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Title: Cranial Nerves Associated with Eyes In Relation With Ayurvedic Concepts.

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ABSTRACT: There are mainly 6Cranialnerveswhich contributefor the properfunction of eyes, Both Sensory and motor functions. Motor activityaffecting the direction of gaze, position of the eyelid and size of pupilare done by III, IV, and IV cranial nerves. Optic nerve is a sensorycranial nerve. The sensory functions of eyes are carried by Optic nerve.This article mainly deals with the anatomy, functions and Pathologicalconditions related with this6 cranial nerves in relation with eyes. Also,there is a brief mentioning about Ayurveda correlations in respect toCranialnerves.

I. INTRODUCTION: -

There are over 7 trillion nerves in this human body. The nervous system is a very intricate grouping of nerves andcells (neurons) that sends messages throughout the body. The central nervous system is comprised of the brain andspinal 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 12pairs of Cranial nerves, the nerves related with eyes are the Optic, Oculomotor, Trochlear, Trigeminal, Abducensand Facial nerves. Among them, The Optic is related with sight. Oculomotorfor Movement of eyelid and eyeballand adjusts the pupil and lens of the eye. The Trigeminal for corneal sensation and Abducens for eyeball movement.Facial nerve helps in tear secretion generally. In Ayurveda Acharyas mentioned Ayurvedic correlations for each oftheCranial nerve andtheirfunctions.

CranialNervesAssociated withEyes

There are 12 pairs of cranial nerves. Among them Optic Nerve, Oculomotor, Trochlear, Trigeminal, Abducens andFacialnervesare associatedwitheyes.

OpticNerve

The optic nerve is the second cranial nerve and is about 47-50 mm length. It has basically four portions;
Intraocularportion,Intraorbitalportion,Intracanalicularportionand Intracranial portion[1].
The visual pathway starts from the retina and ends in the cortical areas. There are basically seven levels throughwhich the visual impulses pass. They are: (i) Retina, (ii) Optic nerve, (iii) Optic chiasma, (iv) Optic tract, (v) Lateralgeniculatebody, (vi) Optic radiation, and(vii)Cortical areas[2].

OpticChiasma

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

TypesofOpticChiasma[3]

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

The optic chiasma lies over the

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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 indicatessuprasellarextension. Posteriorly,the chiasmaiscontinuouswiththeoptic tracts.

OpticTract

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

LateralGeniculateBody

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

OpticRadiations

Optic radiation or optic radiation of Gratiolet is a fresh relay of fibers that carry the visual impulses from the LGB tothe occipital lobe. They pass forwards and then laterally through the area of Wernicke as the optic peduncle, anteriorto the lateral ventricle and traversing the Retro lenticular part of the internal capsule behind the sensory fibers andmedialto the auditorytract. Thefiberspread outfanwise toformtheMedullaryopticlamina.

Visual Cortex

Cortex

Thevisualpicturefromboththeeyesunitesintheparastriatecortexcalledarea18.Thelipsofthelunatesulcusseparatearea17 fromarea19.Area18isburiedwithinthewallsofthesulcusand isinbetweenarea17 andarea19.

PeristriateCortex

Thisisarea19.Mostofarea19liesintheposteriorparietallobebutinferiorlyitformspartofthetemporallobe.Inarea19the objectseenisrecognized.

(i)TheOculomotor(iii), Trochlear(iv)AndAbducens(vi)Nerves[4]

Theoculomotor(III),trochlear(IV)andabducens(VI)nervesinnervatetheextrinsicocularmuscleswhichmovethe eyeball. It is artificial to consider these nerves separately since both eyes move simultaneously to fix on a singlepoint:eyemovementsare thus saidtobe conjugate.

Functions

Thesenervesinnervatetheextrinsicocular muscles.Oculomotor(III):

1. Superiordivision:levatorpalpebraesuperioris(LPS),superiorrectus.
2. Inferiordivision:medialrectus,inferiorrectus,inferioroblique.
3. Trochlear(IV):superioroblique.
4. Abducens(VI):lateralrectus.

Throughitsparasympatheticcomponents,theoculomotornervealsocausesconstrictionofthepupil(miosis)andhasa role inaccommodationofthe lens.

FacialNerve

Thefacialnerve,CNVII,istheseventhpairedcranial nerve.Inthisarticle,weshalllookattheanatomicalcourseofthenerve, andthemotor,sensoryandparasympatheticfunctionsofitsterminal branches.

Thefacialnerveisassociated withthederivativesofthesecondpharyngealarch.

