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1) Circulating T cells contact blood vessels either when they extravasate across the walls of microvessels into inflamed tissues or when they enter into the walls of larger vessels in inflammatory diseases such as atherosclerosis. The blood vessel wall is largely composed of three cell types: endothelial cells lining the entire vascular tree, pericytes supporting the endothelium of microvessels and smooth muscle cells forming the bulk of large vessel walls. Each of these cell types interacts with and alters the behavior of infiltrating T cells in different ways, making these cells active participants in the processes of immune-mediated inflammation. Immune-mediated inflammation of peripheral tissues depends upon local recruitment of circulating leukocytes into an extravascular site. In most instances, leukocytes are recruited across the wall of post-capillary venules, which are composed of a continuous, one cell thick inner lining of endothelial cells (ECs) supported by an incomplete outer layer of pericytes (PCs) located within the basement membrane to whichcauses the ECs are attached.

Larger vessels are not directly involved in leukocyte trafficking into tissues, but may themselves be a target of inflammation, for example when arteries become involved by cell-mediated immune responses as occurs in atherosclerosis. In the arterial wall, the EC lining of the vessel is completely covered by vascular smooth muscle cells (SMCs), some of which are located within the vessel intima, consisting of the EC lining and the anatomic space immediately beneath the basement membrane of the ECs. However, most SMCs are densely concentrated in a multilayered, circumferentially oriented array within the vessel media, which surrounds and is separated from the intima by the internal elastic lamina. The arterial adventitia is external to the media and separated from it by the external elastic lamina. The adventitia contains fibroblasts, nerve endings, microvessels (known as vasa vasorum) and vascular stem cells. Some mononuclear leukocytes may also be present in each of these compartments that can increase dramatically in number with inflammation. It is increasingly appreciated that resident cell populations within the environment in which an immune response develops can play a major role in shaping the form of that immune response. While much of this emphasis has been on the roles played by parenchymal cells in peripheral tissues, cells of the blood vessel wall are also positioned to affect lymphocytes and recent observations have provided a deeper understanding of how blood vascular ECs, PCs and SMCs interact with infiltrating T cells in adaptive immune responses that occur near microvessels of inflamed peripheral tissues and within the wall of inflamed macrovessels. In this review we consider how these interactions impact the nature of the immune response, with focus on observations made with human cells and tissues. We discuss the issues surrounding the cell source in these experiments, and, when possible, emphasize conclusions based on in vivo observations. We caution against generalizing about the immunological functions of vascular cells, as in “ECs do the following but SMCs do something else.” While each vascular cell type displays specific characteristics that define it as an EC, PC or SMC, each of these populations may vary significantly in both phenotype and function depending on the anatomic location; i.e. their most defining feature is simply their anatomic position within the vessel wall. Heterogeneity among vascular cells arises from several causes. The body’s natural barriers against disease-causing intruders – for example, our skin, the mucous and hairs in our nose, and the acid in our stomachs – are part of our innate immune systems. Adaptive immunity develops over a lifetime of contact with pathogens and vaccines, preparations which help our immune systems to distinguish friend from foe. Until a vaccine is available, our immune systems will need to adapt clinicunaided to COVID-19.

2) The **adductor canal** which is also known as **subsartorial or Hunter’s canal** is an aponeurotic tunnel in the middle third of the thigh, extending from the apex of the femoral triangle to the opening in the adductor magnus, the abductor hiatus. It is approximately 15cm long. The canal serves as a passageway from structures moving between the anterior thigh and posterior leg.

**Borders**

The adductor canal is bordered by muscular structures:

Anteromedial: Sartorius.

Lateral: Vastus medialis.

Posterior: Adductor longus and adductor magnus.

The adductor canal runs from the apex of the femoral triangle to the adductor hiatus – a gap between the adductor and hamstring attachments of the adductor magnus muscle.

## **Contents**

The adductor canal serves as a **passageway** for structures moving between the anterior thigh and posterior leg.

It transmits the**femoral artery**, femoral vein (posterior to the artery), nerve to the vastus medialis and the saphenous nerve – the largest cutaneous branch of the femoral nerve.

