

Neuroclinical Anatomy of the third Cranial Nerve

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Abstract

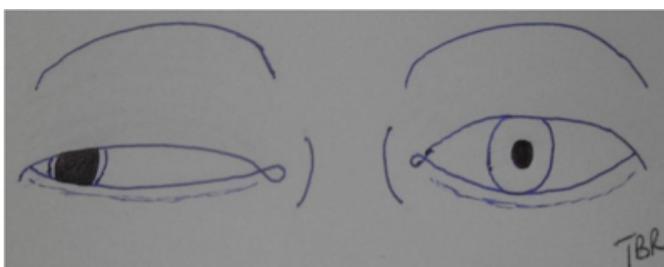
The oculomotor (third) cranial nerve arises from the upper part of the mesencephalon (midbrain). It carries both a somatic efferent (motor) supply to some of the extra-ocular muscles and a general visceral efferent (parasympathetic) supply to the sphincter pupillae and ciliaris muscles. Its contribution to the pupillary light reflex represents an important aspect of its function and is an essential component of the evaluation of neurological patients. This paper reviews the neuroclinical aspects of the nerve's anatomy and provides easily understandable guidance to an effective assessment of its dysfunction. The contributions of the third cranial nerve to pupillary reaction to light, the changes in pupillary size reflecting the various stages of its dysfunction in intracranial pathologies, and its association with neurotrauma as well as some of the eponymous syndromes associated with it are discussed.

INTRODUCTION

Oculomotor (third cranial nerve) palsy is one of the most commonly encountered cranial nerve deficits in clinical practice. Milanes-Rodriguez and his colleagues¹ have given a concise description of the origin and course of the nerve. It is the first nerve to arise from the brain stem (the first {olfactory} and second {optic} nerves arise in the nasal olfactory epithelium and ganglion cells of the neural retina respectively)². The classic finding in third nerve palsy is ptosis and downwards and outwards position of the ipsilateral eye (looking down and out).(Fig. 1)

Figure 1

Fig. 1: Right ptosis, pupillary dilatation and a 'down and out' eye in third nerve palsy



FIBERS AND NUCLEI

The dysfunction of the nerve includes that of the peripherally located fibers mediating pupillary constriction (parasympathetic supply to the sphincter pupillae muscles) as well as the motor supply to all extra-ocular muscles with the exception of the superior oblique (supplied by the fourth cranial {trochlear} nerve) and the lateral rectus (supplied by

the sixth cranial {abducens} nerve) muscles. (A useful mnemonic is LR6, SO4; that is, Lateral Rectus supplied by the 6th nerve; Superior Oblique supplied by the 4th nerve). The two nuclei of the nerve (oculomotor nucleus {somatic efferent} and the Edinger-Westphal nucleus {parasympathetic} are located in the upper part of the midbrain².

THE PUPIL IN THIRD NERVE PALSY

An examination of the pupil provides a good insight into the functional status of the oculomotor nerve. Specifically, the pupillary light reflex assesses both the optic nerve (afferent) as well as the oculomotor nerve (efferent). As such, the absence of direct light reflex in the ipsilateral eye in the presence of consensual light reflex in the contralateral eye indicates a third nerve deficit rather than an optic nerve problem. In the patient with acute traumatic brain injury, this finding is highly indicative of brainstem compression.

Incomplete or partial oculomotor nerve paresis can be pupil-sparing or non-pupil sparing. This phenomenon is a reflection of the topographic arrangements of the nerve fibers. The fibers supplying the sphincter pupillae muscles are located peripherally and are therefore affected early in extrinsic compression of the nerve³ by, for example, posterior communicating artery aneurysm, tumours and brainstem herniation in intracranial mass lesions. In contrast, intrinsic lesions of the nerve, affecting the inner fibers which supply the extra-ocular muscles, spares the pupillary fibers (and therefore the pupillary light reflex is preserved) until

very late in the course of the disease. This commonly results from micro-ischaemia as caused by, for example, diabetes mellitus, atherosclerosis (as in chronic hypertension), temporal arteritis and myasthenia gravis⁴. Rarely, pupil-sparing oculomotor palsy has been described following an intra-axial lesion such as midbrain infarction⁵.

