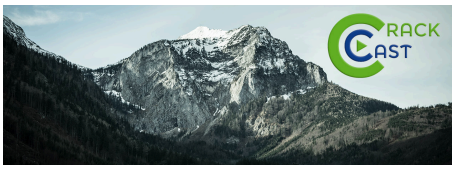


Chapter 21 – Diplopia

Episode overview:

1) List the differential diagnosis (critical emergent, urgent) for Diplopia
    - Including at least 7 causes of binocular diplopia
2) Describe the mechanisms of normal extraocular movements
3) Describe the specific cranial nerve palsies causing diplopia

Wisecracks:

1) What are the 5 most important questions to ask yourself about diplopia?
2) Describe your approach to diplopia in the sick patient

Rosen's in Perspective:

Diplopia is uncommon presentation to the ED, representing 1.4% of eye emergencies; however, it can be extremely debilitating for patients when it does occur. Diplopia can be:

- Monocular (15%) – persists in one eye even if the other eye is closed
- Binocular (85%) – resolves when either eye is closed

Monocular Diplopia = distortion with the light path through the eye (typically an eye issue)
Binocular Diplopia = pathology can exist in multiple locations, including:

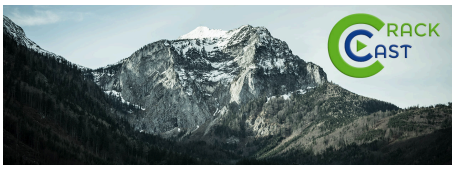
- Ocular
- Ocular muscle
- Cranial nerves (CN VI palsy a **very common cause** of diplopia)
- Upper versus lower neuron disease
- Ocular centre dysfunction in CNS
- Brainstem

1) List the differential diagnosis (critical emergent, urgent) for Diplopia

- **Including at least 7 causes of binocular diplopia**

Table 21-1

Critical:	Basilar Artery Thrombosis
	Botulism
	Basilar Meningitis
	Aneurysm



Crack Cast Show Notes – Diplopia
May 2016 – **www.crackcast.org**

Emergent: Vertebral Dissection
Myasthenia Gravis
Wernicke's Encephalopathy
Orbital Apex Syndrome / Cavernous Sinus Process

Urgent: Brainstem Tumour
Miller-Fisher Syndrome
Multiple Sclerosis
Graves Disease
Ophthalmoplegic Migraine
Ischemic Neuropathy
Orbital myositis / pseudotumor
Orbital apex mass

Table 21-1 Important Causes of Diplopia

DIPLOPIA-CAUSING ENTITY	MECHANISM AND MORTALITY	DISTINGUISHING FEATURES
Tier 1—Critical		
Basilar artery thrombosis	Acute thrombosis of the basilar artery with brainstem ischemia; untreated, mortality 70-90%	Vertigo, dysarthria, other cranial nerve involvement; risk factors for stroke
Botulism	Toxin inhibits of release of acetylcholine (ACh) at cholinergic synapses and presynaptic myoneural junctions; untreated, mortality 60%	Dysarthria, dysphagia, autonomic dysreflexia, pupillary dysfunction
Basilar meningitis	Infection; untreated, mortality close to 100% if bacterial (25-40% if treated)	Headache, meningismus, fever
Aneurysm	Enlarging aneurysm directly compresses cranial nerve; untreated, rupture risk is 1% per year; mortality 26-50% per rupture	CN III palsy with pupillary involvement
Tier 2—Emergent		
Vertebral dissection	Dissection causes vertebrobasilar ischemia; acute untreated, mortality 28% (2-5% if neurologically asymptomatic)	Neck pain, vertigo; risk factors for vertebral dissection
Myasthenia gravis	Autoantibodies develop against ACh nicotinic postsynaptic receptors; untreated, crisis mortality 42% (5% if treated)	Fluctuating muscle weakness, ptosis, and diplopia worsen with activity and improve with rest
Wernicke's encephalopathy	Thiamine-dependent metabolic failure and tissue injury; untreated, mortality 20%	Nystagmus, ataxia, altered mental status, and ophthalmoplegia; risk factors and nutritional deficiency
Orbital apex syndrome, cavernous sinus process	Inflammation or infection in the orbital apex or cavernous sinus directly affects oculomotor cranial nerves; acute mortality low unless infectious and complicated by meningitis	A combination of palsies of CN III, IV, or VI, with retro-orbital pain, conjunctival injection, and possible periorbital or facial numbness
Tier 3—Urgent		
Brainstem tumor	Tumor involvement at the supranuclear level; acute mortality low (long-term mortality variable)	Skew deviation vertical diplopia, internuclear ophthalmoplegia
Miller-Fisher syndrome	Autoantibodies develop to a cranial nerve ganglioside, GQ1b; acute mortality low if fully differentiated from GBS; mortality 2-12% if GBS	Ophthalmoplegia, ataxia, areflexia
Multiple sclerosis	Demyelinating lesions; acute mortality low	Internuclear ophthalmoplegia
Thyroid myopathy (Graves' disease)	Autoimmune myopathy; acute mortality low with regard to ocular complaints	Proptosis, restriction of elevation and abduction of the eye, signs of Graves' disease
Ophthalmoplegic migraine	Inflammatory cranial neuropathy; low mortality—self-limited disease	Ipsilateral headache, CN (usually III) palsy
Ischemic neuropathy	Microvascular ischemia; mortality low—self-limited disease	Isolated CN palsy (pupil-sparing if CN III)
Orbital myositis, pseudotumor	Autoimmune or idiopathic myositis; acute mortality low with regard to ocular complaints	Eye pain, restriction of movement, periorbital edema; exophthalmos and chemosis when more severe
Orbital apex mass	A tumor, infiltration, or mass effect in the orbital apex or cavernous sinus directly compresses oculomotor cranial nerves; acute mortality low	A combination of palsies of CN III, IV, or VI, and possible periorbital or facial numbness, with retro-orbital pain, proptosis, signs of venous congestion

