

PROTECTION OF ISCHEMIA-REPERFUSION INDUCED CELL DEATH BY NON-  
STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDs) IN THE RAT RETINA

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The degeneration of retinal neurons result in loss of vision. It can result from such disorders as central artery occlusion, glaucoma and diabetic retinopathy.

It was known that NSAID can protect from ischemic damage by inhibition of COX activation and other pathway such as oxidative stress, formation of reactive oxygen species.

The present study was performed to investigate the effect of aspirin, sulfasalazine and sulindac in ischemic retina. Retinal ischemia was induced by high intraocular (at 160 mmHg) for 60 minutes. Drugs were administered before ischemia night. Protective effect were observed by light microscopy after 24 hrs ischemia. Retinal ischemia induced the degeneration of neurons in ganglion cell layer and inner nuclear layer.

All drugs, aspirin, sulfasalazine and sulindac reduced retinal cell death induced by ischemic reperfusion. Sulindac showed protective effect in low concentration than others. Aspirin and sulfasalazine had the protective effect in between 1 and 5 mM concentration. Sulindac had neuroprotective effect in between 50 and 500  $\mu$ M concentration.

These results showed that non-steroidal anti-inflammatory drugs of aspirin series can prevent retinal degeneration by ischemia-reperfusion injury.

Key Words: Ischemia, Retina, Aspirin, Sulfasalazine, Sulindac

DEVELOPMENT OF SPEECH PROCESSOR FOR ELECTRICAL STIMULATION ON  
THE SPIRAL GANGLION NEURON OF THE COCHLEA

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Introduction: The hair cells in the cochlea are responsible for translating mechanical information into neural information. If the hair cells are damaged, the hearing system has no way of transferring acoustic signals to neural spikes. Previous studies reports that deaf individuals lacking hair cells, spiral ganglion neurons are severely degenerated, and that in turn leads to hearing impairment. The purpose of this study is to develop the speech processing system for electrical stimulation through the surviving link.

Method: A microphone that picks up the sound, the sound is first pre-amplified using an automatic gain controller and passed through a series of 4th-order bandpass filters. The filtered waveforms are then extracted by RMS-to-DC converter. The signals are finally compressed and then used to modulate PWM pulses. A non-linear compression function is used to ensure that the extracted signals in the hearing range of electrically evoked response.

Results: We generated waveforms of time-varying  $\log$  produced by a speech processing system. The sound amplitudes reflect the RMS of the compressed signals for each channel. The advantage of this RMS continuous averaged pulse type of stimulation is that the signals are delivered in a non-overlapping fashion, thereby minimizing channel interaction. The transmission link system transmits the signals through a 2.5 MHz radio frequency.

Conclusion: The developed system is expected to use a non-overlapping pulse pattern for electrical stimulation on the spiral ganglion neuron in the cochlea.

Key Words: Cochlea, Hair cell, spiral ganglion neuron, Speech processor, Hearing