 119–125 dB for the chinchilla.

In the above study conducted on human subjects, it is
unlikely that mechanical fatigue was developed. Indeed, Trémolières and Hétu (1980) have studied this very signal to determine the critical exposure level, i.e., the peak pressure that induced 15 dB of TTS at 4, 6, or 8 kHz following an exposure to 60 impacts at a rate of 0.5 pps. The mean peak level for the 12 subjects was 133 dB, while the median was 131 dB. These peak levels were 27 to 31 dB higher than those used in the present study (102 and 104 dB).

It is obvious that the hypothesis regarding the transition region given by Henderson and Hamernik (1986) does not permit an explanation of the mechanism responsible for acquisition of an asymptote, which is at least 16 times faster for impulse noises than it is for continuous noises. Another explanation for the faster reaching of the asymptote must, therefore, be sought. One possibility is that the location of the fatigue could be at a very specific location that could differ with the type of noise. It is now known that the rigidity and the stiffness of the stereocilia are affected during an exposure to noise. Indeed, three sites of stereocilia alterations are suggested, which are: (1) the paracrystalline core of the stereocilia, (2) the rootlet, and (3) the interconnection links (Flock and Streljoff, 1984). Such alterations would manifest themselves gradually as a loss of rigidity or stiffness. Saunders et al. (1986) have demonstrated this process by mechanically displacing stereocilia with an oscillatory water jet (200 Hz) and by following the evolution of the mechanical thresholds of the stereocilia during the exposure. One of the most interesting observations is the appearance of a plateau, i.e., the stabilization of the mechanical thresholds after 5 or 6 min of mechanical excitation.

For recovery to take place, it is suggested that the links joining the stereocilia should not be severely damaged (Miller et al., 1985; Saunders et al., 1985; Saunders and Flock, 1986). Saunders et al. (1987) have put forward a hypothesis on the active mechanism responsible for recovery: It may involve activity in the protein structure of the cell or a repair process. Because recovery is so rapid, it is more likely that the cell insure the recovery process rather than the synthesis of new material.

Two studies concerning this effect have been conducted on animals, one with narrow-band noises (Liberman and Beil, 1979) and the other with high-level impulses (155 dB)
Unfortunately, very few data on ATS have been obtained at nick (1974) has tested an octave-band noise (300-600 Hz) these levels would have revealed a faster acquisition of ATS. Data from Miller (1958), obtained with six human subjects exposed during 3 min to a white noise of 80-95 dB. Their results do not reveal a shift in the function of peak levels of a given signal and determine, on animals, the level at which injuries begin to appear. Indeed, below these levels, it is unlikely that we would measure any significant ATS, i.e., higher than 5 dB. If this transition region really exists, and if it is related to the stimulation of the basilar membrane, it could be postulated that, at a corresponding peak level of continuous noise, a similar amount of ATS would be observed. Data from Miller (1958), obtained with six human subjects exposed during 3 min to a white noise of 85-120 dB, give some clues regarding this hypothesis. TTS growth curves as a function of pressure level clearly demonstrate a rapid increase of TTS, which begins between 90 and 100 dB. We can ask ourselves if a prolonged exposure to these levels would have revealed a faster acquisition of ATS. Unfortunately, very few data on ATS have been obtained at levels higher than 90 dB with human subjects. Mills et al. (1981) have used wideband noises of 76-91 dB, while Melnick (1974) has tested an octave-band noise (300-600 Hz) of 80-95 dB. Their results do not reveal a shift in the function relating ATS to the exposure level. A study done by Mills et al. (1979) has, however, demonstrated a clear shift of the TTS growth curves during an exposure to an octave band centered at 4 kHz at a level of 88 dB compared to an exposure of 83 dB. It is important to note, however, that these studies were not conducted in order to analyze this particular phenomenon. Furthermore, many factors can explain these differences, such as the mathematical models used to estimate the TTS growth curves, which differ from one study to another, the number of experimental data collected to set the curves, the type of signals used, and the frequency at which TTS was measured.

One electrophysiological study done by Salt et al. (1981) yields information that is more than interesting with regard to our hypothesis. Indeed, they have exposed guinea pigs to continuous and impulse noises of equal energy (95-105 dB SPL) and have analyzed the cochlear microphonic (CM) and the action potentials (AP) during exposures to these types of noises. Their results have clearly demonstrated that the CM is suppressed to a greater extent by impact noise than by continuous noise of equal energy. They concluded that the short-term effects of noise on the CM, and, hence, possibly on the hair cells, are significantly greater for impact noise than for continuous noise. Further studies of this type would have to be conducted simultaneously with behavioral and histological studies in order to better understand the mechanism responsible for the fast acquisition of ATS.

