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The role and clinical implications of rectus extraocular muscle pulleys

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Abstract

Aim: **To review the clinical implications of rectus extraocular muscle (EOM) pulleys in normal subjects and those with incomitant strabismus.**
***Methods*: A literature-based review of rectus EOM pulleys in normal and abnormal ocular motility is presented.**
***Results*: Rectus EOM pulleys stabilise the EOM path and support the dynamic role of the direction of muscle pull in ocular motility. The coronal plane location of each rectus pulley in relation to the orbital wall is highly uniform and stable across normal subjects when the eyes move from primary into secondary gaze positions. Studies of incomitant strabismus associated with advancing age, alphabet patterns, high myopia or dysfunction of oblique EOMs, have provided evidence that resultant patterns of strabismus and/or abnormal ocular motility may depend on static pulley positions, pulley instability and coexisting globe translation.**
***Conclusion*: Rectus EOM pulleys are fundamental to ocular motility and should be considered in the differential diagnosis of patients with incomitant deviations.**

Key words: Incomitant strabismus, Ocular motility, Rectus extraocular muscle pulleys

Introduction

In recent years the traditional theory of extraocular muscle (EOM) action has been challenged by the identification of rectus EOM pulleys. Studies on rectus muscle pulleys have utilised high-resolution magnetic resonance imaging (MRI) to visualise the path of each rectus EOM.[1–6] MRI[1–6] and dissection[7–11] of orbital anatomy has been performed on patients of varying age with normal and abnormal ocular motility. This review presents literature since 1993 identified from Medline and PubMed using the following search terms: EOM pulleys and EOM anatomy. The paper considers the structural anatomy and mechanical function of rectus EOM pulleys and discusses the effect of abnormalities in anatomy and function on ocular motility.

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Gross structural anatomy

The location and structure of rectus EOM pulleys is very similar and comparable across humans.[2,3,12] In the primary position they are located between the globe equator in the Tenon fascia[1] and the globe–optic nerve junction.[12] Each of the rectus EOM pulleys consists of a ring or sleeve of collagen, elastin and smooth muscle encircling its EOM[2,10,11] (Fig. 1).[1] The pulleys are coupled to the orbital wall and adjacent EOMs by sling-like connective tissue bands. These bands joining adjacent pulleys are particularly dense between the superior and lateral rectus muscles, and between the medial rectus and the inferior rectus complex.[12]

Mechanical function

MRI cannot directly reveal the mechanical properties of EOM pulleys; however, imaging can give strong hints about mechanical properties by showing the types and densities of tissues.[7] The smooth muscle suspensions of the rectus EOM pulleys receive rich innervation[10] by sympathetic, parasympathetic and nitroxidergic neurons.[12] Demer *et al.*[1] advanced the notion that rectus EOM pulleys must be actively translated along the muscle axis during eye movements. In alert subjects MRI has demonstrated that rectus EOM pulleys minimise changes in movement relative to the orbit and EOM paths during globe rotation.[1–4] Therefore the pulleys determine the effective pulling direction of each EOM and they support the dynamic role of ocular motility[4] (Fig. 2)[13] in an orderly way as the eye changes position.[3]

EOMs contain two distinct compartments: an inner global layer and an outer orbital layer[8] (Fig. 1).[1] The global layer of each rectus EOM inserts into the sclera and is specialised for initiation of transient high tension in the muscle to enable globe rotation.[9] The orbital layer is specialised for sustaining tension in order to control the direction of globe rotation.[4] The orbital layer of each EOM inserts on its rectus EOM pulley to position the pulley linearly.[1] This arrangement of dual layers influences the EOM's rotational axis.[1,4,6,9] Therefore EOM pulleys have important implications for EOM action because the functional origin of an EOM is at its pulley.[1–6]

