CMSC 828D Report - Vestibulo-Ocular Reflex

Philip Dasler

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1 Introduction

The vestibular-ocular reflex (VOR) is a reflexive movement of the eyes in order to keep images stable on the retina. This is accomplished by moving the eyes contrary to any head or body movement and covers both rotational and translational movement.

2 Mechanism

This movement is caused by an inhibition of the muscles on one side of each eye while exciting the muscles on the other side. These inhibitory and excitation signals come mainly from the inner ear, in which several key pieces play a role in sensing motion.

For example, the inner ear has a series of semicircular canals that, each of which is oriented to sense the effects of rotational motion in one of three dimensions. The canals contain a fluid known as endolymph and many tiny hairs. When the head is rotated, the endolymph lags behind the motion due to inertia, causing the sensory hairs to bend in the direction corresponding to the movement.

Additionally, the ear contains otolithic organs, that is, organs designed to sense linear acceleration. These organs contain tiny particles suspended in endolymph. When the head moves, these particles again bend tiny hairs within the organs and the motion is sensed.

Pathways in the brain have been found that directly map these sensing organs to the extraocular muscles (i.e. the muscles that control eye movement), inhibiting and exciting them as necessary. Thus, this is a true reflex, rather than simply a conscious effort. In fact, this reflex is often used on comatose patients as a method of diagnosis. If one were to rotate the head of such a patient and the eyes remained in a fixed position, then this is an indication that the brainstem remains undamaged, as it is a critical juncture in the path from the vestibular organs to the extraocular muscles.

These direct paths control the velocities of the eyes, but there is an indirect path that exerts some control as well. There exists "neural integrators" within the medulla and the midbrain that accumulate signals for the positioning of the eyes. Without this accumulation of the information gathered from the vestibular organs, the eyes would simply roll back to center once the head movement stops and the direct signals to the motorneurons has ceased.

3 Theories

There has been some question as to whether or not the vestibular feedback is the only contributing factor. For example, perhaps there is some visual processing that plays a part in the VOR. After all, given the paper by Lettvin et al, we know that there is some processing that occurs in the eye. Wouldn't it be possible for this information to be passed to the extraocular muscles? Thus far, there is little evidence that this is the case. The most damning evidence against this theory is the simple fact that the VOR occurs even when the eyes are closed.

Perhaps, then, an efference copy of anticipated body movements is sent to the extraocular muscles. This seems a little more promising, as Herdman et al found that unpredictable head movements cause a greater decrement in visual acuity than predictable ones.

4 Neural Plasticity

The final component of the VOR is the concept of “gain”, that is, how much the eyes move in response to head movements. The gain is the ratio of the change in eye angle to the change in head angle. Ideally, the eyes would move as much as the head, yielding a gain of 1.0. However, a gain significantly far from 1.0 (due, perhaps, to weak eye muscles) results in image motion on the retina and blurred vision. Often, VOR adaptation can overcome issues in eye/head angle matching. Multiple studies have shown that the speed at which the eyes adjust to motion is a learned response and can be altered.

Specifically, Boyden et al discovered that the VOR gain in mice could be altered by moving an image along with head, decreasing the necessary gain. Additionally, it was found that the gain would eventually return to normal if the mice were left in a natural environment.

Ornitz et al and Weissman et al have shown that the VOR gain is significantly different in developing children within the first few months of life, getting increasingly better until a gain of roughly 1.0 is reached.

References

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