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REVIEW ARTICLE

CRANIAL NERVES ASSOCIATED WITH EYES IN RELATION WITH AYURVEDIC CONCEPTS.

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Abstract

There are mainly 6 Cranial nerves which contribute for the proper function of eyes, Both Sensory and motor functions. Motor activity affecting the direction of gaze, position of the eyelid and size of pupil are done by III, IV, and V cranial nerves. Optic nerve is a sensory cranial nerve. The sensory functions of eyes are carried by Optic nerve. This article mainly deals with the anatomy, functions and Pathological conditions related with this 6 cranial nerves in relation with eyes. Also there is a brief mentioning about Ayurveda correlations in respect to Cranial nerves.

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Introduction:-

There are over 7 trillion nerves in the human body. The nervous system is a very intricate grouping of nerves and cells (neurons) that sends messages throughout the body. The central nervous system is comprised of the brain and spinal cord. While all nerves are important, there are two sets of nerves that are the most important in the body: cranial and spinal. There are 12 pairs of cranial nerves that reach from the inferior side of the brain. Among the 12 pairs of Cranial nerves, the nerves related with eyes are the Optic, Oculomotor, Trochlear, Trigeminal, Abducens and Facial nerves. Among them, The Optic is related with sight. Oculomotor for Movement of eyelid and eyeball and adjusts the pupil and lens of the eye. The Trigeminal for corneal sensation and Abducens for eye ball movement. Facial nerve helps in tear secretion generally. In Ayurveda Acharyas mentioned Ayurvedic correlations for each of the Cranial nerve and their functions.

Cranial Nerves Associated With Eyes

There are 12 pairs of cranial nerves. Among them Optic Nerve, Oculomotor, Trochlear, Trigeminal, Abducens and Facial nerves are associated with eyes.

Optic Nerve

The optic nerve is the second cranial nerve and is about 47-50 mm length. It has basically four portions; Intraocular portion, Intraorbital portion, Intracanalicular portion and Intracranial portion¹.

The visual pathway starts from the retina and ends in the cortical areas. There are basically seven levels through which the visual impulses pass. They are: (i) Retina, (ii) Optic nerve, (iii) Optic chiasma, (iv) Optic tract, (v) Lateral geniculate body, (vi) Optic radiation, and (vii) Cortical areas².

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Optic Chiasma

Optic chiasma is a commissure formed by the junction of the optic nerve. This provides for crossing of the nasal retinal fibers to the optic tract of the opposite side and for passage of temporal fibers into the optic tract of the ipsilateral side. It is a flattened oblong band, some 12 mm in its transverse diameter and 8 mm from before backwards.

Types of Optic Chiasma³

Central chiasma: This is present in about 80 percent of cases. It lie directly above the sella, so that expanding pituitary tumors will involve the chiasma first; Prefixed chiasma: This is seen in about 10 percent of cases. In these cases, the chiasma is present more anteriorly over the tuberculum sellae. In such a situation, the pituitary tumor may involve the optic tracts first.; Postfixed chiasma: This is seen in about 10 percent of cases. In these cases, the chiasma is located more posteriorly over the dorsum sellae so that pituitary tumors are apt to damage the optic nerve first.

The optic chiasma lies over the diaphragma sellae and is ensheathed in pia mater surrounded by cerebrospinal fluid. As it lies over the diaphragma sellae, presence of a visual field defect in a patient with a pituitary tumor indicates suprasellar extension. Posteriorly, the chiasma is continuous with the optic tracts.

Optic Tract

Each optic tract is a cylindrical band, which runs from the optic chiasma to the crus cerebri. It runs laterally and backwards from the posterolateral angle of the chiasma between the tuber cinereum and the anterior perforated substance. Becoming more flattened and strap-like it is united to the upper part of the anterior then lateral surface of the cerebral peduncle (crus cerebri).

Lateral Geniculate Body

Lateral geniculate bodies (LGBs) are a pair of bodies, which are part of the thalamus and form an end station for all fibers subserving vision in the optic tracts. It is an oval or cap-like structure. It is situated on the posterior aspect of the thalamus.

Optic Radiations

Optic radiation or optic radiation of Gratiolet is a fresh relay of fibers that carry the visual impulses from the LGB to the occipital lobe. They pass forwards and then laterally through the area of Wernicke as the optic peduncle, anterior to the lateral ventricle and traversing the Retrolenticular part of the internal capsule behind the sensory fibers and medial to the auditory tract. The fibers spread out fanwise to form the Medullary optic lamina.

