Estimates of blast injury and acoustic trauma zones for marine mammals from underwater explosions

Darlene R. Ketten

Department of Otology and Laryngology, Harvard Medical School, Boston, Massachusetts, 02114, U.S.A. Email: eplunix!drk@.mit.edu

Summary

The physical parameters of two underwater detonations of Class A explosives (TNT derivatives with fast rise-time waveforms) are analyzed for their potential to induce blast injury and acoustic trauma in marine mammals. Anatomical differences between marine and land mammal ears that may affect the incidence and severity of acoustic trauma and the basic concepts of temporary versus permanent threshold shifts are summarized. Overpressure calculations are combined with experimental data from the literature to calculate theoretical zones for acute trauma, permanent hearing loss, and temporary threshold shifts for mid-water explosions at two charge weights.

Key words: blast injury, threshold shift, ship shock, Cetacea, acoustic trauma

Introduction

This paper is a theoretical overview. In it, the physical parameters of two hypothetical hazards, large and small underwater explosions, are outlined and analyzed for their potential interactions with marine mammal ears. For the last decade, there has been growing concern about the effects of man-made sounds in the oceans. These concerns are timely, and more stringent reviews are underway of many activities that could acoustically impact marine life. However, at the moment, we have no direct experimental evidence or controlled measures of energy levels that induce blast or acoustic trauma in marine mammals.

How labile is the ear of any marine mammal species? Given that marine mammals are highly aural animals, we expect serious impact from even minor acoustic trauma. The alternative view argues that highly prized resources, including sensory systems, are generally well protected. To date, there is no definitive study of temporary or permanent threshold shifts in any marine mammal. Pinniped and cetacean ears provide an interesting functional paradox: they are essentially land mammal ears immersed in a biologically rich but acoustically harsh environment. As marine mammals evolved from land mammals, their ears coupled a terrestrial blueprint to extensive adaptations for rapid pressure changes and large concussive forces. On one hand, they have basically acoustically fragile land mammal ears. On the other hand, they evolved in a high noise environment, and some adaptations; e.g., those that prevent inner ear barotrauma, may deter noise and shock trauma. Little is known on the effects of intense sounds and

Sensory Systems of Aquatic Mammals (1995)
R.A. Kastelein, J.A. Thomas and P.E. Nachtigall (editors)
De Spil Publishers, Woerden, The Netherlands ISBN 90-72743-05-9

concussive forces on hearing in water for virtually any species. Evidence from human divers is largely anecdotal and often contradictory. There are few audiograms for marine mammals and no published data on temporary or permanent threshold shifts. Thus, at this stage, there are no direct measures of underwater acoustic trauma for any marine mammal and detailed descriptions of the ear are available for fewer than 20% of cetacean and pinniped species.

The "experiment" presented here is not, in any sense, a definitive analysis of marine mammal acoustic hazards, nor is it an expert opinion on trauma. Rather, it provides an overview of the physiologic aspects of acoustic trauma and a discussion of the relevance and limitations of currently available data (primarily from land mammal ears) for predicting the effects of man-made sounds on marine mammals. The scope is limited to two tangible impacts: shock wave and acoustic trauma from blast zones. The values calculated are impact estimates only and are largely speculative. Conservative estimates of the zones of potential lethal, sublethal, transitional, and low to zero impacts are extrapolated from arbitrary charge weights, published anatomical and physiological data on blast injury, and related anatomical data on marine mammal ears. It does not attempt to assess any of the possible, equally grave, but subtle, effects sound can have; e.g., disruption of communication, breeding behaviour, or navigation. Regrettably, there is little direct factual evidence that can be brought to bear on any of these issues. In essence, this experiment is directed not at addressing a single, well defined question but at construing our current, relatively small volume of knowledge into a more coherent form for objective discussion of potential marine acoustic impacts.

Materials and methods

The following model assesses the theoretical hazards of impulse pressures and peak overpressures that marine mammals may encounter within a 15 km radius of multi- tonnage mid-water explosions. The specific detonations addressed are 1200 lbs (544 kg) and 10000 lbs (4535 kg) charges of Class A explosives commonly used in inshore and offshore industrial and military activities (DOC, 1993; Ketten *et al.*, 1993; Czaban *et al.*, 1994; Reeves and Brown, 1994). Class A explosives are TNT-derivative water-gel compounds that generate complex, fast-rise time impulse waveforms. Detonation velocities typically range 4,000 - 10,000 m/sec. The two charge weights chosen for this model span the conventional range used in demolition, construction, and live-fire military testing (DOC, 1993, Ketten *et al.*, 1993). Several simplifying assumptions are made in the model:

1. detonation occurs at ≥ 100m depths.
2. bottom depth is ≥ 20 times the detonation depth.
3. the bottom is thick sediment with no discernible terrain.

A charge depth < 5% total water depth is largely a practical constraint. Given these assumptions, the pressure spread is spherical for 100 m, becoming planar as surface influences emerge. This model more closely approximates military ship shock trials than near shore industrial activity. In- and nearshore explosions imply multiple irregular influences; e.g., bottom topography, artificial structures, etc., that rapidly distort the wave front and produce complex cancellation-enhancement patterns.

Ship shock trials are widely accepted procedures for live-fire testing of new or existing ship designs. Because they are highly regulated test procedures, they provide con-

trolled measures of a ship's ability to withstand a near-miss underwater explosion, and, as a consequence, also provide empirical data (Czaban *et al.*, 1994) which, in this paper, act as controls for theoretical estimates from ideal models.

Charge parameters in this model are based on published manufacturer's standards for HBX-1, a common ship shock test explosive in North American trials (DOC, 1993; Reeves and Brown, 1994):

Explosive	HBX-1
Charge Weights	1,200/10,000 lbs (544/4,535 kg) (non-sequential)
TNT equivalents (HBX factor = 1.12)	1,344/11,200 lbs (609/5,080 kg)
Detonating velocity	8,800 m/sec

Model analyses include calculated pressures and overpressures at eight arbitrary distances, ranging 1 to 10,000 m from the source, and estimated distances from the source for five zones that cover major stages of blast injury and trauma. These stages are designated lethal, sublethal with permanent threshold shifts, temporary to permanent threshold shift transitions, and minimal to zero injury transitional zones. Related data from previous studies are presented in an overview of marine mammal hearing and in a brief summary of known factors for acoustic trauma and blast injury in mammals.

Results

Marine mammal ears

There are three essential parts to the mammalian auditory periphery: 1) an outer ear which captures sound, 2) a middle ear which filters and amplifies, and 3) the inner ear (cochlea) which is a band-pass filter and electro-mechano-chemical transducer of sound into neural impulses. Although marine mammal ears clearly follow the land mammal blueprint, they have both gross and microscopic, aquatic-related adaptations at all auditory system levels (Ketten, 1992, 1993).

