PERSPECTIVE

Does extraocular muscle proprioception influence oculomotor control?

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Disorders of ocular motility are encountered on a regular basis within ophthalmic practice. They include a wide variety of conditions from non-paralytic strabismus commonly seen in paediatric clinics to acquired restrictive and paralytic conditions, which may be indicative of more serious underlying pathology. For an accurate diagnosis to be made an understanding of the basic mechanisms involved in oculomotor control is desirable.

Eye movements are mediated by a complex hierarchy of neuronal systems. While the final common pathway consists of the motor nuclei and associated structures in the brainstem, oculomotor behaviour is shaped by the cerebellum, the superior colliculus, the basal ganglia, and the cortical eye fields.[1] In order to coordinate the movement of the eyes, a process vital for both vision and visually guided behaviour, these brain centres must "know" the direction in which the eyes are pointing.

If the eyes were fixed within the orbits then retinal (that is, visual) information would be sufficient to tell us where we are looking. However, the eyes, as well as the visual world, can and do move, and under these circumstances extraretinal (that is, non-visual) information is required to determine gaze direction. There are two broad hypotheses that seek to explain the source of this extraretinal information; while not mutually exclusive, they are often presented as alternatives. The "inflow" hypothesis holds that afferent signals from the effector muscles in the oculomotor system, the extraocular muscles (EOM), provide the necessary information about the positions of the eyes in the orbits and about movements of the eyes. This view can be attributed to Sherrington,[2] although it fell out of favour, particularly in the 1960s, when the role of muscle receptors in general came to be doubted (see Matthews[3] for review). The outflow hypothesis, attributed to Helmholtz,[4] holds that central monitoring of a copy of the motor command sent to the EOM (efference copy[5] or corollary discharge[6]) provides the necessary extraretinal information. While in reality both afferent and efferent copy signals are probably involved, the relative contribution of each has been the subject of considerable debate for over a century. In this review we summarise the evidence concerning the existence of proprioceptive inflow from the EOM, and review recent physiological and clinical findings that it plays some part in oculomotor control.

Evidence for the presence of inflow
INNERVATION
While the motor innervation of the EOM is well established, their sensory innervation has been the subject of considerable controversy.[7 8] Two types of sensory receptor have been identified within the EOM—namely, muscle spindles[9 10] and myotendinous cylinders (palisade endings).[11]

Muscle spindles are located in the proximal and distal thirds of human extraocular muscles.[12 13] Their structure differs from that of other species and also from spindles of other skeletal muscle.[9] They consist of thin intrafusal fibres within a connective tissue capsule and lie in parallel with the extrafusal fibres. Two types of sensory ending are normally present in muscle spindles—namely, group I afferent fibres which arise from primary (annulospiral) endings and group II fibres which arise from secondary ("flower spray") endings. While spindle sensitivity is usually modulated by the gamma motor innervation, little is know about the role of gamma innervation in the EOM. Although EOM spindles are found in infant and elderly subjects at a density similar to that for spindles in hand and neck muscles, which suggests a role in fine motor control,[10 14] their proprioceptive capacity has been questioned.[15 16] Ludvigh[15] believed that "muscle spindles give rise to little, if any, acceptable information concerning the position of the eyes" while Ruskell[16] found structural features within muscle spindles such as the presence of anomalous fibres within the connective tissue capsule, which he argued could jeopardise their proprioceptive function. However, structural considerations alone cannot settle the issue and are no replacement for empirical findings (see below). The other main sensory receptors of skeletal muscle, Golgi tendon organs, are not present in human extraocular muscles[11 17] although they have been identified in other species such as the sheep[18] and the monkey.[19]

Myotendinous cylinders (palisade endings) appear to be a class of muscle receptor unique to the EOM and are found within the distal myotendinous junction of the extraocular muscles both in humans and monkeys.[11 20] They consist of networks of fine neural filaments closely associated with the end of a single extrafusal muscle fibre and are surrounded by a thin capsule.[21] Greater numbers of myotendinous cylinders are found within the horizontal compared with the vertical recti or oblique muscles.[20] Given their intimate association with extrafusal fibres, a specialist role in monitoring EOM function has been suggested,[11] in particular a response to active contraction of their associated muscle fibres. However, as no one has yet succeeded in recording from palisade afferent fibres, the precise information they transduce is not known. Nevertheless, in view of their location at the distal myotendinous junction, the very area at which the majority of strabismus procedures are performed, it is tempting to speculate that disrupting these receptors during surgery could lead to alterations in oculomotor control and affect the outcome of surgery.