Visual Cortex

Parastriate Cortex

The visual picture from both the eyes unites in the Parastriate cortex called area 18. The lips of the lunate sulcus separate area 17 from area 19. Area 18 is buried within the walls of the sulcus and is in between area 17 and area 19.

Peristriate Cortex

This is area 19. Most of area 19 lies in the posterior parietal lobe but inferiorly it forms part of the temporal lobe. In area 19 the object seen is recognized.

The Oculomotor (Iii), Trochlear (Iv) And Abducens (Vi) Nerves⁴

The oculomotor (III), trochlear (IV) and abducens (VI) nerves innervate the extrinsic ocular muscles which move the eyeball. It is artificial to consider these nerves separately since both eyes move simultaneously to fix on a single point: eye movements are thus said to be conjugate.

Functions

These nerves innervate the extrinsic ocular muscles.

Oculomotor (III):

1. Superior division: levator palpebrae superioris (LPS), superior rectus.
2. Inferior division: medial rectus, inferior rectus, inferior oblique.
3. Trochlear (IV): superior oblique.

4. Abducens (VI): lateral rectus.

Through its parasympathetic components, the oculomotor nerve also causes constriction of the pupil (miosis) and has a role in accommodation of the lens.

Facial Nerve

The facial nerve, CN VII, is the seventh paired cranial nerve. In this article, we shall look at the anatomical course of the nerve, and the motor, sensory and parasympathetic functions of its terminal branches.

The facial nerve is associated with the derivatives of the second pharyngeal arch.

1. Motor: Innervates the muscles of facial expression, the posterior belly of the digastric, the stylohyoid and the stapedius muscles.
2. Sensory: A small area around the concha of the auricle.
3. Special Sensory: Provides special taste sensation to the anterior 2/3 of the tongue.
4. Parasympathetic: Supplies many of the glands of the head and neck, including:
 1. Submandibular and sublingual salivary glands.
 2. Nasal, palatine and pharyngeal mucous glands.
 3. Lacrimal glands

Trigeminal Nerve

The trigeminal nerve, CN V, is the fifth paired cranial nerve. It is also the largest cranial nerve. In this article, we shall look at the anatomical course of the nerve, and the motor, sensory and parasympathetic functions of its terminal branches.

Sensory:

The three terminal branches of CN V innervate the skin, mucous membranes and sinuses of the face. Their distribution pattern is similar to the dermatome supply of spinal nerves (except there is little overlap in the supply of the divisions).

Motor:

Only the mandibular branch of CN V has motor fibres. It innervates the muscles of mastication: medial pterygoid, lateral pterygoid, masseter and temporalis. The mandibular nerve also supplies other 1st pharyngeal arch derivatives: anterior belly of digastric, tensor veli palatini and tensor tympani.

Parasympathetic Supply:

The post-ganglionic neurones of parasympathetic ganglia travel with branches of the trigeminal nerve. (But note that CN V is NOT part of the cranial outflow of PNS supply)

Ayurvedic Approach To Cranial Nerves

Table 3:-Cranial Nerves and Ayurvedic Correlations⁵

Optic Nerve	Drishti Nadi
Oculomotor Nerve	Netra cheshtani
Troclear Nerve	Kadakshini
Trigeminal Nerve	Tridhara
Abducens Nerve	Netraparswiki
Facial Nerve	Vaktra Nadi

Clinical significance of cranial nerves in eye diseases

Lesions of visual pathway with main causes

1. Optic Nerve : Glaucoma, Optic neuritis and Optic atrophy
2. Optic Chiasma : Optic chiasmal Neuritis and Radionecrosis
3. OPTIC TRACT : Optic tract syndrome
4. LATERAL GENICULATE BODY: Atrophy of LGB in glaucoma and Trans-synaptic degeneration of LGB
5. OPTIC TRACT: Meningitis, Meningial TB and Tumours of thalamus

6. LATERAL GENICULATE BODY : Intra cranial space occupying lesions.
7. OPTICRADIATION: Primary and secondary intracranial tumour and Intra cranial vascular occlusions
8. VISUALCORTEX: Intra cranial space occupying lesions

Lesions Of The Visual Pathway And Field Defects⁶

Optic nerve type field defects

Retinal nerve fibers enter the optic disk in a specific manner. So, nerve fiber bundle defects are of three basic types:

Papillomacular Bundle

Macular fibers enter the temporal aspect of the disk. A defect in this bundle of nerve fibers results in one of the following:

1. Central scotoma—a defect covering central fixation
2. Centrocecal scotoma—a central scotoma connected to the blind spot (the cecum)
3. Paracentral scotoma—a defect of some of the papillomacular fibers lying next to but not involving central fixation.