External (outer ear) canal adaptations range from extreme (possibly dysfunctional) in Cetacea to broad, essentially terrestrial, air-adapted canals in the true-eared seals and mustelids. Four outer ear adaptations are common to all cetaceans: there are no pinnae, no air-filled external canals, no encapsulated pneumatized areas, and exceptionally dense temporal bones. These appear to be correlated largely with locomotion and diving; i.e., pinnae would provide hydrodynamic drag, and thin-walled, air-filled, bony chambers would be a liability in rapid, deep dives. However, these adaptations are most derived in echolocating cetaceans, where they subserve an acoustic function (Ketten, 1992). In odontocetes, the external ear canals are completely occluded by wax and debris, ending in a blind pouch that does not contact the middle ear. The temporal bones are suspended by ligaments in a peribullar sinus filled with a spongy mucosa. This ligament-mucosa complex isolates the ear from bony sound conduction and aligns the middle ear cavity with two orthogonal, specialized fatty channels. Anatomical and behavioral studies suggest these discrete fatty tubes are low impedance sound channels (Brill *et al.*, 1988; Ketten, 1992 for review). Mysticetes similarly have occluded external canals, but explicit tissue channels to the ear have not been identified. Pinniped and mustelid ears are less derived than cetacean. The external pinnae are reduced or absent. Ear canal diameter and closure mechanisms vary widely in pinnipeds, and the exact role of the canal in submerged hearing has not clearly been determined.

As in the external ear, most middle ear adaptations in marine mammals appear to be related to diving. The dense-walled middle ear cavity in whales is lined with a thick, distensible *corpus cavernosum* which may regulate middle ear cavity volume or pressure. The Eustachian tube in both seals and dolphins is broad, fibrous, and resists collapse, thereby assuring rapid middle ear equalization and decreasing the probability of implosive injury or middle ear barotrauma. Ossicular configurations and stiffness characteristics are highly species-specific and therefore may reflect frequency specializations.

As in other mammals, whale inner ears are fluid-filled membranous labyrinths that house two sensory organs: a three-ringed vestibular system (for balance) and a spiral cochlea (for hearing). The vestibular system is exceptionally small in whales and dolphins, and vestibular innervation is reduced proportionately; i.e., < 10% of cetacea VIII-th nerve fibers are vestibular, compared to 40% in most mammals (Gao and Zhou, this volume). Vestibular down-sizing may be a corollary of fused cervical vertebrae in whales, or alternatively, a valuable adaptation for rapid, continuous rotations in water. Maculae are present, implying that geotactic and linear acceleration cues at least are perceived.

Dolphin, baleen whale, and seal inner ears have the same general format as other mammalian ears (Fig. 1a); i.e., a fluid-filled, tripartate spiral with a resonating membrane supporting mechano-sensory receptors (Ketten, 1992; Ketten, 1993). Mammalian basilar membranes are essentially tonotopic bivariate resonators built of repeated, relatively uniform modules. The range of the stiffness and mass of these modules in each ear determines the range of cochlear resonances and therefore the hearing limits of that ear. Because all mammalian basilar membranes have a similar cellular structure, most interspecific differences in stiffness and mass are determined largely by thickness and width distributions along the membrane's length. Highest frequencies are encoded in the base of the spiral where the membrane is narrow and stiff. Progressively lower frequencies are encoded as the membrane becomes broader and more pliant towards the spiral apex. The primary sound transducer is the Organ of Corti (Fig. 1b), an array of hair cells with stereocilia that are deflected when the basilar membrane is deformed by a pressure wave.

Membrane distortions depend on the interaction of intensity-frequency distributions of incoming sounds with the resonance characteristics of the basilar membrane. In a responsive membrane region, as the cilia bend, graded chemical changes in the hair cells produce a depolarization in the auditory nerve, and impulses coding spectral and temporal patterns of the sound are sent to the brain. The absolute range of frequencies encoded by the ear varies naturally for each species according to its average distribution pattern of inner ear stiffness and mass characteristics. Some mammals hear well into the ultrasonic range (> 20 kHz); others, into the infrasonic (<50 Hz) (Fay, 1988). In addition to differences in absolute range, species vary in their sensitivity at each frequency band. Species ranges are fixed by basic structure; however, acuity varies widely by individual according to the health or integrity of the ear.

Specializations in marine mammal ears relate not only to extended frequency-intensity ranges, but also to the differences in acoustic power and transmission characteristics for waterborne versus airborne sound.

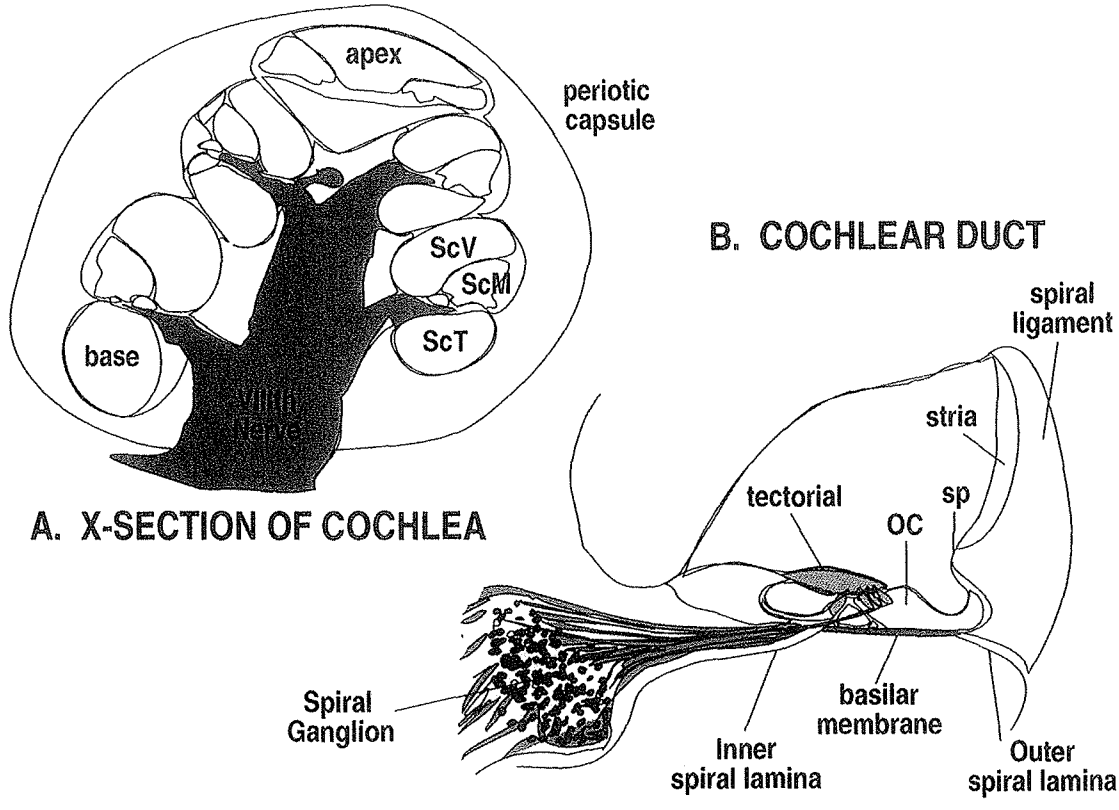

Figure 1. Schematized marine mammal inner ear (a) and details of the cochlear duct (b) containing the Organ of Corti (OC) which is the cochlear structure most directly and frequently impacted by sound. (Sc = scala; M = media; V = vestibuli; T = tympani; sp = spiral prominence)