Clinical correlation

MRI scanning of the orbits in patients with abnormal

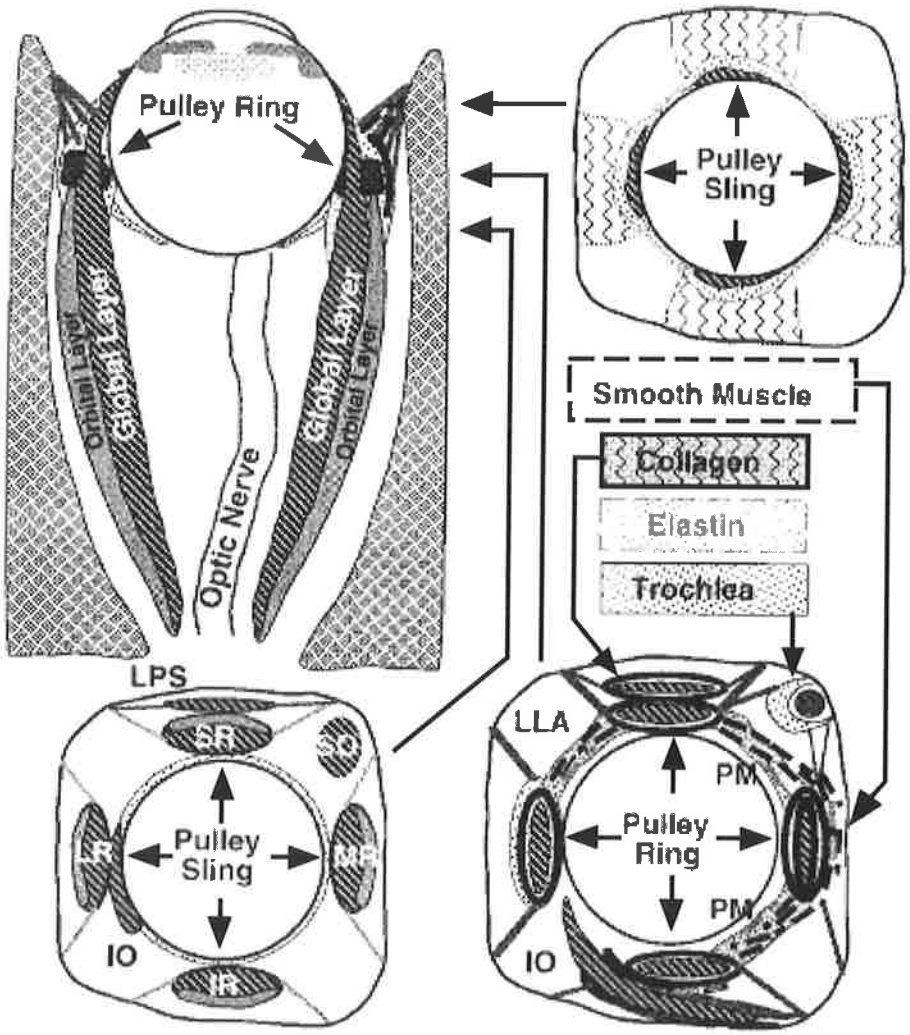


Fig. 1. Diagrammatic concept of the anatomy of the EOMs.[1] The structure of the orbital connective tissues and their relationship to the fibre layers of the rectus muscles is shown. Coronal views are represented at levels indicated by arrows in horizontal section. PM, peribulbar smooth muscle; LLA, lateral levator aponeurosis; LPS, levator palpebrae superioris; LR, lateral rectus muscle; MR, medial rectus muscle; SO, superior oblique muscle; IO, inferior oblique muscle; SR, superior rectus muscle; IR, inferior rectus muscle. (Reproduced with permission from *Investigative Ophthalmology & Visual Science.*[1])

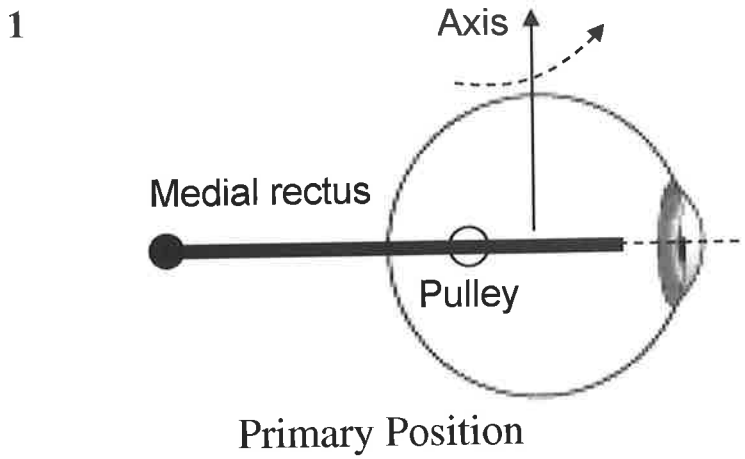
ocular motility has shown disorders in the rectus EOM pulleys in either location or/and stability. These abnormalities have been reported to cause incomitant strabismus.[3,5,14–16]

