**Introduction**

Glucocorticoids are routinely used in otology to treat sudden hearing loss (1, 2). Common applications include systemic and/or intratympanic injection of dexamethasone for the treatment of idiopathic sudden sensorineural or autoimmune hearing loss. Despite their widespread use, the benefit of glucocorticoid treatment for hearing disorders remains controversial. This is in part due to the observed hearing preservation and the cellular mechanisms and clinical outcomes of glucocorticoid use for the treatment of hearing loss.

The potentially beneficial effects of glucocorticoids are mediated through several pathways. The anti-inflammatory effects of these steroids are due, in part, to blocking the transcription of pro-inflammatory molecules through inhibition of NF-κ, AP-1, and AP-2 (3). Recruitment of transcription factors that genes encoding pro-inflammatory mediators to the promoter of the inflammatory genes has also been shown to down regulate inducible nitric oxide synthase (iNOS) which results in a decrease in inflammation and damage to neurons (4). Another mechanism of action of glucocorticoids is through inhibition of cell death pathways, especially the mitogen activated protein kinase (MAPK)/JNK/ERK signal pathway (5). Glucocorticoids have been localized to the inner ear to: the organ of Corti; auditory nerve; stria vascularis; spiral ligament; and the vestibular sensory epithelia (6). The location and properties of glucocorticoid receptors (GRs) on the cell surface of the inner hair cells, it can be inferred that glucocorticoids can be beneficial in the treatment of inner ear disease.

It has been shown that immediately following electrode insertion, damage occurs to the basilar membrane of the cochlea in a similar fashion to that of a naturally occurring toxin (i.e. 4-hydroxy-2,3-nonenal; HNE). In a recent clinical study of cochlear implantation, epithelial cell destruction of the organ of Corti has been shown to occur within 12 hours of electrode insertion (7). Given the beneficial properties and localization of glucocorticoid receptors (GCRs) within the cochlea, it can be inferred that glucocorticoids can be beneficial in the treatment of inner ear disease.

**Materials and Methods**

**Experimental Animals:** Approved by the ACUC of the University of Miami Miller School of Medicine. Forty-one guinea pigs were randomly assigned to three experimental groups: 1) electrode insertion trauma only (EIT, n=14), 2) EIT plus artificial perilymph (EIT + AP, n=15), and 3) EIT plus dexamethasone base in AP (EIT + DXM, n=12) was used. Ears were randomly selected and contralateral unoperated cochleae served as internal controls. Auditory brainstem responses (ABR) testing was performed prior to surgery, immediately after surgery, and on post-EIT day 3, 7, 14 and 30. Five hundred Hz and 4, 8 kHz. There was no statistical difference (p > 0.05) between the hearing thresholds of unoperated (control) and contralateral operated (experimental) ears, and f = 0.006.

**Results:**

There was a statistical difference (p < 0.05) between the hearing thresholds of unoperated (control) and contralateral operated (experimental) ears, and f = 0.006. There was no statistical difference (p > 0.05) in hearing thresholds in the EIT + DXM vs. control groups at 500 Hz, 4, and 8 kHz.

**Conclusions:**

- The electrode insertion trauma, untreated (EIT) causes both an immediate loss followed by a progressive loss of hearing thresholds that becomes a permanent hearing loss.
- Perfusion of the scala tympani with artificial perilymph (AP) only (EIT + AP) in 8 days following electrode insertion trauma (EIT) does not prevent either the immediate or progressive losses of hearing threshold caused by this trauma. However, there is a plateau in the amount of hearing loss caused when the scala tympani is perfused with AP.
- Perfusion of the scala tympani with a high dose of dexamethasone base (DXM-100 μg/ml) in AP (EIT + DXM) for 8 days beginning immediately after EIT conserves hearing function by preventing the progressive elevation of ABR thresholds that develops post-EIT and a recovery of hearing thresholds to near pre-surgical values.
- The scala tympani with a high dose of dexamethasone base (DXM-100 μg/ml) in AP (EIT + DXM) for 8 days beginning immediately after EIT conserves hearing function by preventing the progressive elevation of ABR thresholds that develops post-EIT and a recovery of hearing thresholds to near pre-surgical values.

**Bibliography**

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