The odontocete inner ear, like that of bats, is primarily adapted for echolocation; the dolphin is capable of producing, perceiving, and analyzing ultrasonics. These ears have exceptional frequency discrimination abilities but are comparatively poor at perceiving lower frequency sounds (Au, 1993). Balaenid ears are adapted to the other end of the spectrum; i.e., they hear poorly over 20 kHz but are probably exceptionally acute at frequencies in the 10-100 Hz range (Ketten, 1992; 1993). Some cetaceans produce source levels as high as 180 to 220 dB re 1 µPa (Richardson *et al.*, 1991; Würsig and Clark, 1993; Au, 1993). Pinnipeds are equally variable. Some, like the high-frequency harbour seal (*Phoca vitulina)*, are effectively - acoustically - sea-going cats; others, like the aptly named elephant seal (*Mirounga angoustirostris*), appear to have low to infrasonic adapted ears (Ketten, pers obs.).

In summary, marine mammals are acoustically diverse with wide variations not only in ear anatomy but also in hearing range and sensitivity. Both mysticetes and odontocetes appear to use soft tissue channels for sound conduction to the ear, which may decrease received acoustic power. Whale middle and inner ears are most heavily modified structurally from those of terrestrial mammals in ways that accommodate rapid pressure

changes. The end-product is an acoustically sensitive ear that is simultaneously adapted to sustain moderately rapid and extreme pressure changes, and appears capable of accommodating acoustic power relationships several magnitudes greater than in air. It is possible that these special adaptations coincidentally may provide protective mechanisms that lessen the risk of injury from high intensity noise, but no behavioural or psychometric studies are available which directly address this issue.

Noise trauma

For this discussion, sound-related trauma is divided into two simplistic divisions: lethal and sublethal impacts. Lethal impacts are those that result in the immediate death or serious debilitation of the majority of animals in or near an intense source; i.e., profound injuries from shock wave or blast effects. Lethal impacts are not, technically, pure acoustic trauma; they are discussed at the end of this section. Sublethal impacts in which a hearing loss is caused by exposures to perceptible sounds are correctly termed acoustic or noise derived trauma. In these cases, some sound parameters exceed the ear's tolerance; i.e., auditory damage occurs from metabolic exhaustion or over-extension of one or more ear components. In some cases, sublethal impacts may ultimately be as devastating as lethal impacts, causing death through impaired foraging or predator detection, but they do not carry the immediate, broad devastation of a blast injury.

To determine whether an animal is subject to hearing loss from a particular sound requires understanding how its hearing sensitivity interacts with that sound. Basically, if you can hear a noise, at some level it can damage your hearing by causing decreased sensitivity. The minimal level at which a sound is perceived is the threshold. If an individual requires a significantly greater intensity (than the species norm) to perceive a particular frequency, there is a hearing deficit marked by a threshold shift. Noise induced threshold shifts are a moderately well investigated phenomenon for air-adapted ears (Lehnhardt, 1986; Lipscomb, 1978; Richardson *et al.*, 1991 for detailed reviews). Any noise at sufficient levels will significantly shift hearing thresholds, but all noises at the same levels do not cause equivalent shifts. The important issue is whether the impact produces a recoverable (TTS - Temporary Threshold Shift) or permanent (PTS - Permanent Threshold Shift) loss.

Major research efforts have been directed at understanding the relationships of frequencies, intensities, and duration of exposures in producing damage; that is, what sounds, at what levels, for how long, or how often will produce temporary versus permanent hearing loss. A comprehensive discussion of the complexities involved in TTS versus PTS is not possible in the scope of this paper. Excellent reviews of noise trauma mechanisms are available in Lehnhardt (1986); Miller *et al.* (1987); and Liberman (1990). Only the briefest, and therefore necessarily simplistic, introduction is given here. Two fundamental effects are generally accepted:

1. for pure tones, the loss centers around the incident frequency.
2. for all tones, at some balance of noise level and time, the loss is irreversible.

TTS may be broad or punctate, according to source characteristics, subject hearing sensitivity, and relative health of the ear impacted. The majority of studies have been conducted with cats and rodents, using relatively long duration stimuli (> 1 hr) and mid to low frequencies (1-4 kHz) (Lehnhardt, 1986). The extent and duration of such threshold shifts generally are proportional

to the extent of inner ear damage. Virtually all studies show that losses from short to moderate term, narrowband stimuli (< 1 hr, CF ± 2 kHz) are centered around the peak spectra of the stimulus and are largely recoverable although recoverability is strongly influenced by individual sensitivity of the subject (Henderson *et al.*, 1983; Miller *et al.*, 1987). Histologic data from impacted ears showed very discrete, localized cell damage at short-duration exposures progressing to widespread lesions with increasing intensity and bandwidth (Liberman, 1987). Recoverable (TTS) to non- recoverable (PTS) losses were characterized by a progression from simple stereocilia fatigue to loss of hair cell bodies to broadening patches of strial, ligament, and neuronal degeneration.

In the typical air-adapted ear, a pure tone with an intensity 80 dB (re 20 μPa) higher than the normal minimum response threshold for that frequency is generally sufficient for stereocilia and hair cell damage. Most damaged cells will recover from this impact, but repeated exposures compound the insult. Recovery periods can vary from hours to weeks among individuals. This finding led to the current allowable limit of 90 dB re 20 μPa for human workplace exposures (Lehnhardt, 1986); i.e., a 90 dB limit for all signals reduces the probability of encountering a broadband signal with components ≥ 80 dB over threshold at the most sensitive frequency range (500-5000 Hz) for humans.

Repeated exposures to TTS level stimuli without adequate recovery periods can induce permanent, acute threshold shifts (PTS). Liberman (1987) showed that losses were directly correlated with levels of damage to hair cell stereocilia, the hair cell bodies, stria vascularis, the spiral ligament, and nerve fibers. The duration of a threshold shift, is correlated with both the length of time and the intensity of exposure. This does not, however, mean that TTS and PTS are simply gradations of the same damage mechanisms. A major complication in any TTS versus PTS comparison is that signal rise- time and duration of peak pressure are significant factors in PTS. If the exposure is short, hearing is recoverable; if long, or has a sudden, intense onset and is broadband, hearing, particularly in the higher frequencies, can be permanently lost. Experimentally, PTS is induced in air with multi-hour exposures to narrowband noise. In humans, PTS typically results accidentally from protracted, repeated intense exposures (continuous occupational background noises) or sudden onset of intense sounds (repeated gun fire). Sharp rise-time signals have been shown also to produce broad spectrum PTS at lower intensities than slow onset signals both in air and in water (Lipscomb, 1978; Lehnhardt, 1986; Liberman, 1987).