Arcuate Nerve Fiber Bundle

Fibers from the retina temporal to the disk enter the superior and inferior poles of the disk. A defect in these bundles may cause any of the following:

1. Seidel scotoma—a defect in the proximal portion of the nerve fiber bundle causes a comma-shaped extension of the blind spot called a Seidel's scotoma
2. Bjerrum, arcuate or scimitar scotoma—this arcuate portion of the field at 15 degrees from fixation is known as Bjerrum's area
3. Isolated scotoma within Bjerrum's area—this is due to a defect of the intermediate portion of the arcuate nerve fiber bundle
4. Nasal step of Ronne—a defect in the distal portion of the arcuate nerve fiber bundles produces a nasal step of Ronne. Since the superior and inferior arcuate bundles do not cross the horizontal raphe of the temporal retina, a nasal step defect respects the horizontal (180 degrees) meridian.

Nasal Nerve Fiber Bundle Defects

Fibers that enter the nasal aspect of the disk course in a straight (nonarcuate) fashion. The defect in this bundle results in a wedge-shaped temporal scotoma arising from the blind spot and does not necessarily respect the temporal horizontal meridian. Remember, nerve fiber bundle defects arise from the blind spot and not from the fixation point. They do not respect the vertical meridian but respect the nasal horizontal meridian. If a person has a quadrantic field defect, then check if the field defect originates from the fixation point or from the blind spot. If it originates from the fixation point it is a retrochiasmal lesion and if it originates from the blind spot it is an optic nerve lesion. Other findings to check for an optic nerve lesion is decreased visual acuity, which generally will not occur in retrochiasmal lesions.

Optic Chiasma Lesions

The following defects can occur in optic chiasmal lesions.

Bitemporal Hemianopia

The nasal retinal fibers including the nasal half of the macula of each eye cross in the chiasma, to the contralateral optic tract. The temporal fibers remain uncrossed. Thus, a chiasmal lesion will cause a bitemporal hemianopia due to interruption of the decussating nasal fibers.

Central Bitemporal Hemianopia

Macular crossing fibers pass in the posterior part of the chiasma and are related to the supraoptic recess. Lesions here can produce a central bitemporal hemianopia.

Junctional Scotoma

A central scotoma in one eye with a superotemporal quadrantic defect in the other eye indicates a lesion at the junction of the optic nerve (RE in this case) and the chiasma. The lower nasal fibers cross in the chiasma and course anteriorly approximately 4 mm in the contralateral optic nerve. This is Wilbrand's knee. Then they turn back to join uncrossed lower temporal fibers in the optic tract. A lesion involving the Wilbrand's knee creates the junctional

scotoma. An important gem to remember this is the J Lawton Smith super gem. If a patient comes with poor vision in the right eye, the important eye for visual field examination is the left eye. There may be an upper temporal defect with respect for the vertical meridian, due to involvement of the Wilbrand's knee. The lesion is now intracranial at the junction of the right optic nerve and chiasma. The field defects constitute a junctional scotoma.

Upper Temporal Quadrantic Defects

The lower nasal fibers travel low and anteriorly in the Optic chiasma. Thus, pituitary tumors can affect them. Thus, they produce upper temporal quadrantic defects.

Lower Temporal Quadrantic Defects

The upper nasal fibers travel high and posteriorly. Thus, a lesion from above the chiasma like a craniopharyngioma can produce a lesion here. These produce a lower temporal quadrantic defect.

Optic Tract Lesions

All Retrochiasmal lesions result in a contralateral homonymous hemianopia. In the optic tracts and LGB, nerve fibers of corresponding points do not yet lie adjacent to one another. This leads to incongruous visual field defects. When we use the term congruous it means homonymous hemianopic defects that are identical in all attributes like location, size, shape, depth and slope of margins. Thus in optic tract lesions, there is an incongruous homonymous hemianopia.

Lateral Geniculate Body Lesions

A lesion in the lateral geniculate body is extremely rare. Two types of defects can occur. They are:

1. Incongruous homonymous hemianopia
2. Relatively congruous homonymous horizontal sectoranopia associated with sectorial optic atrophy. This is due to vascular infarction of the LGB.

Optic Radiations And Visual Cortex Lesions

Various lesions can occur in the optic radiations and visual cortex. Depending on the site of lesion, various field defects can occur.