PERIPHERAL PATHWAYS AND CENTRAL CONNECTIONS
The primary afferent pathway from the EOM to central processing structures has also generated a degree of controversy. Animal studies have shown that afferent fibres

travel for a variable distance with the motor cranial nerves (III, IV, and VI) before crossing to travel in the ophthalmic branch of the trigeminal nerve.[22–24] The balance of the evidence indicates that the vast majority if not all the primary afferent cell bodies are located within the trigeminal ganglion in various species, including monkeys,[25] cats,[26] birds,[27] and rabbits.[28] Primary afferent fibres terminate in the ipsilateral spinal trigeminal nucleus in the cat[29] and in the monkey, in which there is also a secondary projection to the cuneate nucleus.[25] The effects of the stimulation of EOM afferent signals have been detected in a large number of visual and oculomotor structures including the cerebellum,[30] the vestibular nuclei,[31] the abducens nucleus,[32] and the superior colliculus.[33]

Function of extraocular muscle proprioception

The evidence not only for the presence of sensory receptors within the EOM, but also for their capability of conveying afferent signals to all of the important structures involved in visual and oculomotor control has been outlined above. The next issue we have to consider is to what extent and in what ways do EOM afferent signals alter information processing in these structures? There is increasing evidence from experiments in both animals and humans that EOM afferent signals are important in three broad areas of visuomotor control. Firstly, oculomotor control, which will be discussed in more detail below. Secondly, the development and maintenance of normal binocular visual function (for reviews see Steinbach[34] and Buisseret[35]), and, thirdly, in spatial localisation, by providing afferent information about the position of the eye within the orbit, which in turn helps to determine visual direction.[34 35] The remainder of this article will consider the potential role of proprioception in oculomotor control specifically.

Oculomotor control

ANIMAL STUDIES

There is evidence derived from animal studies that proprioceptive input influences both gaze holding and gaze shifting systems. For example, EOM deafferentation of animals by sectioning the ophthalmic branch of the trigeminal nerve affects fixation stability in cats[36] and causes deviation of the eye position in lambs.[37] Section of the III, IV, and VI cranial nerves of one eye in the cat, disrupting afferent feedback, alters the fixation stability of the contralateral eye in the dark.[38] O'Keefe and Berkley[39] demonstrated that in anaesthetised cats, retrobulbar injection of a paralytic drug reduced eye movements in both the ipsilateral treated eye *and* the contralateral untreated eye. They concluded that EOM afferent signals from extraocular muscles mediated this effect possibly by influencing the central motor command signal. Further studies have also shown that proprioception contributes to the maintenance of ocular alignment during fixation in monkeys.[40]

In addition to the stability of the eyes within the orbits, proprioception also modifies eye movements. For example, the conjugacy of saccadic eye movements in monkeys is impaired by deafferentation.[40] There is considerable evidence that EOM afferent signals modify the processing of vestibular information and in so doing alter eye movements generated by the vestibular system.[41] Removal of the proprioceptive input from extraocular muscles by sectioning the ophthalmic branch of the trigeminal nerve disrupts the slow phase and reduces the gain of the vestibulo-ocular reflex (VOR) in rabbits.[28 42] Manipulating EOM afferent signals by imposing movements on one eye modifies the output of the VOR of the contralateral eye in pigeons and indicates that such signals may be important in the moment to moment control of the VOR.[43 44] These effects have been demonstrated not only in reduced experimental preparations, but also in the alert behaving animal.[45] In addition, Kimura *et al*[42] have shown that interrupting the EOM afferent pathway in rabbits modifies the gain and velocity of optokinetic nystagmus.