B. Methodological implications

In order to derive accurate rules of the growth of and the recovery from ATS, it would become important to adopt a standard methodology. Indeed, the limits encountered in explaining some phenomena rest, in large part, on the differences between methodologies used in animal studies as well as in human studies. The reaching of an asymptote represents, according to Saunders et al. (1985), an anchor point that allows the comparison between studies, providing that equal ATS means equal risk of PTS.

This anchor point is define as the value of TTS at the time at which the asymptote is just attained. In order to be able to compare studies, it would be important to determine if the asymptotic phenomenon is similar with animal and human subjects. To do so, the same exposure conditions would have to be equal for the same signals. From these data, it would be possible to obtain equinoxious contours and to extrapolate result from humans to animals and vice versa.

From a pathophysiological point of view, some studies have demonstrated that ATS can predict the upper bound of PTS (Bohne and Clark, 1982, 1987; Bohne, 1983). Thus ATS studies would allow associations between given amounts of TTS and risk of damage. In an equinoxious context, it would be possible to eliminate conversion problems between TTS and the parameters of the noise signals. For example, we could study the evolution of TTS in dB as a function of peak levels of a given signal and determine, on animals, the level at which injuries begin to appear.

ATS studies on human subjects, however, present methodological and ethical problems. TTS has to be as low as possible (15-20 dB), in order to avoid any permanent damage. It has been shown in many studies (Mills, 1976; Hétu and Trémolieres, 1977; Hétu and Boudreault, 1978; Trémolières and Hétu, 1980) that such an amount of TTS is always completely recovered. The amount of TTS also has to be higher than the audiometric margin of error, i.e., 5 dB (Robinson, 1960; Hétu, 1979). These upper and lower limits of TTS prescribe a progression of exposure levels until a maximal TTS of 15 dB is reached. This kind of methodology requires many preexperimental trials, which could take many hours distributed over many days.

Minimal TTS also means constraints for the study of the recovery process. Within a few minutes following the end of the exposure, TTS is low and may even be within the margin of error. It could be suggested that any TTS less than or equal to 5 dB be considered as being recovered. If this criterion is adopted, there is a risk of missing potential damage other than loss of sensitivity, such as loss of frequency selectivity, recruitment, alteration of the differential limen in intensity, etc. (McFadden and Plattsmier, 1982; Laroche and Hétu, 1988).

Exposure duration also represents a methodological
constraint. Subjects have to be exposed during long periods of time (8–150 min). This could be difficult to tolerate, considering the fact that they must remain motionless during the exposure.

In summary, the methodological approach presented in this study offers a great potential for the study of the effects of noise on hearing but involves many constraints.

C. Practical implications

The present results bear practical implications regarding damage risk criteria recently put forward in different countries, as shown in Table III. Each of these criteria differ in terms of the maximal peak pressure allowed, the corresponding $L_{\text{Aeq,8 h}}$, and the limits on TTS. For the damage risk criteria and regulations based on an energy rule, the curve proposed by Smoorenburg (1980), which corresponds to an $L_{\text{Aeq,8 h}}$ of 85 dB, was used to determine the difference between the peak level and the $L_{\text{Aeq,8 h}}$. This 21-dB difference was then added to each allowable $L_{\text{Aeq,8 h}}$ to find the corresponding peak level. Because CHABA and the Canadian regulation are not based on energy, it is difficult to derive the corresponding $L_{\text{Aeq,8 h}}$.

It is interesting to compare the above results with these criteria. For example, according to CHABA (1968), a peak pressure of 128 dB would be allowed for an exposure to 9000 impacts (which is about 150 min at a rate of 1 pps) with a $B$ duration of 470 ms. This is a pressure about 24 dB higher than that implied by our exposure conditions. Because CHABA and the Canadian regulation are not based on energy, it is difficult to derive the corresponding $L_{\text{Aeq,8 h}}$

Our results correspond more closely to the limits proposed by Smoorenburg (1980), Pfander (1975), Pfander et al. (1980), and the French legislation (Arrêté du 12 août, 1975). In the case of Smoorenburg, this is true, providing that the spectral content is similar to that of our signals. Indeed, according to the above criteria, levels of 106, 107, and 106 dB SPL are allowed, respectively, when subjects are exposed to 9000 impacts with a $B$ duration of 470 ms [or $C$ duration of 157 ms (Smoorenburg, 1980) ]. Peak levels, for the criteria of Smoorenburg and the French legislation, are based on an permissible $L_{\text{Aeq,8 h}}$ of 85 dB or $L_{\text{Aeq,8 h}}$ of 85 dBA, respectively. Our exposure conditions are close to these limits; they are 81 and 83 dBA. One can assume that a $L_{\text{Aeq,8 h}}$ of 85 dBA would limit the amount of TTS between 15 and 20 dB. However, if the amount of ATS predicts the amount of PTS acquired after 10 or 20 years of work in noisy places (Mills, 1979), a limit at such $L_{\text{Aeq,8 h}}$ would not prevent all hearing loss from occurring.