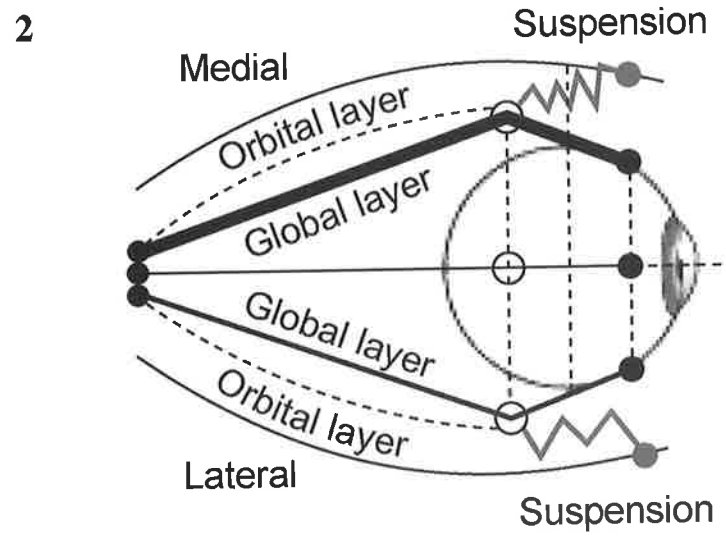
Clark *et al.*[3] documented that pulley position is highly uniform and stable across normal subjects. The rectus EOM pulleys exhibit shifts with eccentric gaze that are so small they do not produce clinically detectable misdirection of EOM forces.[3] In subjects with a variety of incomitance and/or strabismus it has been consistently demonstrated that gaze-related shifts are found in EOM paths, as well as shifts in components of the pulley itself.[1–3] This 'stable' malpositioning of pulleys, known as heterotopy, alters the position of the pulley, thus changing the course and action of the EOM.[1–4] This in turn affects the action of the fellow eye depending on the displacement.[3] If one or several pulleys are unstable, marked shifts within gaze combined with heterotopy can alter EOM action.[3–5] The following reports a variety of ocular motility defects that have been discussed in current literature associated with heterotopy or instability of one or more rectus EOM pulleys.

High myopia

von Noorden[17] discussed incomitant strabismus in high myopes and proposed that the restrictions in ocular motility are caused by contact between the elongated globes and the orbital walls and apices of the orbits. Hence progressive high myopia produces a progressive compression of the enlarged globe against the lateral wall of the orbit leading to pulley hindrance. In eyes with extreme myopia there is an association with esotropia and hypotropia in the primary position.[13,17] Such patients exhibit a large inferior path of the lateral rectus muscle; the normal abducting action of the lateral rectus is thus converted to depression.[5] Due to the lateral rectus muscle's inferior displacement, its pulley will also be in an abnormal location. Therefore an abnormal anterior pulley location or failure of a pulley to move posteriorly during EOM contraction could result in a pulley collision with the scleral location, hindering ductions, and cause incomitancy in high myopes.