CN, cranial nerve; *GBS*, Guillain-Barre syndrome.

2) Describe the mechanisms of normal extraocular movements

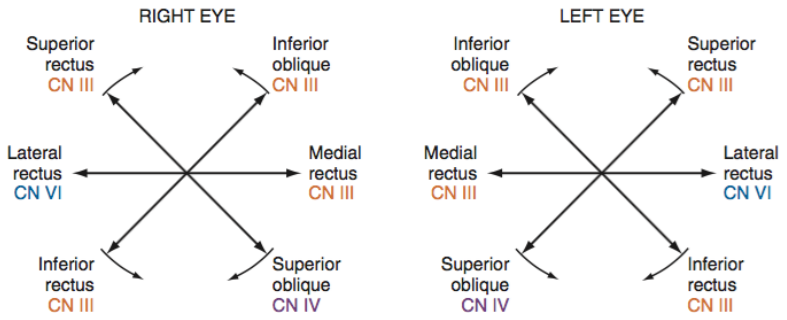


Figure 21-1. Cardinal movements of the eyes, with the oculomotor muscles that create them, and the nerves that supply those muscles. Small curved arrows denote intorsion or extorsion of the eye by the muscle indicated. *CN,* cranial nerve.

- **Remember: "SO4 - LR6 (and the rest are 3)"**

3) Describe specific cranial nerve palsies causing diplopia

NERVE PALSY	MUSCLE(S) "OFF"	SYMPTOMS	EXAM FINDINGS
Normal	N/A	N/A	
Oculomotor (CN III)	Medial, inferior, and superior rectii muscles • Inferior oblique muscle • Levator palpebrae (eye lid) • Ciliary and constrictor pupillae muscles (pupil)	Multidirectional horizontal and vertical diplopia, except on lateral gaze to the affected side • eyelid "droop"	
Trochlear (CN IV)	Superior oblique muscle	Rotational diplopia that worsens on looking down and toward the nose	
Abducens (CN VI)	Lateral recturs muscle	Horizontal diplopia on gaze toward the affected side	

Figure 21-4. Corresponding muscle dysfunction, symptoms, and examination findings for each oculomotor cranial nerve palsy. *CN,* cranial nerve.

Wisecracks

1) What are the 5 most important questions to ask yourself about diplopia?

1. Is the diplopia monocular?
2. Is the binocular diplopia a result of a restrictive, mechanical orbitopathy?
3. Is the binocular diplopia a result of a palsy of the oculomotor CNs (III, IV, or VI) in a single eye?
4. Is the binocular diplopia a result of a neuroaxial process involving the brainstem and related CNs?
5. Is the binocular diplopia a result of a neuromuscular disorder?