There is no agreement on the physical parameters of impulse noises that determine noxiousness. Of the peak level or the total energy, which is more important in determining the noxiousness? Which other parameters could be considered in damage risk criteria?

Few clues could be found in the literature to answer these questions. The equal energy principle, tested by many researchers (Ceypek et al., 1975; Taylor and Pelmear, 1976; Henderson et al., 1982; Roberto et al., 1985), does not always seem to account for the noxiousness of impulse noises. A recent study done by Patterson et al. (1987) confirms the fact that the predictive value of the peak pressure or the energy is limited. Studies would have to be done on other parameters, such as the spectral content, the $B$ duration, and the pressure-time profile, to derive damage risk criteria that are truly safe for hearing. Price’s studies (1981) have already shown that it is important to consider the spectral content. Experiments done in our laboratories confirm the importance of this parameter (Laroche et al., 1986).

### Table III: Comparison of damage risk criteria with respect to (1) allowable peak levels for exposure to 9000 impacts at a rate of 1 pps, (2) the corresponding $L_{\text{Aeq,8 h}}$, and (3) the limits on TTS.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Allowable peak pressure (dB peak)</th>
<th>Corresponding $L_{\text{Aeq,8 h}}$ (dB)</th>
<th>Limits on TTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHABA (1968)</td>
<td>128</td>
<td>...</td>
<td>TTS$_1$ = 10 dB; $f &lt; 1$ kHz</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>15 dB; $f = 2$ kHz</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>20 dB; $f &gt; 3$ kHz</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>for 5% of the population</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>95% recovery of TTS in 24 h</td>
</tr>
<tr>
<td>Pfander (1975)</td>
<td>107</td>
<td>86</td>
<td>TTS$_2$ = 15 dB; $\bar{f} = 1,2,3$ kHz</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>for 10% of the population</td>
</tr>
<tr>
<td>Smoorenburg (1980)$^a$</td>
<td>106</td>
<td>85</td>
<td>Nonapplicable</td>
</tr>
<tr>
<td>Code of practice U. K. (1972)$^b$</td>
<td>111</td>
<td>90</td>
<td>Nonapplicable</td>
</tr>
<tr>
<td>Canadian Legislation</td>
<td>140</td>
<td>...</td>
<td>Nonapplicable</td>
</tr>
<tr>
<td>(Canada Labor code, 1976)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>French Legislation (Arrêté du 12 août, 1975)$^b$</td>
<td>106</td>
<td>85</td>
<td>Nonapplicable</td>
</tr>
</tbody>
</table>

$^a$This criterion is based on an admissible $L_{\text{Aeq,8 h}}$.

$^b$These criteria are based on an admissible $L_{\text{Aeq,8 h}}$.
VI. CONCLUSION

The present data collected with human subjects exposed to impact noise confirm the results obtained previously on animals: An asymptote is reached with 1–2 h compared to 8–16 h for continuous noises. The mechanism responsible for this rapid onset of ATS could be the loss of rigidity or stiffness of stereocilia, which may be faster with impulse noises than with continuous noises due to a larger displacement of the basilar membrane.

Within the limits of the exposure conditions studied, the recovery process seems to be independent of the exposure duration. Too few data are yet available to try to explain this result.

Peak levels required to induce 15–20 dB of ATS are of the order of 102–104 dBA in this study. Comparison between these levels and those allowed by current damage risk criteria has demonstrated that many of them are too lax and could lead to PTS. Only Smoorenburg, Pfander, and the French legislation are in good agreement with our results. However, it is not yet demonstrated that a criterion based on energy will accurately predict the risk of PTS. Many other physical parameters, such as the effect of the spectral content, the B duration, and the pressure-time profile, would have to be investigated before setting safe criteria. To achieve this, the study of ATS seems promising. This type of methodology would allow an easy comparison between studies done on animals or with human subjects. Despite numerous constraints, this appears feasible with human subjects based on the present results. The application of this method to animal studies could be a step forward in coordinating research efforts for understanding and predicting the effects of impulse noise on hearing.

ACKNOWLEDGMENTS

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Arrêté du 12 août 1975 "fixant une méthode de mesure des niveaux sonores en milieu de travail en vue de la protection de l'audition" (J. O. du 12 octobre 1975).


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