Temporal Lobe Lesions

Inferior fibers course anteriorly from the LGB into the temporal lobe, forming Meyer's loop, approximately 2.5 cm from the anterior tip of the temporal lobe. They are separated from the superior retinal fibers, which course directly back in the optic radiations of the parietal lobe. Anterior temporal lobe lesions tend to produce midperipheral and peripheral contralateral homonymous superior quadrantanopia. This is called a pie in the sky field defect.

Parietal Lobe Lesions

The superior fibers cross directly through the parietal lobe to lie superiorly in the optic radiations. The inferior fibers course through the temporal lobe (Meyer's loop) and lie inferiorly in the optic radiations. Thus, there is a correction of the 90 degree rotation of the visual fibers that occurred through the chiasma into the tracts. Parietal lobe lesions tend to produce contralateral inferior homonymous quadrantanopia as they affect the superior fibers first.

Occipital Lobe Lesions

Central homonymous hemianopia In the visual cortex, the macular representation is located on the tips of the occipital lobes. A lesion affecting the tip of the occipital lobe tends to produce a central homonymous hemianopia. Macular sparing The macular area of the visual cortex is a watershed area with respect to the blood supply. Terminal branches of the posterior cerebral and middle cerebral arteries supply the macular visual cortex. Only the posterior cerebral artery supplies the visual cortex subserving midperipheral and peripheral fields. A more proximal (not terminal) vessel supplies the area. Therefore, when there is obstruction of flow through the posterior cerebral artery, ipsilateral macular visual cortex may be spared, because of blood supply provided by the terminal branches of the middle cerebral artery. This may be an explanation of macular sparing. However, when there is a generalized hypoperfusion state (e.g. intraoperative hypotension), the first area of the visual cortex to be affected is that supplied by terminal branches, the macular visual cortex, resulting in central homonymous hemianopia. To say the patient has macular sparing at least 5 degrees of the macular field must be spared in both eyes, on the side of the hemianopia. Temporal crescents When we fixate with both eyes and achieve fusion of the visual information gained by both eyes, there is superimposition of the corresponding portions of the visual fields—the central 60 degrees radius of

field in each eye. There remains in each eye, a temporal crescent of field for which there are no corresponding visual points in the other eye. This temporal crescent of field, perceived by a nasal crescent of retina, is represented in the contralateral visual cortex, in the most anterior portion of the mesial surface of the occipital lobe along the calcarine fissure. If a patient has a homonymous hemianopia with sparing of the temporal crescent, the patient has an occipital lobe lesion, since this is the only site where the temporal crescent of fibers are separated from the other nasal fibers of the contralateral eye.

Riddoch phenomenon This is a rare visual field sign. Riddoch believed that patients with severe field loss from occipital lobe involvement perceive form and movement separately. He postulated that perception of movement recovers before perception of form and that this phenomenon was of some prognostic value for recovery of field. This phenomenon is illustrated in the patient with extensive dense homonymous hemianopia as a result of an occipital lobe lesion. The patient cannot see a large stationary object in the blind field but can see a smaller object, if it is moving.

Altitudinal defect Injury to both occipital poles may result in altitudinal field defects. When the upper portions of the visual cortex or posterior radiation are damaged, the resultant field defects are altitudinal with loss of the entire lower field of vision of both eyes. If the lower portion of the lobes are damaged, death usually occurs after intracranial bleeding as a result of lacerate.

The Oculomotor (Iii), Trochlear (Iv) And Abducens (Vi) Nerves Lesiond And Vision⁷

Midbrain lesions:

Oculomotor nerve Vascular or other lesions of the midbrain can affect the Oculomotor nerve. They may also affect the Substantia nigra causing Parkinsonian symptoms (e.g. resting tremor), the red nucleus (also causing extrapyramidal symptoms), and the descending Corticospinal fibres in the cerebral peduncles leading to a contralateral upper motor neuron lesions (UMNL). Benedikt's syndrome involves the nerve as it passes through the red nucleus: Oculomotor paralysis with contralateral extrapyramidal dyskinesia. In Weber's syndrome the lesion is more ventral, also involving motor fibres in the cerebral peduncles: Oculomotor paralysis is associated with contralateral UMNLs.

Oculomotor nerve injury

The Oculomotor nerve is liable to be stretched as it crosses the Tentorial notch in cases of raised intracranial pressure. Complete section of the Oculomotor nerve would lead to Ptosis (partial paralysis of LPS), lateral squint (unopposed action of superior oblique and lateral rectus), pupillary dilatation (unopposed sympathetic activity), loss of accommodation and light reflexes. Irritation of the nerve may cause spasm of the muscles supplied by it (e.g. spasm of medial rectus leading to a medial squint).