Pupillary changes in uncal herniation in neurotrauma, brain tumours or other intracranial supratentorial space occupying lesions may be staged⁶ as follows:

Hutchinson’s stage I: ipsilateral miosis due to oculomotor nerve irritation

Hutchinson’s stage II: oculomotor nerve paresis causing ipsilateral pupillary dilatation and a sluggish response to light

Hutchinson’s stage III: Progressive dilatation of the ipsilateral pupil and miosis of the contralateral pupil (heralds progressive third nerve palsy due to temporal lobe herniation)

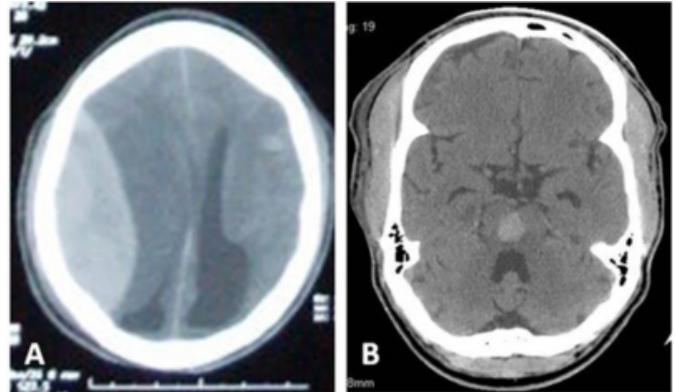
HUTCHINSON’S STAGE IV: BILATERAL DILATION OF THE PUPIL (TERMINAL STAGE) THE THIRD NERVE IN HEAD INJURY

Head injuries are a major cause of morbidity and mortality worldwide. It is estimated that a head injury occurs every 15 seconds and a patient dies of head injury every 12 minutes⁷. Its incidence has been reported as 109 per 100,000 population⁸. While road traffic accidents are the leading cause of head injury worldwide⁷, the contribution of war and conflicts are nonetheless very significant. In the Iraqi war, an estimated 1700 military personnel were thought to have sustained this injury to varying degrees⁹.

Cranial nerve injuries are common in head injuries. The incidence of oculomotor nerve damage in head injury has been reported as 2.7% in one study¹⁰. The nerve may be damaged directly by, for example, skull fractures or brainstem haematoma involving its nuclei (Fig. 2) or indirectly by intracranial mass lesion, such as an haematoma (Fig. 2) which cause uncal herniations and pressure on it. Depending on the affected part of the nerve, the injury might be complete or partial. Lesions in the orbit tend to affect the branches of the nerve in unequal proportion. Lesions affecting the superior division of the nerve cause ptosis and impaired elevation of the eye while those of the inferior division cause impairment of depression, adduction and pupillary reaction⁴.

Figure 2

Fig. 2: Examples of causes of oculomotor nerve damage as found on cranial computerized tomography: huge right parietal acute extradural haematoma (A) and midbrain haematoma .



EPONYMOUS SYNDROMES

Lesions affecting the midbrain (mesencephalon) may present with disorders of the oculomotor nerve functions in combination with other deficits¹¹, in particular, motor deficits may be concomitant findings as a result of the involvement of the descending corticospinal tracts by such lesions. Though mostly described for vascular lesions such as cerebrovascular disease (CVD), the syndromes may also be found in traumatic injury of the midbrain. (Table 1).

Figure 3

Table1: Eponymous syndromes associated with third nerve palsy

| Syndrome | Ipsilateral | Contralateral | Lesion site |
|----------------|-----------------------------|---------------------------|--|
| Weber’s | Oculomotor palsy | Hemiparesis | Cranial nerve III, Cerebral peduncle |
| Benedikt | Oculomotor palsy | Tremors + Hemianaesthesia | Cranial nerve III, Red nucleus ± Medial longitudinal fasciculus, Spinothalamic tract |
| Claude’s | Oculomotor palsy | Tremor and ataxia | Cranial nerve III, Red nucleus, Brachium conjunctivum |
| Nothnagel’s | Oculomotor palsy and ataxia | Oculomotor palsy ± ataxia | Cranial nerve III, Brachium conjunctivum |
| Plus-minus lid | Ptosis | Lid retraction | CN III fascicles to the levator palpebrae muscle; Nucleus of posterior commissure |

CONCLUSION

The third cranial nerve provides an essential and invaluable guide in the accurate assessment of the neurological patient. An understanding of the various clinical presentations of its disorder is essential to accurately localize the cause of the deficit as well as the location of the offending lesion.

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