Oh *et al.*[5] studied pulley stability in 4 cases of incomitant strabismus using MRI. For 2 subjects with





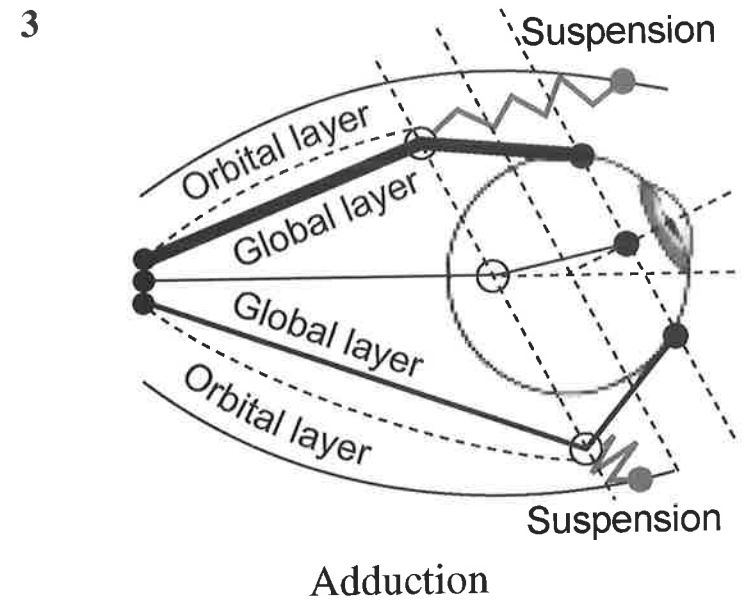


Fig. 2. Relationship of pulleys to the rotational axis of horizontal rectus muscles[13]. (1) The medial rectus muscle's rotational axis is perpendicular to the segment between the pulley and the scleral insertion, and is thus vertical in central gaze. (2) Axial view showing pulleys (depicted as rings) of the horizontal rectus muscles in central gaze. (3) In adduction, the contracting medial rectus orbital layer shifts its pulley posteriorly, while the relaxing lateral rectus orbital layer allows its pulley to move anteriorly. (Adapted from *Paediatric Ophthalmology and Strabismus.*[13])

axial high myopia, MRI revealed an inferior shift of the lateral rectus pulley of up to 1 mm during vertical gaze shifts. Both subjects had a V-pattern esotropia and an upshoot of the right eye in adduction. This finding did not explain the upshoot but the V pattern could be related to the pulley displacement.

V pattern

The remaining 2 subjects in Oh *et al.*'s study had V patterns with differing aetiologies.[5] MRI for 1 subject with simulated Brown syndrome showed both lateral rectus EOMs were displaced inferiorly by approximately 4 mm near the orbital apex and the left lateral rectus pulley had marked inferior heterotopy of 3.8 mm. An inferior shift of the lateral rectus in elevation would increase its elastic tension in that gaze position to produce greater abduction and thus the typical V pattern. In addition the left globe translated inferiorly in abduction and depression by 3 mm and by 1.5 mm in adduction. The right globe translated by 0.2 mm superiorly in depression and by 0.1 mm inferiorly in abduction and adduction. The large inferior shift of the left lateral rectus pulley in adduction in this subject would be expected to redirect the passive elastic force of the lateral rectus inferiorly, even with reduced active innervation of the lateral rectus. This would account for restrictive hypotropia and limited elevation in adduction and also the observed ipsilateral hypotropia in elevation.[5]

The final subject was a 10-year-old child with Marfan's syndrome who had a V-pattern exotropia. MRI revealed heterotopy of the right medial rectus pulley, which was located posteriorly by 2 mm and superiorly by 2 mm from the globe centre, giving rise to instability of both medial rectus muscles inferiorly in elevation.[5] This heterotopy corresponded to the version abnormality detected in ocular motility testing.

Ageing process

It has recently been proposed that some deviations are due to abnormal connective tissue laxity.[13] Most tissues in the human body undergo slow involution with ageing.[15] Histological examination shows attenuation of connective tissues around the pulleys in the elderly, particularly in the orbital layer of EOMs.[6] In the orbit, such changes in connective tissue might complicate control of eye movements by displacing or destabilising the precise placement of the EOM pulleys.[13] This absence of adequate structural support for pulleys possibly leads to gaze-related pulley shifts or globe translation.[3] This suggests that pulley abnormalities may reflect an exaggeration of the connective tissue changes observed in normal ageing, and that some age-related strabismus may be due to connective tissue degeneration.[5,6]. Marfan's syndrome is a disorder of connective tissue, and the version abnormalities in the child reported by Oh *et al.*[5] could be a reflection of premature ageing.