TTS has been produced in humans with underwater sound sources at levels of 150 - 180 dB re 1 μPa for frequencies between 700 - 5600 Hz (Smith and Wojtowicz, 1985; Smith *et al.*, 1988). These intensities are similar to those that induce TTS in air in humans because: a) intensity is a power ratio that depends on sound speed and density of the medium and b) the absolute value depends on the reference pressure used. The following calculations illustrate this difference:

$$I = pv = p^2/c\rho$$
$$I_{air} = p^2/(340\ m/sec)(0.0013\ g/cc)$$
$$I_{water} = p^2/(1530\ m/sec)(1.03\ g/cc)$$

where I = intensity, v = vibration velocity, p = sound pressure, c = the speed of sound in that medium, ρ = the density of the medium. Appropriate comparisons can be made in terms of acoustic power (W/m^2); however, in many texts the reference pressures will be

dB re 20 μPa for airborne sound versus dB re 1 μPa for in-water thresholds. Ears are essentially sound energy difference detectors. If an auditory percept depends on the same level of intensity in air and in water, the underwater sound level that will produce an equivalent sensation must be 61.5 dB greater than its airborne counterpart; i.e., 150 dB re 1 μPa ≈ 90 dB re 20 μPa.

Blast injury

Simple, intensity related loss is not synonymous with blast injury. Blast injuries can be divided into three groups based on the severity of the symptoms:

1. MILD - Recovery
   Pain, vertigo, tinnitus, hearing loss, tympanic tears

2. MODERATE - Partial loss
   Otitis media, tympanic membrane rupture, tympanic membrane hematoma, serum or blood in middle ear, dissection of mucosa

3. SEVERE - Permanent loss or death
   Ossicular fracture or dislocation, round/oval window rupture, CSF leakage into middle ear, cochlear and saccular damage.

Moderate to severe losses result most often from blasts, extreme intensity shifts, and trauma in which explosions or blunt cranial impacts cause sudden, massive systemic pressure increases and surges of circulatory or spinal fluid pressures (Schuknecht, 1993). Hearing loss in these cases results from an eruptive injury to the inner ear; i.e., with the rarefactive wave of a nearby explosion, cerebrospinal fluid pressures increase and the inner ear window membranes blow out due to massive surges in the inner ear fluids. Inner ear explosive damage frequently coincides with fractures in the bony capsule of the ear or middle ear bones and with rupture of the eardrum. Although technically a pressure induced injury, hearing loss and the accompanying gross structural damage to the ear are more clearly thought of as the result of the inability of the ear to accommodate the sudden, extreme pressure differentials and over-pressures from the shock wave.

At increasing distance from the blast, the effects of the shock wave lessen. Even though there is no overt tissue damage, mild damage with some permanent hearing loss occurs (Burdick, 1981, in Lehnhardt, 1986). This type of loss generally is called an asymptotic threshold shift (ATS) because it derives from a saturation effect. Like TTS, the hair cells are damaged, but as in any PTS, no recovery takes place. Because ATS depends on complex interactions of rise- time and waveform, not simply intensity at peak frequency, asymptotic hearing losses typically are broader and more profound than simple PTS losses.

There is no well-defined single criterion for sublethal ATS (Roberto *et al.*, 1989), but eardrum rupture, which is common to all stages of blast injury, has been moderately well investigated. Rupture *per se* is not synonymous with permanent loss; eardrum ruptures have occurred at as little as 2.5 kPa overpressure. However, the incidence of tympanic membrane rupture is strongly correlated with distance from the blast (Kerr and Byrne, 1975). As frequency of rupture increases, so does the incidence of permanent hearing loss. In zones where > 50% tympanic membrane rupture occurred, 30% of the victims had long-term or permanent loss.

Recent experimental work showed that weighted sound exposure level is a more robust predictor of permanent loss than peak pressure (Patterson, 1991). Data with weighted levels are rare; overpressure data are more common and are highly correlated with received levels (Roberto *et al.*, 1989). Table 1 summarizes the experimental results on overpressures associated with > 50% rupture. The data indicate complex and fast rise-time sounds cause ruptures at lower overpressures than slow rise-time waveforms, and smaller mammals are injured by smaller overpressures than larger animals. Of the animals listed, sheep and pigs have ears anatomically closest to those of whales and seals. The air-based data for pigs and sheep imply that overpressures > 70 kPa are needed to induce 100% tympanic membrane rupture. However, cross-study comparisons and extrapolations are risky because of radically different experimental conditions as well as differences in power transmission in air and water.

Data available for submerged terrestrial and aquatic vertebrates imply that lower pressures in water than in air induce serious trauma (Myrick *et al.*, 1989; Richardson *et al.*, 1991). For submerged terrestrial mammals, lethal injuries occurred at overpressures > 55 kPa (Yelverton, 1973, in Myrick *et al.*, 1989; Richmond *et al.*, 1989). In a blast study in Lake Erie using Hydromex, a TNT-derivative explosive, the overpressure limit for 100% mortality for fish was 30 kPa (McAnuff and Booren, 1976), suggesting overpressures between 30 and 50 kPa are sufficient for a high incidence of severe blast injury. Minimal injury limits in both land and fish studies coincided with overpressures of 0.5 to 1 kPa.

Marine mammal blast injuries

Very few reports of blast induced trauma in marine mammals detail injuries to the head region. Bohne *et al.* (1985) reported on inner ear damage in Weddell seals (*Leptonychotes weddelli*) that survived blasts, but they did not have access to exposure levels or number of exposures per animal. Scattered reports of opportunistic examinations of animals exposed to large blasts include one on sea otters (*Enhydra lutris*) with extensive trauma from nuclear explosions (cited in Richardson *et al.*, 1991). The study concluded that peak pressures of 100-300 psi were invariably lethal.

Recently, several humpback whales (*Megaptera novaeangliae*) exposed to TOVEX blasts were shown to have severe blast injuries (Ketten *et al.*, 1993; Lien *et al.*, 1993). TOVEX, like Hydromex, is a TNT clone explosive similar to HBX-1 with an average detonation velocity of 4,500 m/sec. Received levels in the whales could not be calculated with confidence; however, the charge weights associated with the injuries ranged between 1700 to 5000 kg. The animals died within three days of the blasts, and the extent of the injuries implied they were close to the blast site. Mechanical trauma in these ears included round window rupture, ossicular chain disruption, bloody effusion of the peribullar spaces, dissection of the middle ear mucosa with pooled sera, and bilateral periotic fractures (Fig. 2). These observations are consistent with classic blast injuries reported in humans, particularly with victims near the source who had massive, precipitous increases in cerebrospinal fluid pressure and brain trauma. There was no evidence of ship collision or prior concussive injury in these whales, and no similar abnormalities were found in ears from humpback whales not exposed to blasts.

