BRITISH JOURNAL OF HOSPITAL MEDICINE

What you need to know about assessing a patient with diplopia

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Abstract

Diplopia occurs when two images are subjectively seen of a single object. It has a variety of causes, which range in severity from benign to sight or life threatening if left untreated. Clinical assessment of diplopia includes taking a comprehensive history to delineate the exact type of visual disturbance experienced by the patient and examination to pinpoint the aetiology, which is crucial for further management. This article discusses the causes of diplopia, along with risk factors and other associated symptoms, to facilitate diagnosis and management. Efficient and effective diagnosis is important, as some pathologies require immediate treatment to save the eyesight and/or life of the patient. This article will concentrate solely on binocular diplopia.

Key words: Cranial nerve palsy; Diplopia; Myopathy; Orbitopathy; Strabismus

Submitted: 23 January 2022; accepted following double-blind peer review: 14 October 2022

Introduction

Diplopia is the term used to describe when a patient sees two images of the same object. Patients experiencing this may present to the local emergency department, GP or urgent care centre. An American study identified that there were over 800 000 ambulatory and 49 000 accident and emergency visits for diplopia over a 10-year period in the USA (De Lott et al, 2017).

There are many differential diagnoses for diplopia, so a systematic approach to history taking, identifying important clinical signs the patient may display, and undertaking relevant investigations is important to rule in or out any underlying pathologies.

This article provides an approach to assessing a patient with diplopia in the emergency department or primary care setting. The learning objectives are:

- Be able to take a comprehensive history of diplopia symptoms, with presenting features and associated symptoms
- Be able to perform a baseline assessment of eye movements and determine which extraocular muscles are involved
- Understand the physiology of eye movement and the anatomical considerations that determine the pathophysiology of the presenting signs and symptoms.

How to assess diplopia: history

The first clarification any clinician should make when a patient presents saying 'I have double vision' is whether it is truly double vision. Ask direct questions such as 'Are you seeing two of this pen?' or 'Are you seeing a shadow of the pen to the side of it?'. Next, it is important to clarify whether the patient is experiencing monocular (only experienced with one eye open) or binocular (only experienced with both eyes open) diplopia. Figure 1 shows a simple way of identifying whether the patient's symptoms are caused by monocular or binocular diplopia. Monocular diplopia is confirmed by asking the patient to cover one of their eyes, and if the diplopia persists, then it is monocular. In contrast, binocular diplopia is when double vision occurs with both eyes open, but disappears when either eye is covered (Modi and Arsiwalla, 2021). This is because, in binocular diplopia, there is disparity of the stimulation of the retina between the two eyes. The characteristics of binocular diplopia can be further delineated by asking the patient if the images are side by side (horizontal diplopia) or on top of each other (vertical diplopia).

How to cite this article:
Chishty F, Attzs M. What you need to know about assessing a patient with diplopia. Br J Hosp Med. 2023. https://doi.org/10.12968/hmed.2022.0055

Table 1. Important elements of history taking to assess diplopia	
Onset of symptoms	Acute onset
	Gradual or intermittent onset
Associated symptoms	Ataxia
	Difficulty in swallowing
	Dizziness
	Dysarthria
	Headaches
	Jaw claudication
	Periocular pain
	Scalp tenderness
	Vertigo
	Weakness or fatigue
	Weight loss
Ocular history	Amblyopia or lazy eye
	Childhood strabismus
	Ocular surgery
	Spectacle wearer
Medical history	Autoimmune disease
	Cancer
	Diabetes
	Thyroid eye disease
	Vasculopathy
Trauma	Blow-out fracture
	Facial trauma
	Head trauma

Adapted from Low et al (2015)

Monocular diplopia and binocular diplopia have different underlying pathologies, with the former being less common (Tan and Faridah, 2010). Discussion of monocular diplopia is beyond the scope of this article, which will now focus on binocular diplopia.

After delineating the type of diplopia the patient is experiencing, the clinician can focus on the history of the presentation. Table 1 highlights key elements of the history, and discussion of associated symptoms, that are important in determining differential diagnoses.

How to assess diplopia: examination

Aside from the initial assessment of whether the patient is clinically stable, observing any abnormal head position, eyelid position or proptosis is important in the initial examination of a patient experiencing diplopia. Visual acuity, colour vision, pupils and visual fields to confrontation should all be included in the initial eye assessment.