1. Motor:Innervatesthemusclesoffacialexpression, theposteriorbellyofthedigastric,thestylohyoidandthestapediusmuscles.
2. Sensory:Asmallareaaround theconchaoftheauricle.
3. SpecialSensory:Providesspecialtastesensationtotheanterior2/3ofthetongue.
4. Parasympathetic:Supplies manyoftheglandsoftheheadandneck,including:

1. Submandibularandsublingualsalivaryglands.
2. Nasal,palatineandpharyngealmucousglands.
3. Lacrimalglands

TrigeminalNerve

The trigeminal nerve, CN V, is the fifth paired cranial nerve. It is also the largest cranial nerve. In this article, weshalllook attheanatomicalcourseof thenerve, andthemotor, sensory andparasympatheticfunctions of itsterminalbranches.

Sensory**:**

The three terminal branchesof CN Vinnervate theskin,mucousmembranes andsinuses of the face.Theirdistribution pattern is similar to the dermatome supply of spinal nerves (except there is little overlap in the supply ofthedivisions).

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Motor:

Only the mandibular branch of CN V has motor fibers. It innervates the muscles of mastication: medial pterygoid,lateral pterygoid, masseter and temporalis. The mandibular nerve also supplies other 1st pharyngeal arch derivatives:anteriorbellyofdigastric, tensor valipalatiniandtensor tympani.

AyurvedicApproachtoCranialNerves

ParasympatheticSupply**:**

The post-ganglionic neurons of parasympathetic ganglia travel with branches of the trigeminal nerve. (But note thatCNV isNOTpartofthe cranial outflowofPNSsupply)

Table3:-CranialNervesandAyurvedicCorrelations[5]

OpticNerve	DrishtiNadi
OculomotorNerve	Netracheshtani
TrochlearNerve	Kadakshini
TrigeminalNerve	Tridhara
AbducensNerve	Netraparswiki
FacialNerve	VaktraNadi

ClinicalsignificanceofcranialnervesineyediseasesLesionsofvisualpathwaywithmaincauses

1. OpticNerve: Glaucoma,Opticneuritisand Opticatrophy
2. OpticChiasma:OpticchiasmalNeuritisandRadio necrosis
3. OPTICTRACT:Optictractsyndrome
4. LATERALGENICULATEBODY:AtrophyofLGBinglaucomaand Trans-synapticdegenerationofLGB
5. OPTICTRACT:Meningitis, MeningealTBandTumorsofthalamus
6. LATERALGENICULATEBODY:Intracranial spaceoccupyinglesions.
7. OPTICRADIATION:Primaryandsecondaryintracranialtumorand Intracranialvascularocclusions
8. VISUALCORTEX:Intracranialspaceoccupying lesions

LesionsofTheVisualPathwayandFieldDefects[6]Optic nerve type fielddefects

Retinalnervefibersentertheopticdiskinaspecific manner.So,nervefiberbundledefectsareofthreebasictypes:

PapillomacularBundle

Macularfibersenterthetemporalaspectofthedisk.Adefectinthisbundleofnervefibersresultsinoneofthefollowing:

1. Centralscotoma—adefectcoveringcentralfixation.
2. Centrocecalscotoma—acentralscotomaconnectedtotheblindspot(thececum)
3. Paracentralscotoma—adefectofsomeofthepapillomacularfiberslyingnexttobutnotinvolvingcentralfixation.

ArcuateNerveFiberBundle

Fibers from the retina temporal to the disk enter the superior and inferior poles of the disk. A defect in these bundlesmaycause anyofthefollowing:

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

NasalNerveFiberBundleDefects

Fibers that enter the nasal aspect of the disk course in a straight (nonacute) fashion. The defect in this bundleresultsin awedge-shapedtemporalscotomaarisingfrom theblindspotanddoesnotnecessarily respectthetemporal horizontal meridian.

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Remember, nerve fiber bundle defects arise from the blind spot and not from thefixation point. They do not respect the vertical meridian but respect the nasal horizontal meridian. If a person has aquadratic field defect, then check if the field defect originates from the fixation point or from the blind spot. If itoriginates from the fixation point it is a retro chiasmal lesion and if it originates from the blind spot it is an opticnerve lesion. Other findings to check for an optic nerve lesion is decreased visual acuity, which generally will notoccurinretro chiasmal lesions.

OpticChiasmaLesions
Thefollowingdefectscanoccurinopticchiasmallesions.

BitemporalHemianopia
The nasal retinal fibers including the nasal half of the macula of each eye cross in the chiasma, to the contralateraloptic tract. The temporal fibers remain uncrossed. Thus, a chiasmal lesion will cause a bitemporal hemianopia due tointerruptionofthe decussatingnasalfibers.

CentralBitemporalHemianopia
Macular crossing fibers pass in the posterior part of the chiasma and are related to thesupraoptic recess. Lesionsherecanproduce a central bitemporalhemianopia.

UpperTemporalQuadranticDefects
The lower nasal fibers travel low and anteriorly in the Optic chiasma. Thus, pituitary tumors can affect them. Thus,theyproduce uppertemporal quadrantic defects.