Kono *et al.*[6] analysed the structure of the EOM pulley system of 5 human orbits of various ages by MRI. The study concluded that although general features of pulleys were preserved from fetal life up to a 93-year-old specimen, there was qualitative evidence of age-related degeneration within the system. Elastin fibres were clumped and shredded in the rectus EOM pulleys within the older population.[6] These degenerative connective tissue changes in ageing correlated with symmetrical limited ocular ductions in the elderly.[7]

MRI has provided evidence of downward displacement of horizontal rectus pulley positions by 1–2 mm from young adulthood to the seventh decade.[15] This inferior displacement of the horizontal rectus EOM pulleys converts some of the force to depression whilst vertical rectus muscle pulleys mostly show stability.[15] The horizontal rectus pulleys of normal older people sag inferiorly and symmetrically,[13] which may probably be a cause of reduced elevation. Asymmetrical occurrence of such changes in the two orbits may contribute to the observed impairment of elevation in older people and predispose them to a characteristic pattern of incomitant strabismus. Therefore acquired heterotopy may result from ageing.

Globe translation

Oh *et al.*[5] described 4 patients with incomitant strabismus who had significantly greater horizontal and vertical globe translation with gaze compared with normal subjects.[13] Globe translation relative to the bony orbit can be estimated by measuring the shift during eccentric gaze of the area central to the bony orbit at the globe–optic nerve junction. As the bony orbit does not shift during gaze changes, movement of the orbital area actually reflects translation of the globe centre.[18] Globe rotation can therefore vary the relationship of the EOM and/or EOM pulleys by altering the alignment of the muscle insertions, and hence the vector forces would be changed in both magnitude and direction.[17] According to this proposition, marked instability of EOM pulleys and globe translation can be an associated factor within facial dysmorphism. Any malformation of the bony orbit could be a cause of strabismus. Patients with craniosynostosis syndromes (i.e. Apert's and Crouzon's) typically have small, laterally rotated orbits and abnormally located EOMs.[19] These features could be the cause of the commonly reported marked V-pattern exotropia[19] or incomitant strabismus in patients with facial dysmorphism.

Recti pulley displacement masquerading as oblique dysfunction

It has been commonly reported in the literature that cases of heterotopic rectus EOM pulleys are of aetiological significance in alphabet pattern strabismus.[1,3–5,11] An alphabet pattern strabismus may be related to the complex interplay of ocular muscles, orbital tissues and facial abnormalities.[17] For example a V-pattern strabismus could result from a lateral rectus muscle pulley inferiorly displaced or a MR muscle pulley superiorly displaced in the orbit. Hence A- or V-pattern strabismus and simulations are consistent with the idea that abnormal rectus pulling directions caused by pulley displacement can in turn be clinically diagnosed as an oblique over- or underaction.[13,14,16]

Clark *et al.*[3] reported on a patient who was misdiagnosed with a left superior oblique palsy on the

clinical basis of a left hypertropia, left superior oblique underaction and excyclotropia of 5 °. The cause of the apparent oblique muscle dysfunction resulted from bilateral inferior displacement of the lateral recti and bilateral inferior and lateral displacement of the superior recti as shown by MRI.

Clark *et al.*[14] reported the results of MRI of the orbits in 8 patients in upgaze, downgaze and the primary position. All patients had a clinical diagnosis of orthotropia in the primary position. Four patients had marked superior oblique overaction and mild to marked inferior oblique underaction. Each of these patients had one to eight heterotopic pulleys (considering both orbits) and each patient had superior mislocation of at least one lateral rectus pulley by 1.8–4.9 mm. Three other patients were also discussed who were diagnosed with mild to moderate inferior oblique overaction and mild to moderate superior oblique underaction in only one orbit. These 3 patients had up to three heterotopic EOM pulleys with at least one lateral rectus pulley inferiorly dislocated by 1.9–4.9 mm. The final patient reported was diagnosed with mild inferior oblique underaction and normal superior oblique function bilaterally. This patient had bilateral superior mislocation of the medial rectus pulleys by greater than 2 mm. In all cases above[14] the findings of the measured heterotopic pulley positions reproduced the clinical patterns of incomitant strabismus without abnormalities of oblique muscle innervation or contractility. This heterotopy alters the vertical positions of the pulley, thus changing the course and action of the EOM. Therefore even isolated mislocations of less than 2 mm from the normal stable EOM pulley position,[3] coupled with smaller mislocations of the other pulleys, can produce the clinical appearance of bilateral oblique dysfunction.