Eye position can give a useful clue as to the cause of diplopia, and the Hirschberg test uses a pen torch to assess this using the corneal reflex (Figure 2). To assess ocular motility, patients should be asked to follow a target in all positions of gaze, both binocularly (to assess versions) and monocularly (to assess ductions). Figure 3 shows the muscles that are assessed in different positions of gaze. The extraocular muscles move the eyes in the vertical plane (up and down), horizontal plane (left and right) and about torsional plane (incyclotorsion and excyclotorsion). Vertical eye movements are primarily controlled by

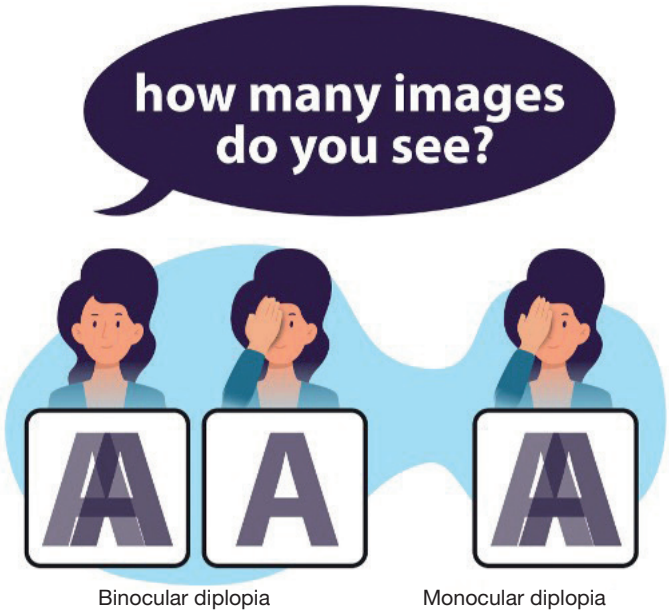

Figure 1. Identifying whether the patient's symptoms are caused by monocular or binocular double vision.

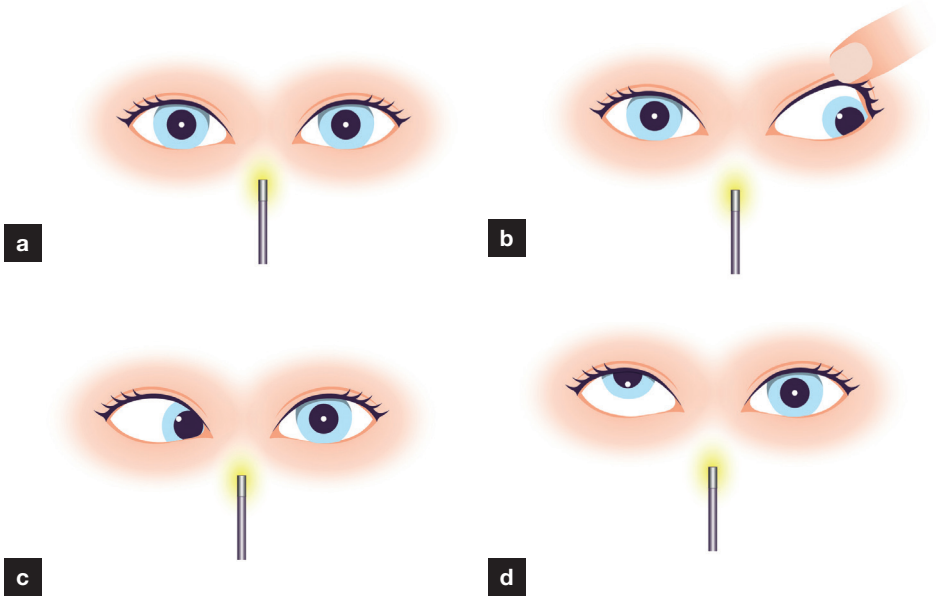

Figure 2. Hirschberg test findings for different pathologies. a. Orthophoria. b. Left eye exotropia and hypotropia secondary to cranial nerve III palsy. c. Right eye esotropia secondary to cranial nerve VI palsy. d. Right eye hypertropia secondary to cranial nerve IV palsy.

the superior and inferior rectus, horizontal eye movements are primarily controlled by the medial and lateral rectus muscles, and cyclotorsion of the eyes is primarily controlled by the superior and inferior oblique muscles. As well as primary actions, the vertical recti and oblique muscles also have secondary and tertiary actions related to eye movement.

The physiological basis of ocular motility depends on varying actions of the agonist and antagonist muscles, determined by two physiological laws: Sherrington's law and Herring's law. Herring's law of equal innervation means that in order for the eyes to move in the same direction, there must be reciprocal innervation to synergist muscles that are yoked, ie neurogenically connected. For example, for both eyes to look simultaneously to the left, there must be equal innervation to the left eye lateral rectus and the right eye medial rectus to allow smooth movement. Alongside this reciprocal innervation, inhibitory

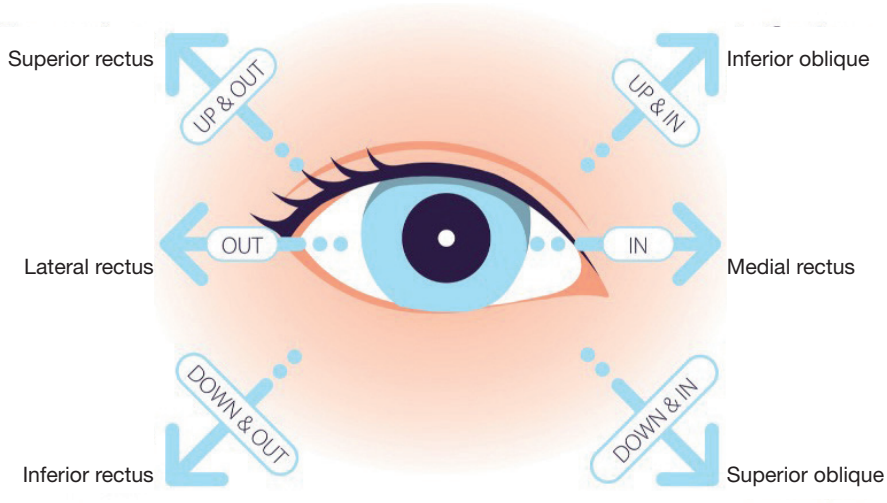

Figure 3. Cardinal positions of the eye and the muscle that determine them.