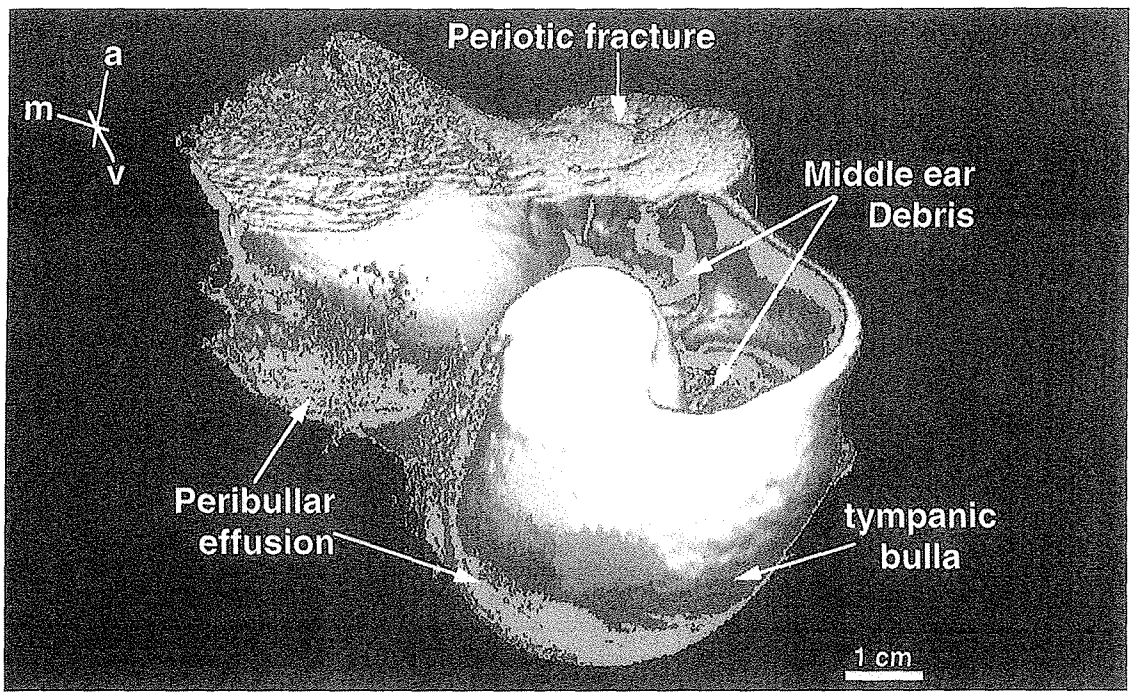

Figure 2. A three-dimensional reconstruction from CT scans of an ear from a humpback whale (*Megaptera noveaengliae*) with blast injuries shows multiple fractures throughout the periotic, which are consistent with intracochlear blood. Blood, serum, and cellular debris of both intra- and extra-cochlear origin filled the middle ear and surrounding peribullar region (reproduced with permission, Ketten *et al.*, 1993).

Table 1. Peak overpressures producing 50% tympanic membrane rupture (Phillips *et al.*, 1989; Richmond *et al.*, 1989; Bruins and Carwood, 1991)

Species	Peak pressure (kPa)	Waveform
Sheep (*Ovis spp.*)	165	Fast-rising, reflected shock
	75-129	Complex
Pig (*Sus spp.*)	152	Complex
Dog (*Canis familiaris*)	78	Fast-rising, reflected shock
	205	Complex
	296	Smooth-rising
	103	Static
Monkey (*Macaca mulatta*)	172	Fast-rising, reflected shock
	138-172	Fast-rising, incident shock
Human (*Homo sapiens*)	57-345	Fast-rising, incedent shock
Rabbit (*Sylvilagus spp.*)	64	Complex
	15	Fast-rising, incident shock
Guinea Pig (*Cavia porcella*)	51	Complex
	15	Fast-rising, incident shock

Discussion and conclusions

The few data points available for blast trauma in marine mammals indicate that external and middle ear structural adaptations in cetaceans and pinnipeds that may minimize barotrauma do not provide immunity to blast trauma. Considering the similarities of seal and whale ears to land mammal ears, it is not surprising that explosions, and the resulting intense transient sound field and shock wave, produce both blast injury and asymptotic acoustic trauma in marine animals. More important, even though the whale ear is a fluid-to-fluid coupler, all marine mammals retained an air-filled middle ear, which makes them subject to all ranges of compressive-rarefactive blast injury.

The level of impact from blasts depends on both an animal's location and, at outer zones, on its sensitivity to the residual noise. Important factors for trauma from explosive sources are the following:

1. Topography
2. Proximity of ear to the source
3. Anatomy and health of ear
4. Charge weight and type
5. Rise time
6. Overpressure
7. Pressure and duration of positive pressure phase

In the specified model, topographic effects are minimal. The bottom is effectively nonreflective; therefore, with a 100 m detonation, surface reflections are the primary interference source.

The health of individual ears that may be impacted cannot be estimated in advance. It is reasonable to assume an average distribution.

HBX-1 has a rapid detonation velocity (8800 m/sec), a relatively long peak pulse (5 to 10 msec), and a complex waveform. It is effectively an instantaneous onset, high peak pressure, broad spectrum blast. Therefore, the explosive front and acoustic signature of the proposed charges (1200 and 10000 lbs) are likely to cause equivalent damage to all species in the target area; i.e., individual hearing ranges and thresholds are largely irrelevant. High impedances and reflections at the air-sea boundary make calculations of received levels for surface animals unreliable, but it is possible that some individuals above or at the boundary layer will be partially protected.

Overpressure is based on a scaled distance normalized to charge weight in TNT equivalents (Bruins and Cawood, 1991) and is calculated as:

$$Z = s/m^{1/3}$$

$$P_{over(kPa)} = (11.3/z - 186/z^2 + 19210/z^3)(101.3)$$

$$P_{over(psi)} = P_{over(kPa)} / 6.9$$

where Z = scaled distance; s = distance (cm); m = charge mass (g) in TNT equivalents; $P_{over(kPa)}$ = overpressure (kPa); $P_{over(psi)}$ = overpressure (psi).
Peak pressure (psi) is calculated using a standard algorithm (Czaban *et al.*, 1994):

$$P = (8.22)(10^3)\,[W^{0.33}/s]^{1.15}$$

where W = weight (kg) in TNT equivalents and s = distance from source (m).

Table 2 compares these calculations for theoretical maximal pressures and overpressures with semi-empirical peak pressure data reported by Czaban *et al.* (1994) for HBX. Under stable field conditions, there is

Table 2. Pressure level estimates at the charge depth contour (100 m) for 1200 lb and 10000 lb HBX charges with simultaneous detonations (dB and measured maximum pressure data excerpted from Czaban *et al.*, 1994)

Distance (m)	dB	Maximum pressure (psi)	Theoretical pressure (psi)	Scaled distance	Overpressure (psi)	Overpressure (kPa)
-1200 lb Charge-						
1	257	95700.0	96121.8	1.18	167753.13	1157496.57
10	233	6790.0	6804.9	11.85	164.17	1132.75
100	206	481.0	481.8	118.48	1.38	9.52
500	188	75.5	75.7	592.42	0.27	1.89
1000	180	34.0	34.1	1184.83	0.14	0.96
4500	164	5.99	6.0	5331.75	0.03	0.21
5000	162	5.4	5.4	5924.17	0.03	0.19
10000	155	2.4	2.4	11848.34	0.01	0.10
-10000 lb Charge-						
1	263	216000.0	216577.0	0.58	1426284.83	9841365.34
10	237	15300.0	15332.5	5.82	1381.99	9535.76
100	211	1080.0	1085.5	58.15	3.49	24.08
500	193	170.0	170.5	290.77	0.55	3.81
1000	185	76.8	76.8	581.53	0.28	1.93
4500	168	13.6	13.6	2616.89	0.06	0.44
5000	167	12.1	12.1	2907.65	0.06	0.39
10000	159	5.4	5.4	5815.31	0.03	0.20

no significant difference between the empirical and theoretical values for peak pressure for either charge weight.