The timescale over which proprioceptive feedback might act upon the oculomotor control system in animals is, again, the subject of some debate. While it has been argued that it functions over the long term to bring about adaptive parametric adjustment of eye movements,[40] much of the physiological evidence discussed above is suggestive of a more immediate effect[44] (see also Knox *et al*[46]).

HUMAN STUDIES

Two main experimental methods have been described for studying the role of EOM afferent signals in human studies: vibration of the muscle tendon and passively moving the whole eye. Vibrating a muscle tendon is a recognised way of stimulating muscle spindles in particular[47] and generates an afferent signal that is interpreted by the central nervous system as stretching of the muscle. This technique has been used specifically to induce EOM afferent signals.[48] The second method involves passively moving an eye using a scleral contact lens held in place with gentle suction. This technique probably has the advantage of modifying the proprioceptive input from all the EOM simultaneously,[49] although how closely the resultant afferent signal resembles that produced by voluntary contraction is unclear. Using these approaches, observations have been made in both normal subjects and patients that suggest a role for EOM afferent signals in the control of eye movements.

Gauthier *et al*[50 51] demonstrated that after a period of passive deviation of one eye a change in phoria is observed which corresponds to the direction of the original deviation. For example, deviating the right eye temporally resulted in an increased exophoria, as measured by the Lancaster red-green dissociating test. This effect, which persisted for several minutes after the suction contact lens was removed, was quickly eliminated by binocular viewing. The authors suggested that the change in ocular alignment was due to an interaction between EOM afferent signals and central control mechanisms. Lennerstrand *et al*[52] have shown, using single EOM vibration, that both the vertical and horizontal position of the non-stimulated eye could be modified depending on the EOM stimulated. For example, vibrating the inferior rectus muscle of one eye in normal subjects induced an upward movement of both eyes, while vibration of the lateral rectus muscle induced an abduction movement of the contralateral eye. The exact mechanism by which this occurs is unclear. However, direct interactions between afferent signals from individual EOM and the motor nuclei of synergistic and antagonist muscles are highly unlikely given the earlier discussion on the route of the afferent pathway. Interestingly, the response of exotropic subjects to vibration of the lateral rectus was opposite to that seen in normal subjects; an adduction movement was noted in the contralateral eye. This suggests an altered pattern of central processing of EOM afferent signals in these subjects.

Both saccades and smooth pursuit can also be modified by EOM proprioception. Saccadic eye movements, because of their short durations and high velocities, are usually considered to be ballistic—that is, ie not under feedback control. While visual feedback is certainly too slow for the control of individual saccades, EOM afferent signals might theoretically be involved. Using the single EOM vibration technique, the programming of memory guided saccades was shown to be influenced by altering EOM proprioception.[53] Recently, Knox *et al*[46] reported that

impeding the movement of one eye using a scleral contact lens reduced the saccade amplitudes in the contralateral eye, an effect caused by a non-visual afferent signal, most likely arising from the EOM. The adaptive response of the smooth pursuit system to changes in target velocity has also been modified using proprioceptive feedback,[54] and indeed smooth pursuit initiation is altered when the contralateral eye is impeded.[55]

It might be argued that all of these studies involve non-physiological manipulations of EOM afferent feedback, thereby inducing aberrant interactions in the oculomotor control circuitry, which in turn leads to altered or degraded oculomotor behaviour. However, allied to the anatomical and structural findings discussed above, these results clearly indicate that EOM afferent signals can influence the control of eye movements.