Binocular alignment

Demer *et al.*[10] identified substantial bands of smooth muscle and elastin extending from the region between the inferior rectus–inferior oblique crossing to the medial rectus pulley. The medial rectus–inferior rectus band was not only the thickest intercoupling compared with other rectus-connecting pulley bands, but it also contained the most collagen, elastin and smooth muscle. These features of the medial rectus–inferior rectus band provide a stiff elastic coupling between the highly stable medial rectus pulley and relatively mobile inferior rectus pulley.[6] It has been proposed that if the tissue cells within the pulley were loosely coupled or irregularly arrayed, the overall effect of EOM contraction might simply increase stiffness of the suspension of the medial rectus, inferior rectus and inferior rectus pulleys, and hence increase the stability of the pulleys themselves.[7] It has recently been demonstrated by MRI that in convergence[1] the four rectus EOM pulleys extort around the orbital axis, with the inferior rectus pulley moving nasally.[6] If contraction resulted in reduction of the distance between the medial rectus and inferior rectus pulleys, there could be complex effects on binocular alignment.[7,10]

Kono *et al.*[6] suggested three possible roles for the pulleys' smooth muscle: to maintain uniform stiffness in the pulley suspensions; to accomplish the slow, adaptive adjustments in pulley locations that are necessary to maintain binocular alignment over a lifetime; and to fulfil a possible dynamic role in eye movements.

In general, abnormally located EOMs are displaced more during gaze changes in their plane of action (as agonists or antagonists) and displaced less during gaze changes out of their plane of action.[3] Displacement of pulleys by only a few millimetres may increase the risk of strabismus to a degree that may exceed compensatory capabilities. It is possible that small malpositionings of pulleys, or perhaps abnormalities in connective tissue of the pulleys themselves,[20] could create heterophorias to decompensate and produce strabismus in patients who are subjected to another stress on motor fusion, or to the effects of ageing.

Strabismus surgery

Rectus EOM pulleys serve as the effective mechanical origins of the EOMs, exerting a profound influence on EOM behaviour.[21,22] Theoretically, strabismus surgery that alters the relationship between EOM insertions and pulleys could compromise the relationship between eye orientation and the rotational axes of EOMs. Surgery could adversely affect neural control of eye movement,[22] if not in primary position, at least in dynamic binocular misalignments in secondary and tertiary gaze positions.[1]

Oh *et al.*[5] reported that surgical interference with the normal anteroposterior travel of rectus pulleys produces a restriction to ocular rotation in the field of action of the involved EOM. For instance an aggressive recession and resection on rectus EOMs can damage the coexisting pulley and create restrictions of free ductions. However, EOM pulleys could also be beneficially manipulated in transposition surgery[20,23] in managing alphabet strabismus. Further research is required to provide evidence of any implications of strabismus surgery on rectus EOM pulleys. It would be vital to include a range of patients, from the young to the elderly, who have undergone strabismus surgery, as children typically have denser connective tissue within the region of the EOM pulleys and older subjects may be more susceptible to globe destabilisation as a result of strabismus surgery.[18]

Summary

The EOMs pass through fibromuscular pulleys that stabilise the muscle path and support the dynamic role of the direction of the muscle pull in ocular motility.[1–4] Pulleys have important implications for EOM action because the functional origin of an EOM is at its pulley[1,11] and it is essential to bear in mind that its path from pulley to scleral insertion determines the pulling direction of an EOM.[5]

Modern techniques of imaging the orbit have supplied valuable information about these rectus EOM pulley structures. The literature presented provides evidence that resultant patterns of strabismus and/or abnormal ocular motility may depend on static pulley positions, pulley instability and coexisting globe translation.[5] The concept of rectus EOM pulleys has important clinical implications for binocular alignment, differential diagnosis and any surgical intervention.

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