Although multiple parameters are associated with both lethal and sublethal effects, studies of lethal or compulsory injury zones for fast-rise time, complex waveforms agree on baseline criteria: 30-50 kPa peak overpressure in water and/or > 180 dB re 20 μPa in air (240 dB re 1 μPa in water) (McAnuff and Booren, 1976; Yelverton and Richmond, 1981; Phillips *et al.*, 1989: Richmond *et al.*, 1989; Myrick *et al.*, 1989). Given the pressure distributions calculated for HBX-1 detonations at 100 m, the 100% lethal impact zone limits for a 1200 lb source are between 40 m (absolute minimum - land mammal) and 300 m (conservative estimate based on otter psi data).

For a 10000 lb charge, the equivalent minima-maxima limits for the killing ground is 70 m to 800 m. Figures 3 and 4 illustrates the variables that are most relevant to these limit estimates. For either charge, within 100 m of the source, death would likely be immediate or would follow in days as a result of concussive brain damage, cranial fractures, hemorrhage, etc. for the majority of animals. For the remainder, permanent, profound deafness from massive inner ear trauma would seriously impair the animal's ability to survive (Lehnhardt, 1986; Bruins and Carwood, 1991; Ketten *et al.*, 1993; Schuknecht, 1993).

The criteria for differentiating PTS or ATS zones from TTS are less clear. The transitional lethal zones in which serious sublethal injury predominates are estimated as 100-500 m (1200 lb) and 150-900 m (10000 lb).

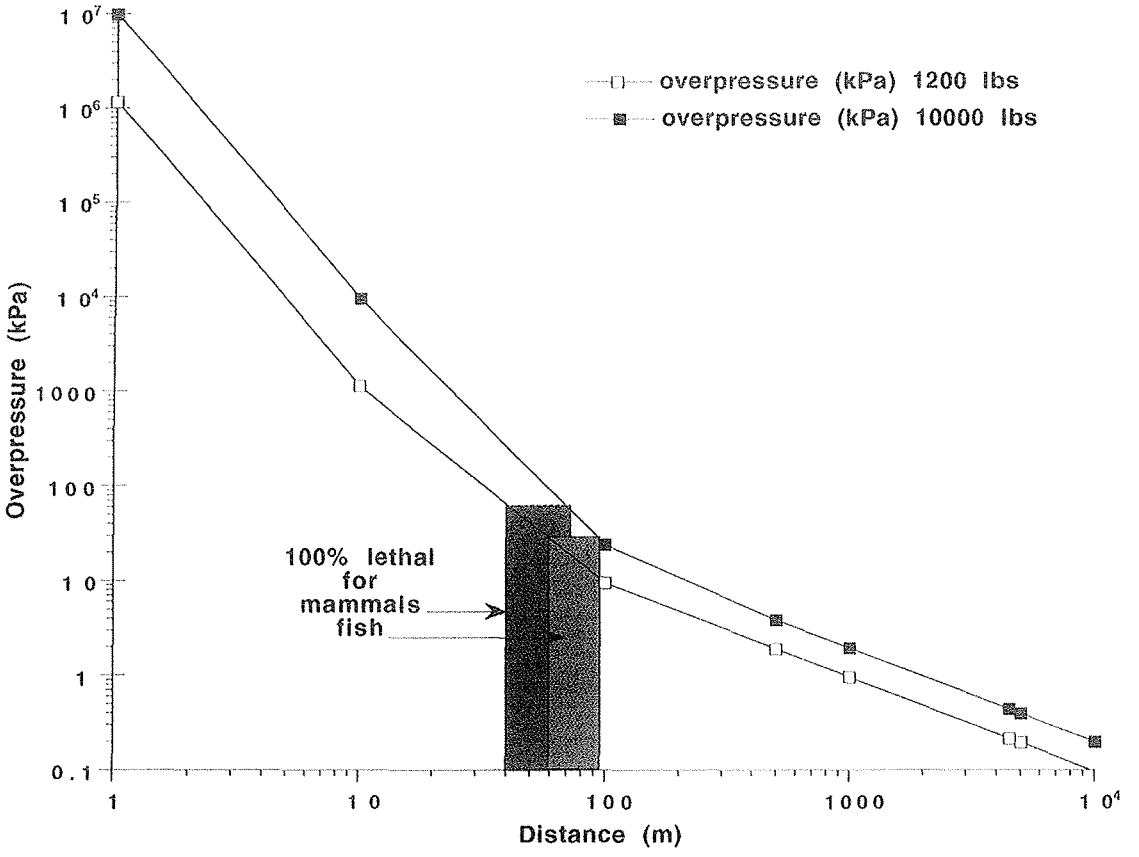

Figure 3. Overpressures at the 100 m depth contour are plotted for each charge weight versus distance from source. The gray vertical bars show the differences in distance from source for the outer edge of 1200 vs 10000 lb charge zones based on the following categories: 100% lethal overpressure for submerged land mammals (57 kPa) and 100% lethal overpressure for fish (30 kPa).

Beyond 500 meters and 900 m, the relative incidence of PTS to TTS largely depends on individual susceptibility. That is, the variables that will determine TTS vs PTS are highly species dependent and the animals are sufficiently mobile that no global, permanent division of these intermediate zones will be correct.

There is general consensus in the literature on the criteria for assigning an outer limit to the mild/moderate TTS zone. Generally, 5-15 psi is accepted as the value at which TTS and even minimal injury is rare (Yelverton and Richmond, 1981; Smith *et al.*, 1985, 1988; Myrick *et al.*, 1989; Roberto *et al.*, 1989). Therefore, between 1-10 km of the source the potential for any acoustic impact drops precipitously (Figs. 3 and 4). Because intensity decays exponentially, 5 km is a reasonably estimate of a safe outer limit for the 10000 lb charge for minimal, recoverable auditory trauma versus 2 km for the 1200 lb charge. Figure 4 provides a schematic of all of the zones extrapolated from the data outlined.

