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Cranial Nerve Palsies What's New?

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Keywords

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Key points

- Palsies of the third, fourth, and sixth cranial nerves are those most commonly encountered by the eye care provider.
- The causes of these palsies include trauma, vasculopathic, autoimmune, compression, inflammation, and infection.
- The workup of these patients may include laboratory and radiographic investigations, and the new MRI sequences used to image the cranial nerves are highlighted here.

INTRODUCTION

The ophthalmologist and optometrist must understand the clinical significance and management of cranial nerve palsies. A wide spectrum of etiologies and workups are required, and with the advent of new neuroimaging techniques, the availability and importance of these studies should be known. We endeavor

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to provide a review of the anatomy, pathophysiology, and laboratory and radiographic investigations needed to sufficiently evaluate cranial nerve palsies.

SIGNIFICANCE

Anatomy

The extraocular muscles are innervated by the third, fourth, and sixth cranial nerves. The third nerve innervates the medial, inferior, superior recti, and the inferior oblique muscles. The fourth nerve innervates the superior oblique, and the sixth nerve innervates the lateral rectus muscle. In addition, the third nerve also innervates the levator palpebrae superioris and pupillary sphincter muscle. The primary actions of the extraocular muscles are abduction (lateral rectus), adduction (medial rectus), elevation (superior rectus), depression (inferior rectus), incyclotorsion (superior oblique), and excyclotorsion (inferior oblique).

The third nerve nuclear complex is found in the midbrain, ventral to the cerebral aqueduct [1], notably with 1 subnuclei for the levator palepbrae superioris, named the central caudal nucleus, and paired superior rectus subnuclei that innervate the contralateral superior rectus [2,3]. The fascicle from the superior rectus subnuclei passes through the contralateral subnucleus, before joining the third nerve fascicle, which would cause bilateral superior rectus palsies in the event of a unilateral lesion, and may be worse ipsilaterally. The preganglionic fibers from the sixth nerve nucleus ascend from the pons to the third nerve nucleus in the brainstem, via the medial longitudinal fasciculus, which allows for conjugate horizontal movement of the eyes [4].

From the midbrain, the third nerve fascicles traverse the red nuclei, the cerebral peduncles, and exit the brainstem into the interpeduncular fossa. Then, the third nerve fascicles enter the subarachnoid space and the cavernous sinuses, where they travel in the lateral walls, until separating into the superior and inferior divisions in the anterior cavernous sinus [5]. Once in the orbit, the third nerve passes through the annulus of Zinn and on to the medial, inferior, and superior recti, and the inferior oblique. The preganglionic parasympathetic fibers to the pupillary sphincter and ciliary body run externally along the third nerve, making them more prone to infiltration or compression. The preganglionic parasympathetic fibers follow the inferior division of the third nerve from the cavernous sinus into the orbit, eventually synapsing in the ciliary ganglion before innervating the pupillary sphincter (miosis) and ciliary body (accommodation).

The fourth nerve has the longest intracranial course of all cranial nerves, originating in the dorsal midbrain. The fourth nerves decussate immediately after exiting the brainstem, in the anterior medullary vellum, so that each superior oblique muscle is innervated by the contralateral fourth nerve nucleus. These nerves travel under the tentorium and enter the cavernous sinus along the lateral wall, below the third nerve. The fourth nerve enters the orbit through the superior orbital fissure and runs medially to innervate the superior oblique muscle.

The sixth nerve nuclei are located in the dorsal pons, and the genu of the seventh (facial) nerve is found immediately proximal to the sixth nerve nucleus.

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