Acute exposures to high-level noise impulses damage the cochlea via mechanical mechanisms that are associated with excessive displacements and stresses developed in the delicate epithelial tissues of the organ of Corti. Such damage has been discussed in the literature a number of times, and an especially clear description was provided by Davis [1]. Davis and his colleagues used continuous noise at levels of nearly 150 dB SPL at the eardrum. They noted that the Hensen cell attachments represent a mechanically weak link in the structural organization of the organ of Corti. This result was confirmed by Beagley [2], who illustrated the separation of cell junctions between the Deiter and Hensen cells following overstimulation. Since then, others (notably Spoendlin [3] and Voldrich [4]), also using high levels of continuous noise, have demonstrated lesions on the basilar membrane of an equivocal mechanical origin, including rupture of the basilar membrane and Reissners membrane. Spoendlin suggested intensities of around 125 dB SPL as the threshold for mechanically-induced lesions as opposed to metabolically-induced damage. However, the dependence of this rms sound pressure on the exposure duration is not clear. Spoendlin is in agreement with Davis and Beagley concerning the susceptibility to acoustic trauma of the Hensen cell attachments, but he further implicates the pillar cells and the medial attachments of the inner hair cell cuticular area as "weak spots." This paper attempts to provide a clear documentation of the morphological sequence of events which is eventually responsible for producing massive structural damage to the organ of Corti. Using blast waves as a vehicle, we will further attempt to qualitatively illustrate a fundamental difference in the way in which continuous and impulse noise may need to be evaluated when assessing the potential for producing trauma.

METHODS

Thirty-eight binaural chinchillas were used in this study; 6 controls and 32 experimental. From the 32 experimental animals, 6 were prepared for standard surface preparations [3,5] and the remaining 26 experimental and 6 control animals were prepared for Scanning Electron Microscopy (SEM). Each experimental animal was exposed at a normal incidence to 100 blast waves having peak over pressures of 160 dB SPL. The impulses were presented at a...
rate of 2/min. The blast waves were generated using a conventional shock tube (compressed air-driven source) with an expansion section terminating in an exponential horn [6]. The 4 ft. x 4 ft. horn exit was mounted in the wall of an anechoic enclosure to reduce reflections. Pressure-time histories of the typical waves that are generated at different operating pressures are shown in Fig. 1.

All animals were killed by decapitation immediately after exposure or at various postexposure times up to 30 days. The cochleas were perfused through the round window with a cold, 5%, glutaraldehyde in veronal acetate buffer at pH 7.3 (630 Mosm). Following overnight fixation at 4 degrees C, the cochleas were postfixed for 5 min. with a 5% glutaraldehyde/2% aqueous osmium mixture in a 5:2 ratio. Following dehydration and dissection of the bony capsule, the specimens of the organ of Corti were either mounted in glycerin on glass slides as surface preparations, or were critical point dried with liquid carbon dioxide and sputtered with gold or gold-palladium using a cold sputtering head. Cochleas prepared for SEM were viewed with a JEOL JS-35 Scanning Electron Microscope (SEM) operating at 10-20 KeV. More complete details concerning histological preparation procedures can be found in Hamernik et al. [7,8].

RESULTS

Figures 2A and B illustrate the gross appearance of the organ of Corti from two different animals immediately following exposure. The low magnification surface preparation view in Fig. 2A illustrates the extensive tearing of the organ of Corti from its basilar membrane attachments for