Oculomotor nerve injury: diabetes

It is not uncommon for diabetics to suffer from an acute vasculitis of the Oculomotor nerve. This causes medial squint (somatic fibres) and Ptosis (sympathetic fibres to LPS). The Oculomotor (III), Trochlear (IV) and Abducens (VI) nerves

Aneurysms of posterior cerebral artery:

Oculomotor nerve Just after the nerve leaves the midbrain it is intimately related to the posterior cerebral artery, aneurysms of which may compress the nerve leading to symptoms as described above.

Trauma: Trochlear nerve

The Trochlear nerve is the thinnest and most fragile nerve. It is vulnerable to trauma. Section of the nerve would result in the affected eye being turned medially.

Intracranial disease:

Diagnostic usefulness of Abducens nerve The Abducens nerve, with a relatively low origin compared to its destination, has the longest intracranial course of any cranial nerve. It may be involved in fractures of the base of the skull or in intracranial disease. Section of the nerve would result in convergent squint (the eye abductor being paralyzed). See also the effects of raised intracranial pressure: Abducens nerve below. Because of this long intracranial course it is often the first cranial nerve to be affected by intracranial disease. So, if you could only test one cranial nerve as part of a neurological investigation, this would be the one! 8 Abducens and facial motor nuclei

Diseases of the brain stem affecting the Abducens nucleus may also involve fibres from the facial motor nucleus which loop around it.

Gradenigo's syndrome: Abducens nerve

Since the Abducens nerve passes over the apex of the Petrous temporal bone, it may be affected by infections of the Petrous temporal (petrositis), thus causing weakness of lateral rectus with consequent medial deviation of the ipsilateral eye (Gradenigo's syndrome: rare but interesting).

The effects of raised intracranial pressure: oculomotor nerve

When an expanding lesion above the tentorium causes raised intracranial pressure, the uncus of the temporal lobe may be 126 Vision, eye movements, hearing and balance squashed into the tentorial notch (herniation of the uncus). This compresses the midbrain which passes through the tentorial notch and the nearby oculomotor nerve. The result is papillary dilatation (unopposed sympathetic action as the parasympathetic fibres in III are affected), at first unilateral and then bilateral. By this stage, the patient will already be unconscious.

The effects of raised intracranial pressure: abducens nerve

As intracranial pressure rises, the cerebrum may be forced backwards and downwards, thus stretching the nerve with its long intracranial course. A lateral rectus palsy (medial squint) would result. Because this may cause an erroneous diagnosis to be made, it is known as a false localizing sign.

Cavernous sinus thrombosis: all three nerves

Cavernous sinus thrombosis may occur as a result of an infection of any part of the head that drains through veins to the cavernous sinus (e.g. face, ear, etc.). It affects all the nerves that pass through or in the wall of the sinus (III, IV, Va, VI). The abducens nerve is usually affected first because it passes through the sinus, causing a paralysis of lateral rectus and a resultant medial squint. Involvement of the ophthalmic nerve may cause severe pain, and the condition may result ultimately in papilloedema and visual loss. Since the advent of antibiotic therapy, this condition is much less often encountered than formerly.

Facial Nerve Lesions And Vision

1. Facial nerve palsy is associated with significant morbidity and can have different etiologies. The most common causes are Bell's palsy, Ramsay-Hunt syndrome and trauma, including surgical trauma.
2. Ophthalmologists play a pivotal role in the multidisciplinary team involved in the evaluation and rehabilitation of these patients.
3. In the acute phase, the main priority should be to ensure adequate corneal protection.
4. Treatment depends on the degree of nerve lesion and on the risk of the corneal damage based on the amount of lagophthalmos, the quality of Bell's phenomenon, the presence or absence of corneal sensitivity and the degree of lid retraction.
5. The main therapy is intensive lubrication. Other treatments include: taping the eyelid overnight, botulinum toxin injection, tarsorrhaphy, eyelid weight implants, scleral contact lenses and palpebral spring.
6. Once the cornea is protected, longer term planning for eyelid and facial rehabilitation may take place. Spontaneous complete recovery of Bell's palsy occurs in up to 70% of cases. Long-term complications include aberrant regeneration with synkinesis. FNP after acoustic neuroma surgery remains the most common indication for FN rehabilitation⁸.

Conclusion:-

Cranial nerves are the direct connections of organs to the brain. They are much complicated structures. Like all organs it plays a major role in functioning of eyes. The inherited or acquired lesions of cranial nerves causes certain changes in the functioning of eye. That starts from movement of eyes to the loss of vision.

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