This viewpoint is further strengthened by observations in patients, which suggest that EOM afferent signals may be important in the aetiology of certain oculomotor disorders. For example, studies in subjects with congenital strabismus have shown alterations in the morphology of EOM proprioceptors, such as smaller size and a disorganised structure.[56] However, it is not possible to be sure whether these changes are the cause or the consequence of the strabismus and further studies are needed to confirm these findings. Mitsui[57] has argued that EOM afferent signals are involved in the pathogenesis of both exotropia and esotropia. It was found that in exotropic patients, slight passive adduction of the non-deviated eye using forceps causes the deviating eye to straighten. This observation was termed the "magician's forceps phenomenon". The underlying cause of the exodeviation was believed to be abnormal proprioceptive input from the non-deviated eye, which caused excessive contraction of the lateral rectus of the contralateral, deviating eye. When the non-deviating eye was passively adducted the resultant stretch of the lateral rectus muscle modified the afferent input to the oculomotor centres which in turn influenced the position of the contralateral eye. Analogous observations could only be made in esotropic patients using electromyography. Although the interpretation of these observations has been questioned[35] they do suggest that an imbalance in EOM afferent information may affect oculomotor control.

Interestingly, modified EOM proprioception has been proposed as a factor in the aetiology and treatment of congenital nystagmus. Optican and Zee[58] suggested that erroneous afferent feedback regarding eye velocity is important in the development of this form of nystagmus. In addition, Dell'Osso *et al*[59] have recently reported damping of congenital nystagmus following staged tenotomy of all the EOM in an animal model. They suggest that this effect is due to an alteration in proprioceptive feedback from the EOM as a result of the tenotomy procedure. While acknowledging that such a procedure risks causing anterior segment ischaemia in humans, they argue that a modified procedure, consisting of bilateral medial rectus recession combined with bilateral lateral rectus tenotomy, may provide a potential surgical therapy for this condition.

The balance of the evidence is, therefore, that EOM afferent signals are not only available to oculomotor and visual control structures, but that they influence the processing of information in these structures, thereby modifying visuomotor behaviour. However, two key pieces of experimental evidence are often quoted as counter evidence to this proposition. The first comes from Keller and Robinson,[60] who reported that in the monkey there is no monosynaptic stretch reflex in the oculomotor system. While recording from single units in the abducens nucleus, they found no alteration in firing rate when either the ipsilateral eye was moved by an external force, or when a self generated movement was impeded. However, it should be remembered that demonstrating that there is no direct ipsilateral feedback pathway onto the motor neurons is not the same as demonstrating that no feedback pathway exists. Guthrie *et al*[61] provided the second key piece of evidence, by showing that monkeys in whom EOM afferent signals had been eliminated could still make accurate saccades. However, once again demonstrating that saccades can be executed accurately in the absence of EOM afferent feedback, is not equivalent to demonstrating that EOM afferent feedback plays no part when it is available. In addition, it may be that when the afferent pathway is damaged or degraded, or indeed manipulated, there is sufficient redundancy and flexibility to ensure that performance recovers (see Knox *et al*[46] for further discussion of this point). This still leaves open the issue of the precise time course of modification. Ludvigh[62] suggested that the mode of action was consistent with a long term adaptive effect in which afferent feedback induces modifications in efferent motor commands. As already noted, however, a number of key experimental results are actually more consistent with action on a far shorter timescale. This might be evidence for a fast adaptive process unique to the oculomotor system[46] or even online control of individual oculomotor or visuomotor acts. The increasing awareness of EOM proprioception is reflected in a recently described theoretical model in which information derived from efference copy and afferent feedback are integrated, with both having a fundamental role in oculomotor control.[63]

Conclusion

Knowledge of the position of the eyes within the orbits is a prerequisite for coordinated eye movements, gaze shifts, and accurate visuomotor behaviour. Although vision itself, combined with central monitoring of outflowing neural discharge to the EOM, provides much of the required information, there is now considerable experimental and clinical evidence that inflowing proprioceptive signals from the EOM make a vital contribution. Animal and human studies have demonstrated that removing or manipulating EOM afferent input not only affects static eye position but can also modify smooth pursuit, saccades and the vestibulo-ocular reflex. A greater understanding of the role of proprioception in oculomotor control would be beneficial not only from a theoretical viewpoint but also in everyday clinical practice as strabismus surgery, a commonly performed procedure, involves manipulating areas of the extraocular muscles richly endowed with proprioceptors. Too little attention has been paid to the visual, oculomotor, and visuomotor sequelae of muscle surgery. Thus, little is known as to what effect, if any, different methods of handling these tissues might have on surgical success. Further studies are clearly required.

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