As the femoral artery and vein exit the canal, they are called the **popliteal artery** and **vein** respectively.

**Clinical Relevance -Adductor Canal Block**

In the adductor canal block, local anaesthetic is administered in the adductor canal to block the saphenous nerve in isolation, or together with the nerve to the vastus medialis.

The block can be used to provide sensory anaesthesia for procedures involving the distal thigh and femur, knee and lower leg on the medial side. The sartorius and femoral artery are used as anatomical landmarks to locate the saphenous nerve.

**Clinical Relevance -Adductor Canal Compression Syndrome**

Adductor canal compression syndrome describes entrapment of the neurovascular bundle within the adductor canal. A rare condition, it is usually caused by hypertrophy of adjacent muscles such as vastus medialis.

It is most common in young males, who may present with claudication symptoms due to femoral artery occlusion (more common) or neurological symptoms due to entrapment of the saphenous nerve.

3) Extraocular and intraocular muscles, are muscles of the eyes. The muscles of the eye are integral to its function and motion. Muscles directly associated with the eye include the extraocular muscles which control the external movement of the eye; the intraocular muscles, which are responsible for pupil accommodation and reaction to light; and the protractor and retractors of the eyelids. Deficits in the muscles or the nerves innervating these muscles can result in functional impairment of the involved structures.

**Extraocular muscles**

The extraocular muscles are located within the orbit, but are extrinsic and separate from the eyeball itself. They act to control the movements of the eyeball and the superior eyelid.

There are seven extraocular muscles – the levator palpebrae superioris, superior rectus, inferior rectus, medial rectus, lateral rectus, inferior oblique and superior oblique. Functionally, they can be divided into two groups:

* Responsible for eye movement – Recti and oblique muscles.
* Responsible for superior eyelid movement – Levator palpebrae superioris.

**Levator Palpebrae Superioris**

The levator palpebrae superioris (LPS) is the only muscle involved in raising the superior eyelid. A small portion of this muscle contains a collection of smooth muscle fibres – known as the superior tarsal muscle. In contrast to the LPS, the superior tarsal muscle is innervated by the sympathetic nervous system.

Attachments: Originates from the lesser wing of the sphenoid bone, immediately above the optic foramen. It attaches to the superior tarsal plate of the upper eyelid (a thick plate of connective tissue).

Actions: Elevates the upper eyelid.

Innervation: The levator palpebrae superioris is innervated by the oculomotor nerve (CN III). The superior tarsal muscle (located within the LPS) is innervated by the sympathetic nervous system.

**Muscles of Eye Movement**

There are six muscles involved in the control of the eyeball itself. They can be divided into two groups; the four recti muscles, and the two oblique muscles.

**Recti Muscles**

There are four recti muscles; superior rectus, inferior rectus, medial rectus and lateral rectus.

These muscles characteristically originate from the common tendinous ring. This is a ring of fibrous tissue, which surrounds the optic canal at the back of the orbit. From their origin, the muscles pass anteriorly to attach to the sclera of the eyeball.

The name recti is derived from the latin for ‘straight’ – this represents the fact that the recti muscles have a direct path from origin to attachment. This is in contrast with the oblique eye muscles, which have an angular approach to the eyeball.

* Superior Rectus

Attachments: Originates from the superior part of the common tendinous ring, and attaches to the superior and anterior aspect of the sclera.

Actions: Main movement is elevation. Also contributes to adduction and medial rotation of the eyeball.

Innervation: Oculomotor nerve (CN III).

* Inferior Rectus

Attachments: Originates from the inferior part of the common tendinous ring, and attaches to the inferior and anterior aspect of the sclera.

Actions: Main movement is depression. Also contributes to adduction and lateral rotation of the eyeball.

Innervation: Oculomotor nerve (CN III).

* Medial Rectus

Attachments: Originates from the medial part of the common tendinous ring, and attaches to the anteromedial aspect of the sclera.

Actions: Adducts the eyeball.

Innervation: Oculomotor nerve (CN III).

* Lateral Rectus

Attachments: Originates from the lateral part of the common tendinous ring, and attaches to the anterolateral aspect of the sclera.

Actions: Abducts the eyeball.

Innervation: Abducens nerve (CN VI).