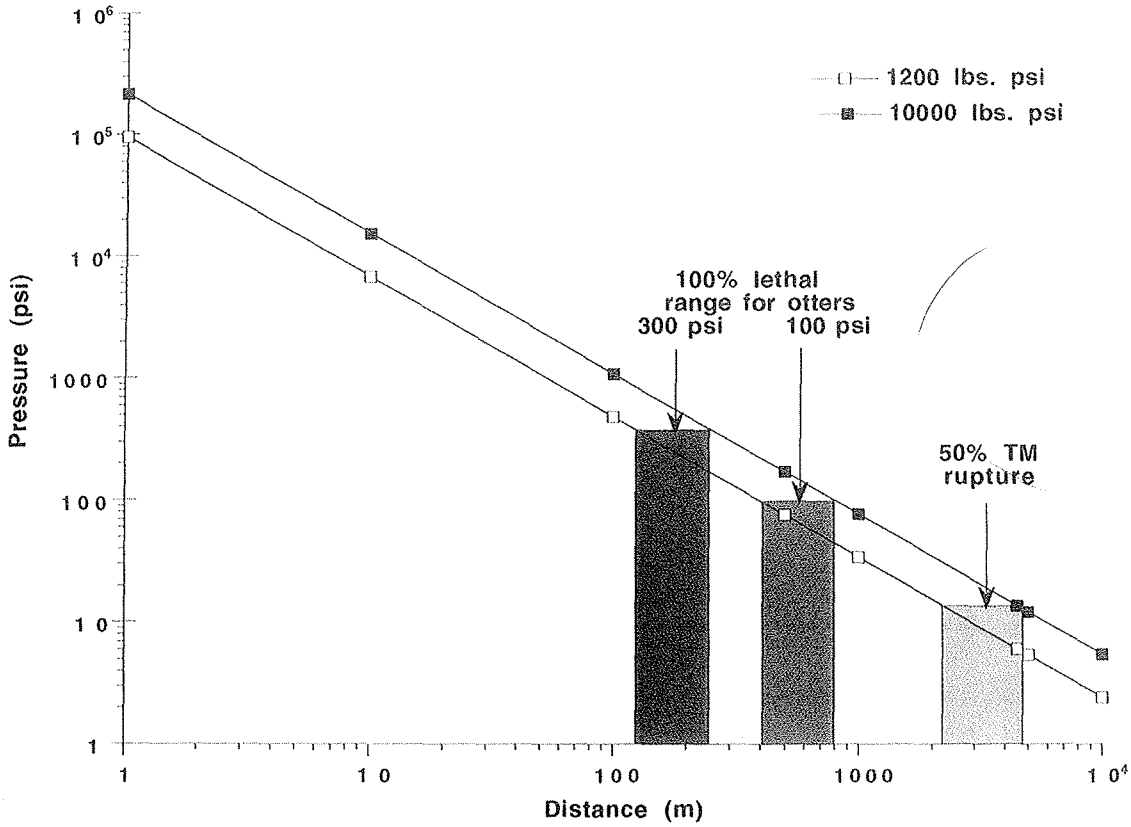

Figure 4. Peak pressures at the 100 m depth contour are plotted for each charge weight versus distance from source. The gray vertical bars show the differences in distance from source for the outer edge of 1200 vs 10000 lb charge zones based on reported pressures for 100% mortality in otters (*Enhydra lutris*) from blast and minimal peak pressure for 50% incidence of eardrum rupture.

It must be recalled here that these conclusions are highly speculative. They depend largely on limited anatomical comparisons. Depending upon its actual, and currently unknown, function, the same specialized structure in a marine ear could aggravate or ameliorate the effects of blasts. For example, dolphins have soft tissue sound conduction pathways and fibro-elastic tissue beds that acoustically isolate the inner ear. Either adaptation could significantly attenuate or enhance conduction of potentially damaging sounds. Without more explicit data, definitive guidelines for safe limits on underwater signals are not possible. Substantially more research is needed on all aspects of marine mammal hearing before wholly reliable estimates, much less solid answers, are available.

Acknowledgements

Moira Brown (East Coast Ecosystems), Randall Reeve, and Jan Czaban (NDHQ, Ontario) were instrumental in the creation of this report. Their assistance and encouragement are gratefully acknowledged, as is the coop-

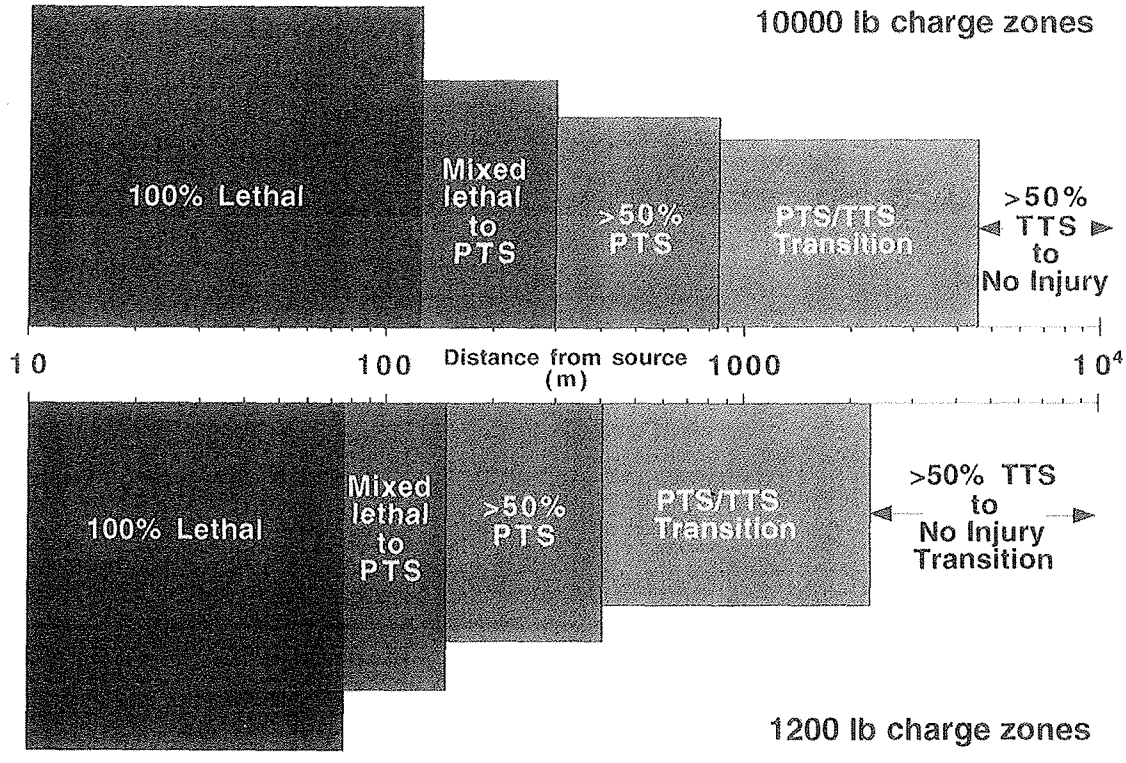

Figure 5. Impact zone summary for 1200 and 10000 lb charges. The 5 divisions shown represent distances from the source in which a) 100% mortality is expected (lethal), b) permanent hearing loss is common and some mortality is expected (sublethal), c) > 50% animals have some permanent hearing loss (all ranges of acoustic trauma), d) the majority of animals have temporary hearing loss but some permanent auditory damage will be found (transitional trauma), and e) little or no clinically detectable auditory damage (low impact). The zones are synthesized based on conservative extrapolations of data from fish, submerged terrestrial mammals, and humans.

eration of the Canadian National Defense Headquarters and Nodeco, Newfoundland. An early version of this paper was prepared at the request and with support from East Coast Ecosystems, Quebec, and was submitted previously to Bio Acoustics. Original research on mysticete and odontocete ears and participation in this symposium was supported by Office of Naval Research contracts N00014-92-J-4000 and N00014-93-1-0940.

