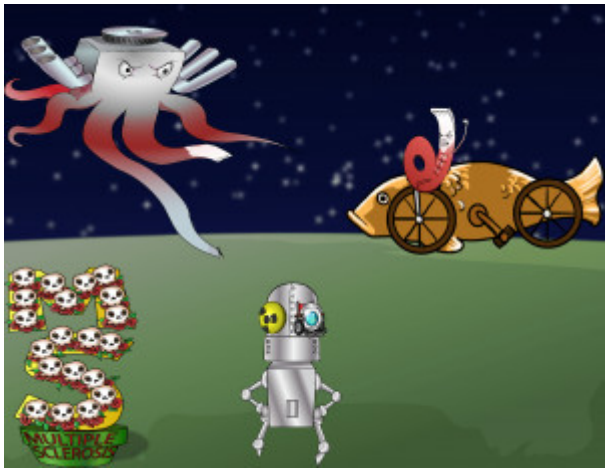
# Internuclear Ophthalmoplegia



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## ETIOLOGY

**Multiple Sclerosis**
Multiple Skull-roses
Multiple sclerosis is an autoimmune demyelinating disorder of the central nervous system (brain and spinal cord). A wide range of clinical manifestations may be seen, including optic neuritis, internuclear ophthalmoplegia, urinary and fecal incontinence, motor abnormalities such as trembling and paresis, sensory changes ranging from pain to numbness and depression, all of which typically, though not exclusively, display a relapsing pattern.

**Medial Longitudinal Fasciculus**
Medially directed fish-cycle
Medial longitudinal fasciculus is a bundle of axons that is situated near the midline of the brainstem and consists of both ascending and descending fibers that arise from a number of sources and terminate in different areas. This is a bundle of highly myelinated fibers that play a crucial role in communication between cranial nerve III and cranial nerve VI, this communication is particularly important for lateral gaze coordination. Let's discuss an example; under normal circumstances when a patient looks to the right, the right eye abducts (moves laterally from the midline) while the left eye adducts (moves medially from the midline); in patients with internuclear ophthalmoplegia, the left eye doesn't adduct when the right eye abducts, and the right eye abduction is accompanied with nystagmus.

## Mechanism

**CN VI Nucleus**
Mr. 6-pack
CN VI nerve innervates lateral rectus muscle and is responsible for the abduction of the eye (moving eye laterally from the center). In patients with internuclear ophthalmoplegia abduction of the eye is accompanied by a brief nystagmus; this happens due to the overfiring of CN VI nucleus, due to the impaired communication between this nucleus and the nucleus of CN III.

**CN III Nucleus**
Octopus-motor
CN III stimulates medial rectus muscle to adduct the eye (move the eye medially from the center), this is impaired in patients with internuclear ophthalmoplegia. Lesions in medial longitudinal fasciculus result in impaired adduction on the ipsilateral side, as medial longitudinal fasciculus communicates with the ipsilateral CN III nucleus and contralateral CN VI nucleus.

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