**Oblique Muscles**

There are two oblique muscles – the superior and inferior obliques. Unlike the recti group of muscles, they do not originate from the common tendinous ring.

From their origin, the oblique muscles take an angular approach to the eyeball (in contrast to the straight approach of the recti muscles). They attach to the posterior surface of the sclera.

* Superior Oblique

Attachments: Originates from the body of the sphenoid bone. Its tendon passes through a trochlear, and then attaches to the sclera of the eye, posterior to the superior rectus.

Actions: Depresses, abducts and medially rotates the eyeball.

Innervation: Trochlear nerve (CN IV).

* Inferior Oblique

Attachments: Originates from the anterior aspect of the orbital floor. Attaches to the sclera of the eye, posterior to the lateral rectus

Actions: Elevates, abducts and laterally rotates the eyeball.

Innervation: Oculomotor nerve (CN III).

**Clinical Relevance**:

* Cranial Nerve Palsies

The extraocular muscles are innervated by three cranial nerves. Damage to one of the cranial nerves will cause paralysis of its respective muscles. This will alter the resting gaze of the affected eye. Thus, a lesion of each cranial nerve has its own characteristic appearance:

Oculomotor nerve (CN III)  – A lesion of the oculomotor nerve affects most of the extraocular muscles. The affected eye is displaced laterally by the lateral rectus and inferiorly by the superior oblique. The eye adopts a position known as ‘down and out’.

Trochlear nerve (CN IV) – A lesion of CN IV will paralyse the superior oblique muscle. There is no obvious affect of the resting orientation of the eyeball. However, the patient will complain of diplopia (double vision), and may develop a head tilt away from the site of the lesion.

Abducens nerve (CN VI) – A lesion of CN VI will paralyse the lateral rectus muscle. The affected eye will adducted by the resting tone of the medial rectus.

**Intraocular muscles**

The intraocular muscles include the ciliary muscle, the sphincter pupillae, and the dilator pupillae. The ciliary muscle is a smooth muscle ring that controls accommodation by altering the shape of the lens, as well as controlling the flow of aqueous humor into Schlemm's canal. The ciliary muscle is attached to the zonular fibers which suspend the lens. Upon contraction of the ciliary muscle, the tension on the lens is lessened which causes it to adopt a more spherical shape to focus on near objects. Relaxation of the ciliary muscle has the opposite effect, optimising distant focus. The sphincter pupillae and dilator pupillae are also composed of smooth muscle. The sphincter pupillae encircles the pupil and is responsible for the constriction of its diameter, while the dilator muscle is arranged radially and increases the pupillary diameter

**Innervation of intraocular muscles**

The extraocular muscles are innervated by nerves that enter the orbit through the superior orbital fissure. The oculomotor nerve (CN III) divides into superior and inferior branches and innervates the superior, medial, and inferior recti, the levator palpebrae superioris, and the inferior oblique. It also carries presynaptic parasympathetic fibers to the ciliary ganglion. Sympathetic fibers of CN III contribute to upper eyelid retraction by innervation of the superior tarsal muscle (Müller's muscle). The trochlear nerve (CN IV) innervates the superior oblique, and the lateral rectus is innervated by the abducens nerve (CN VI). The orbicularis oculi is innervated by the temporal and zygomatic branches of the facial nerve (CN VII).

**Clinical Relevance**

Strabismus occurs when the eyes are misaligned such that an object is not focused simultaneously on the fovea of each eye. A phoria is defined as the turning of an eye in (esophoria) or out (exophoria) upon occlusion of the opposite eye.  Phorias are often asymptomatic but may degenerate into tropias. Tropias are recognized as spontaneous eye turn in the absence of an ocular occlusion. Tropias are often more prevalent with tiredness as phorias become more pronounced and the ability to compensate decreases. There are certain drugs which can result in temporary tropias.

Amblyopia results when the vision in one of the eyes is reduced because the eye and the brain are not working together properly. Strabismic amblyopia is the result of an eye misalignment and is treated initially by patching the good eye to force the child to use the amblyopic eye, and may ultimately require strabismus surgery. After age five, it is difficult to reverse amblyopic vision.