References

Au, W.W.L. (1993) The Sonar of Dolphins, Springer-Verlag, New York, N.Y.

Bohne, B., Thomas, J., Yohe, E. and Stone, S. (1985) Examination of potential hearing damage in Weddell seals (*Leptonychotes weddelli*) in Mc Murdo Sound, Antarctica. Ant. J. of the U.S., 20, 174-176.

Brill, R.L., Sevenich, M.L., Sullivan, T.J., Sustman, J.D. and Witt, R.E. (1988) Behavioral evidence for hearing through the lower jaw by an echolocating dolphin, *Tursiops truncatus*. Mar. Mamm. Sci., 4 (3), 223-230.

Bruins, W.R. and Cawood, R.H. (1991) Blast injuries of the ear as a result of the Peterborough lorry explosion: 22 March 1989. J. Laryn. Otol., 105, 890-895.

Carder, D. and Ridgway, S. (1990) Auditory brainstem response in a neonatal sperm whale. J. Acous. Soc. Am., 88, (S1), s4.

Czaban, Z.J., Atchison, P. and Costain, J. (1994) Report on the underwater noise generated by the CPF shock trial. NDHQ document no. DSE64/CPF -3/94, Ottawa, Ontario.

DOC/NOAA (1993) Incidental take of marine mammals. 50CFR, pt. 228, Federal Register, 58(198), 53491-53497.

Fay, R.R. (1988) Hearing in Vertebrates: A Psychophysics Databook, Hill-Fay Assoc., Winetka, Ill.

Henderson, D., Hamernik, R.P., Salvi, R.J. and Ahroon, W.A. (1983) Comparison of auditory-evoked potentials and behavioral thresholds in the normal and noise-exposed chinchilla. Audiology, 22, 172-180.

Kerr, A.G. and Byrne, J.E. (1975) Concussive effects of bomb blast on the ear. J. Laryngol. Otol., 89, 131-143.

Ketten, D.R. (1992) The Marine mammal ear: Specializations for aquatic audition and echolocation. In: The Evolutionary Biology of Hearing (Eds. Webster, D., Fay, R. and Popper, A.), Springer-Verlag, New York, N.Y., 717-754.

Ketten, D.R. (1993) The Cetacean ear: Form, frequency, and evolution. In: Marine Mammal Sensory Systems (Eds. Thomas, J.A., Kastelein, R.A. and Supin, A.Ya.), Plenum Press, New York, N.Y., 53-75.

Ketten, D.R., Lien, J. and Todd, S. (1993) Blast injury in humpack whale ears: Evidence and implications. J. Acous. Soc. Am., 94, (3, pt. 2), 1849-1850.

Lehnhardt, E. (1986) Clinical Aspects of Inner Ear Deafness, Springer-Verlag, New York, N. Y.

Liberman, M.C. (1987) Chronic ultrastructural changes in acoustic trauma: Serial-section reconstruction of stereocilia and cuticular plates. Hearing Research, 26, 65-88.

Liberman, M.C. (1990) Quantitative assessment of inner ear pathology following ototoxic drugs or acoustic trauma. Toxicologic Pathology, 18 (1, pt. 2), 138-148.

Lien, J., Todd, S., Stevick, P., Marques, F. and Ketten, D. (1993) The Reaction of humpback whales to underwater explosions: Orientation, movements, and behavior. J. Acous. Soc. Am., 94,(3, pt. 2), 1849.

Lipscomb, D.M. (1978) Noise and Audiology, University Park Press, Baltimore, Md.

McAnuff, A.L. and Booren, R.T. (1976) Fish mortality study during underwater blasting operations in Lake Erie of Nanticoke, Ontario. E.T. North Bay Technical Resources, 131-142.

Miller, J., Nilsson, R., Flock, A. and Canlon, B. (1987) Effects of noise on the nervous system. In: The London Symposia (EEG Suppl. 39) (Eds. Ellingson, R., Murray, N. and Halliday, A.), Elsevier Sci. Publ., New York, N.Y., 364-378.

Myrick, A., Cassano, E. and Oliver, C. (1989) Potential for physical injury, other than hearing damage, to dolphins from seal bombs in the yellow fin tuna purse-seine fishery: Implications from open-water tests. NMFS report (non-published), Southwest Marine Fisheries Service, La Jolla, CA.

Patterson, J.J.H. (1991) Effects of peak pres-

sure and energy of impulses. J. Acous. Soc. Am., 90 (1), 205-208.

Phillips, Y., Hoyt, V., Mundie, T. and Dodd, K. (1989) Middle ear injury in animals exposed to complex blast waves inside an armored vehicle. Ann. Otol. Rhinol. Laryngol., 98, 17-22.

Reeves, R. and Brown, M. (1994) Marine mammals and the Canadian patrol frigate shock trials: A literature review and recommendations for mitigating the impacts. NDHQ/CPFPST, National Defense Headquarters, Ottawa, Ontario.

Richardson, W.J., Greene, C.R., Jr., Malme, C.I. and Thomson, D.H. (1991) Effects of noise on marine mammals. USDI/MMA/OCS study 90-0093, LGL Ecological Research Assoc., Bryan, Texas.

Richmond, D., Fletcher, E., Yelverton, J. and Phillips, Y. (1989) Physical correlates of eardrum rupture. Ann. Otol. Rhinol. Laryngol., 98, (5, pt. 2, S140), 35-41.

Ridgway, S. and Carder, D. (1990) Sounds made by a neonatal sperm whale. J. Acous. Soc. Am., 88 (S1), s6.

Roberto, M., Hamernik, R. and Turrentine, G. (1989) Damage of the auditory system associated with acute blast trauma. Ann. Otol. Rhinol. Laryngol., 98, 23-34.

Schuknecht, H. (1993) Pathology of the Ear, 2nd ed., Lea and Febiger, Philadelphia, Penn..

Smith, P.F. and Wojtowicz, J. (1985) Temporary auditory threshold shifts induced by twenty-five minute continuous exposures to intense tones in water. Naval Med. Rsch. and Devel. Cmd., USN, rpt. 1063, 1-13.

Smith, P.F., Wojtowicz, J. and Carpenter, S. (1988) Temporary auditory threshold shifts induced by ten-minute exposures to continuos tones in water. Naval Med. Rsch. and Devel. Cmd., USN, rpt. 1122, 1-10.

Würsig, B. and Clark, C. (1993) Behavior. In: The Bowhead Whale (Eds. Burns, J., Montague, J. and Cowles, C.), Society for Marine Mammalogy, spec. publ. no. 2, Allen Press, Lawrence, Kans..

Yelverton, J.T. and Richmond, D. (1981) Underwater explosion damage risk criteria for fish, birds, and mammals. J. Acous. Soc. Am., 70 (S1), s84.