2) Describe your approach to diplopia in the sick patient

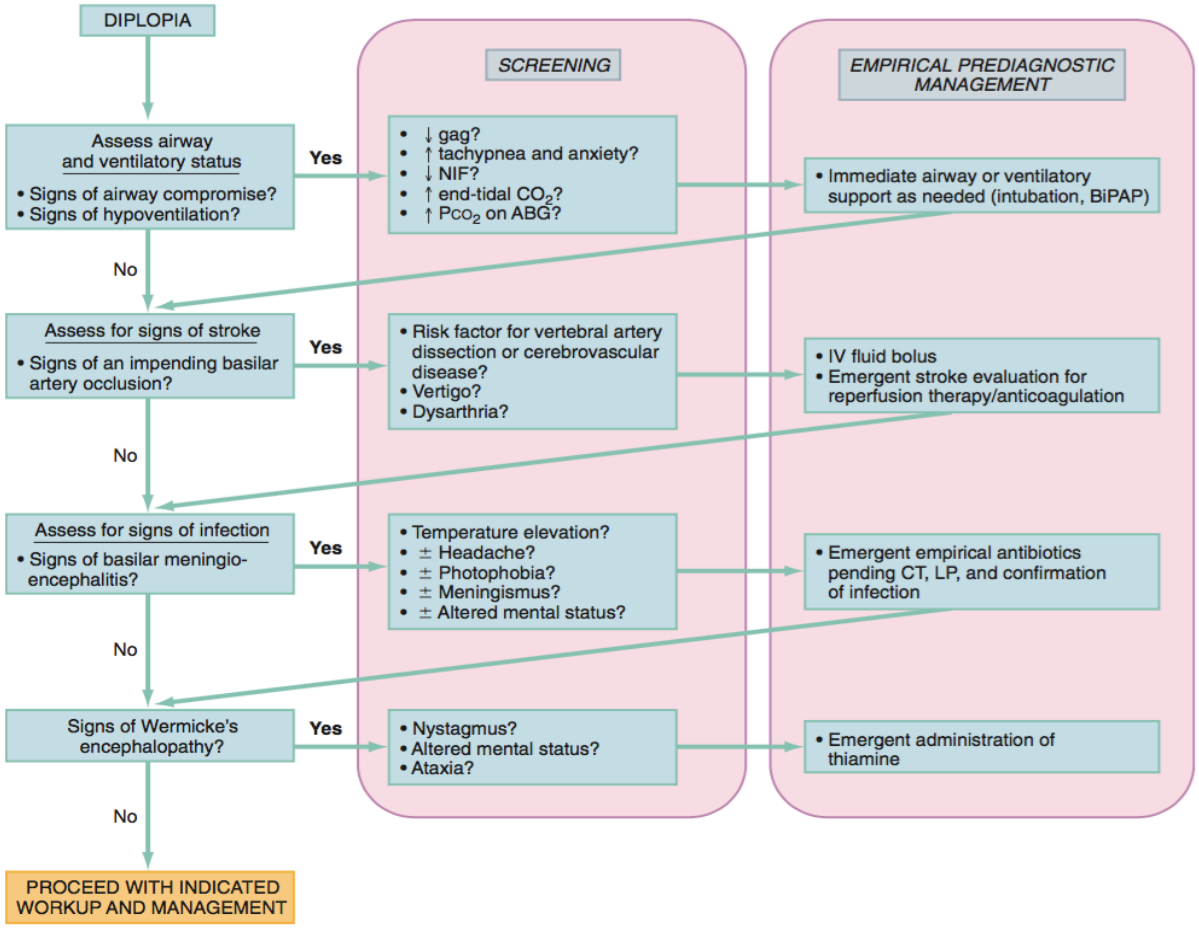


Figure 21-5. Algorithm for the initial stabilization of the patient with diplopia in the emergency department—a guideline. *ABG,* arterial blood gas; *BiPAP,* biphasic positive airway pressure; CO_2, carbon dioxide; *CT,* computed tomography (of the cranium); *IV,* intravenous; *LP,* lumbar puncture; *NIF,* negative inspiratory force; P_{CO_2}, partial pressure of carbon dioxide.