LowerTemporalQuadranticDefects
The upper nasal fibers travel high and posteriorly. Thus, a lesion from above the chiasma like a craniopharyngiomacanproduce alesionhere.Theseproduce a lower temporal quadrantic defect.

OpticTractLesions
All Retro chiasmal lesions result in a contralateral homonymous hemianopia. Inthe optic tracts and LGB, nervefibers 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 hemianopia defects that are identical in all attributes likelocation, size, shape, depth and slope of margins. Thus, in optic tract lesions, there is an incongruous homonymoushemianopia.

LateralGeniculateBodyLesions
Alesioninthe lateralgeniculate bodyisextremelyrare.Twotypesofdefectscanoccur.Theyare:
1. Incongruoushomonymoushemianopia
2. Relativelycongruoushomonymoushorizontalsector anopiaassociatedwithsectorialopticatrophy.Thisisdueto vascularinfarctionoftheLGB.

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

TemporalLobeLesions
Inferior fibers course anteriorly from the LGB into the temporal lobe, forming Meyer's loop, approximately 2.5 cmfrom the anterior tip of the temporal lobe. They are separated from the superior retinal fibers, which course directlyback in the optic radiations of the parietal lobe. Anterior temporal lobe lesions tend to produce midperipheral andperipheralcontralateralhomonymoussuperiorquadrantanopia.Thisiscalledapieintheskyfield defect.

ParietalLobeLesions
The superior fibers cross directly through the parietal lobe to lie superiorly in the optic radiations. The inferior fiberscourse through the temporal lobe (Meyer's loop) and lie inferiorly in the optic radiations. Thus, there is a correctionof the 90-degree rotation of the visual fibers that occurred through the chiasma into the tracts. Parietal lobe lesionstend to producecontralateralinferiorhomonymousquadrantanopiaastheyaffectthesuperiorfibersfirst.

OccipitalLobeLesions
Central homonymous hemianopia in the visual cortex, the macular representation is located on the tips of theoccipital 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. Terminalbranches of the posterior cerebral and middle cerebral arteries supply the macular visual cortex. Only the posteriorcerebral artery supplies the visual cortex subserving midperipheral and peripheral fields. A more proximal (notterminal)

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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 themiddle cerebral artery. This may be an explanation of macular sparing. However, when there is a generalizedhypoperfusion state (e.g. intraoperative hypotension), the first area of the visual cortex to be affected is that suppliedby terminal branches, the macular visual cortex, resulting in central homonymous hemianopia. To say the patient hasmacular sparing at least 5 degrees of the macular field must be spared in both eyes, on the side of the hemianopia.Temporal crescents When wefixatewith both eyes and achievefusion of thevisual information gainedby botheyes,thereissuperimpositionofthecorresponding portionsofthevisualfields—thecentral60degreesradiusoffield in each eye. There remains in each eye, a temporal crescent of field for which there are no corresponding visualpoints in the other eye. This temporal crescent of field, perceived by a nasal crescent of retina, is represented in thecontralateral visual cortex, in the most anterior portion of the mesial surface of the occipital lobe along the calcarinefissure. If a patient has a homonymous hemianopia with sparing of the temporal crescent, the patient has an occipitallobe lesion, since this is the only site where the temporal crescent of fibers are separated from the other nasal fibersofthe contralateral eye.

Riddoch phenomenon This is a rare visual field sign. Riddoch believed that patients with severe field loss fromoccipital lobe involvement perceive from and movement separately. He postulated that perception of movementrecovers 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 anoccipital lobe lesion. The patient cannot see a large stationary object in the blind field but can see a smaller object, ifitismoving.

Altitudinal defect Injury to both occipital poles may result in altitudinal field defects. When the upper portions of thevisualcortex or posterior radiation are damaged, theresultantfielddefects are altitudinalwith loss of theentirelower field of vision of both eyes. If the lower portion of the lobes are damaged, death usually occurs afterintracranialbleedingasa resultoflacerate.

The Oculomotor (iii), Trochlear (iv) And Abducens (vi) Nerves Lesionand Vision[7]Midbrainlesions:

Oculomotor nerve Vascular or other lesions of the midbrain can affect the Oculomotor nerve. They mayalsoaffecttheSubstantianigracausing Parkinsonian symptoms(e.g.,restingtremor),therednucleus(also causingextrapyramidalsymptoms),andthedescending Corticospinalfibersinthecerebralpedunclesleadingtoacontralateral upper motor neuron lesion (UMNL). Benedikt's syndrome involves the nerve as it passes through thered nucleus: Oculomotor paralysis with contralateral extrapyramidal dyskinesia. In Weber's syndrome the lesion ismoreventral,alsoinvolvingmotorfibersinthecerebralpeduncles:Oculomotorparalysisisassociatedwithcontralateral UMNLs.

