

EZERIOHA UGOCHI SCHOLASTICA

200LEVEL

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ANA 210 ASSIGNMENT

Question 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 which 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.

Question 2

The adductor canal (Hunter's canal, subsartorial canal) is a narrow conical tunnel located in the thigh. It is approximately 15cm long, extending from the apex of the femoral triangle to the adductor hiatus of the adductor magnus. 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.

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.

Question 3

Extraocular muscle with their nerve supply

The extraocular muscles are the six muscles that control movement of the eye and one muscle that controls eyelid elevation (levator palpebrae). The actions of the six muscles responsible for eye movement depend on the position of the eye at the time of muscle contraction. They include: The medial, inferior, and superior recti, the inferior oblique, and levator palpebrae muscles, all innervated by the oculomotor nerve; the superior oblique muscle, innervated by the trochlear nerve; and the lateral rectus muscle innervated by the abducens nerve. Its a visual system. Its insertion is the tarsal plate of upper eyelid and eye. The movements of the extraocular muscles take place under the influence of a system of extraocular muscle pulleys, soft tissue pulleys in the orbit. The extraocular muscle pulley system is fundamental to the movement of the eye muscles, in particular also to ensure conformity to Listing's law. Certain diseases of the pulleys (heterotopy, instability, and hindrance of the pulleys) cause particular patterns of incomitant strabismus. Defective pulley functions can be improved by surgical interventions. The extraocular muscles are supplied mainly by branches of the ophthalmic artery. This is done either directly or indirectly, as in the lateral rectus muscle, via the lacrimal artery, a main branch of the ophthalmic artery.

The bodies or nuclear of these cells are found in the brain stem, the nuclear of abducens and oculomotor nerves are connected which is important in coordinations and the motion of the lateral receipts in one eye and medial action on the other. N/S ,Two antagonist muscle in one eye, contraction of one leads to the inhibition of the other.

The extraocular muscles develop along with Tenon's capsule (part of the ligaments) and the fatty tissue of the eye socket (orbit). The development of the extraocular muscles is dependent on the normal development of the eye socket, while the formation of the ligament is fully independent.

FUNCTION

Movement coordination: Intermediate directions are controlled by simultaneous actions of multiple muscles. When one shifts the gaze horizontally, one eye will move laterally (toward the side) and the other will move medially (toward the midline). This may be neurally coordinated by the central nervous system, to make the eyes move together and almost involuntarily. This is a key factor in the study of strabismus, namely, the inability of the eyes to be directed to one point.

There are two main kinds of movement: conjugate movement (the eyes move in the same direction) and disjunctive (opposite directions). The former is typical when shifting gaze right or left, the latter is convergence of the two eyes on a near object. Disjunction can be performed voluntarily, but is usually triggered by the nearness of the target object. A "see-saw" movement, namely, one eye looking up and the other down, is possible, but not voluntarily; this effect is brought on by putting a prism in front of one eye, so the relevant image is apparently displaced.

Clinics significance;

Examination; eye examination

The initial clinical examination of the extraocular eye muscles is done by examining the movement of the globe of the eye through the six cardinal eye movements. When the eye is turned out (temporally) and horizontally, the function of the lateral rectus muscle is tested. When the eye is turned in (nasally) and horizontally, the function of the medial rectus muscle is being tested. When turning the eye down and in, the inferior rectus is contracting. When turning it up and in the superior rectus is contracting. Paradoxically, turning the eye up and out uses the inferior oblique muscle, and turning it down and out uses the superior oblique. All of these six movements can be tested by drawing a large "H" in the air with a finger or other object in front of a patient's face and having them follow the tip of the finger or object with their eyes without moving their head.

INTRAOCULAR MUSCLE WITH ITS NERVE SUPPLY

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. 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. The ciliary muscle and both pupillary muscles are cranial neural crest derivatives and develop from mesenchyme of the choroid. The central retinal artery and the ciliary arteries supplies intraocular structures.

The ciliary ganglion is made up of postsynaptic parasympathetic nerve cell bodies associated with the ophthalmic nerve. The short ciliary nerves originate from the ciliary ganglion and carry parasympathetic and sympathetic fibers to the iris and ciliary body. The long ciliary nerves branch off of the nasociliary nerve and carry postsynaptic sympathetic fibers to the dilator pupillae and afferent fibers from the cornea and iris. The sphincter pupillae is parasympathetically-stimulated while the dilator pupillae is sympathetically-stimulated.

Clinical significant

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.