Oculomotornerveinjury
The Oculomotor nerve is liable to be stretched as it crosses the Tentorial notch in cases of raised intracranialpressure.
CompletesectionoftheOculomotor nervewould leadtoPtosis(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 spasmofthemusclessuppliedbyit(e.g., spasmofmedialrectusleadingto amedialsquint).

Oculomotornerveinjury:diabetes
Itisnotuncommonfordiabeticstosufferfromanacutevasculitisoftheoculomotornerve.Thiscausesmedialsquint(somaticfibers)andPtosis(sympatheticfiberstoLPS).TheOculomotor(III),Trochlear(IV)andAbducens
(VI)nerves

Aneurysmsofposteriorcerebralartery:
Oculomotornerve
Justafterthenerveleavesthemidbrainitisintimatelyrelatedtotheposteriorcerebralartery,aneurysmsofwhich maycompressthe
nerveleadingtosymptomsasdescribed above.

Trauma:Trochlearnerve
TheTrochlearnerveisthethinnestandmostfragilenerve.Itisvulnerabletotrauma.Sectionofthenervewouldresultinthe affectedeye beingturnedmedially.

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Intracranialdisease:
DiagnosticusefulnessofAbducensnerveTheAbducensnerve,witharelativelyloworigincomparedtoitsdestination, has the longest intracranial course of any cranial nerve. It may be involved in fractures of the base of theskull or in intracranial disease. Section of the nerve would result in convergent squint (the eye abductor beingparalyzed).Seealsotheeffectsofraisedintracranialpressure:Abducensnervebelow.Becauseofthislong intracranial course it is often the first cranial nerve to be affected by intracranial disease. So, if you could only testonecranialnerveaspartofaneurologicalinvestigation,thiswouldbetheone!8Abducensandfacialmotornuclei

Theeffectsofraised intracranialpressure:oculomotornerve.

When an expanding lesion above the tentorium causes raised intracranial pressure, the uncus of the temporal lobemay 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 resultis papillary dilatation (unopposed sympathetic action as the parasympathetic fibres in III are affected), at firstunilateralandthenbilateral. Bythisstage,thepatientwillalreadybe unconscious.

Theeffectsofraisedintracranialpressure:abducens nerve.

As intracranial pressure rises, the cerebrum may be forced backwards and downwards, thus stretching the nerve withitslongintracranial course.A lateral rectuspalsy(medial squint)would result.Becausethis maycause anerroneousdiagnosisto bemade,itisknownasafalselocalizingsign.

Cavernoussinusthrombosis:allthreenerves

Cavernous sinus thrombosis may occur as a result of an infection of any part of the head that drains through veins tothe cavernous sinus (e.g., face, ear, etc.). It affects all the nerves that passthrough or in the wall of the sinus (III,IV, V, VI). The abducens nerve is usually affected first because it passes through the sinus, causing a paralysis oflateral rectus and a resultant medial squint. Involvement of the ophthalmic nerve may cause severe pain, and thecondition may result ultimately in papilledema and visual loss. Since the advent of antibiotic therapy, this conditionis muchlessoftenencounteredthanformerly.

FacialNerveLesionsandVision

1. Facialnervepalsyisassociatedwithsignificantmorbidityandcanhavedifferentetiologies.Themostcommoncausesare Bell'spalsy,Ramsay–Huntsyndromeandtrauma, including surgical trauma.
2. Ophthalmologistsplayapivotalroleinthemultidisciplinaryteaminvolvedintheevaluationandrehabilitationofthese patients.
3. Intheacutephase,themainpriorityshould betoensureadequatecornealprotection.
4. Treatment depends on the degree of nerve lesion and on the risk of the corneal damage based on the amount oflagophthalmos, the quality of Bell's phenomenon, the presence or absence of corneal sensitivity and the degreeoflid retraction.
5. Themaintherapyisintensivelubrication.Othertreatmentsincludetapingtheeyelidovernight,botulinumtoxininjection,tarsorrhaphy,eyelidweightimplants,scleral contactlensesand palpebral spring.
6. Oncethecorneaisprotected,longertermplanningforeyelidandfacialrehabilitationmaytakeplace.Spontaneous complete recovery of Bell's palsy occurs in up to 70% of cases. Long-term complications includeaberrant regeneration with synkinesis. FNP after acoustic neuroma surgery remains the most common indicationforFNrehabilitation[8].

II. CONCLUSION: -

Cranial nerves are the direct connections of organs to the brain. They are much complicated structures. Like allorgans it plays a major role in functioning of eyes. The inherited or acquired lesions of cranial nerves causes certainchangesinthefunctioningof eye.That startsfrommovementofeyestothelossof vision.

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