impulses are also sent to the antagonist muscle of the same eye, known as Sherrington's law. In the above example, the antagonists would be the medial rectus of the left eye and the lateral rectus of the right eye.

Causes of binocular diplopia

Strabismus

Strabismus is ocular misalignment and is the most common cause of binocular diplopia (Gräf and Lorenz, 2012). Strabismus can be manifest or latent, comitant or incomitant, paretic or restrictive. Strabismus can be subtle and only reveal itself on disruption of binocular fusion (latent), or it can be a manifest deviation causing the eyes to be misaligned in the primary position, resulting in diplopia. Manifest deviation of the eyes in the primary position can be inwards (esodeviation) or outwards (exodeviation), up (hyperdeviation) or down (hypodeviation) from the primary position. Incomitant deviation means that the size of ocular deviation differs with the position of gaze. Paretic strabismus is normally incomitant (except in congenital or chronic superior oblique palsy), and when assessing ductions and versions, the paresis of the muscle in question is less obvious with duction eye movements compared with version eye movements. Restrictive strabismus has ductions and versions that are equal in magnitude, and is also often incomitant. Comitant strabismus is where the size of ocular deviation is the same in all positions of gaze, which suggests childhood strabismus or chronic acquired strabismus.

Strabismus has ocular and non-ocular causes, as described below.

Ocular causes of strabismus

Thyroid orbitopathy

Thyroid orbitopathy, or thyroid eye disease, can occur following hyperthyroidism, euthyroid or hypothyroidism. Inflammatory processes cause enlargement and fibrosis of the extraocular muscles, resulting in restricted range of movement. The inferior rectus is the most commonly affected muscle, causing restricted supraduction and vertical diplopia, but any of the extraocular muscles can be affected (Margolin and Lam, 2018).

Orbital tumours

Orbital tumours can cause diplopia via two mechanisms: a tumour can either infiltrate the cranial nerves innervating the extraocular muscles, or compress the extraocular muscles themselves (Darsaut et al, 2001). Patients presenting with orbital tumours often have proptosis. Displacement of the globe in the horizontal or vertical planes (known as dystopia) can give diagnostic clues as to where in the orbit the lesion is located (Darsaut et al, 2001).

Ocular myopathy and myasthenia gravis

Ocular myopathies, such as myotonic dystrophy and chronic progressive external ophthalmoplegia, and myasthenia gravis, can have varying degrees of strabismus on presentation or during the clinical course of the disease. Myasthenia gravis may present acutely with other systemic features such as dyspnoea and dysphagia. Diplopia in patients with myasthenia gravis often presents with variable and fatigable ptosis, while the pupils remain normal.

Non-ocular causes of strabismus

Cranial nerve palsies

The nerves that innervate the extraocular muscles are cranial nerves III (oculomotor), IV (trochlear) and VI (abducens), which transverse the superior orbital fissure of the sphenoid bone from the cavernous sinus, to reach the orbital structures. Paresis of these nerves can result in diplopia. The patient's age and associated signs and symptoms are relevant to determining what investigations are needed. It is important to differentiate between central and peripheral cranial nerve palsies, particularly to identify the more life-threatening conditions, which accounted for 16% of diplopia presentations to an emergency department (De Lott et al, 2017). Figure 4 and Table 2 illustrate the brainstem locations of cranial nerves III, IV and VI, and the neuroanatomical and clinical correlations related to the presenting features of central causes of cranial nerve palsies. Table 3 outlines C-VITAMIN, an aide memoire to the causes of cranial nerve palsies.

Cranial nerve III palsy

In a cranial nerve III palsy, the eye has a down and out position with ptosis and pupil involvement related to its innervation (Figure 2). This presentation can vary, and can include a partial ptosis and lack of pupil involvement (Modi and Arsiwalla, 2021). The diplopia caused by a cranial nerve III palsy has both vertical and horizontal elements, which may only be elicited when lifting the ptotic lid, and relieving obstruction of the pupil.

Cranial nerve III can be affected by several microvascular and macrovascular conditions, including aneurysm (most notably of the posterior communicating artery), intracranial neoplasm and haemorrhage. A new onset cranial nerve III palsy associated with pain and pupil changes should be treated as a medical emergency, as this is suggestive of an unruptured aneurysm which requires urgent neuroimaging. Even though a patient may not have pupil

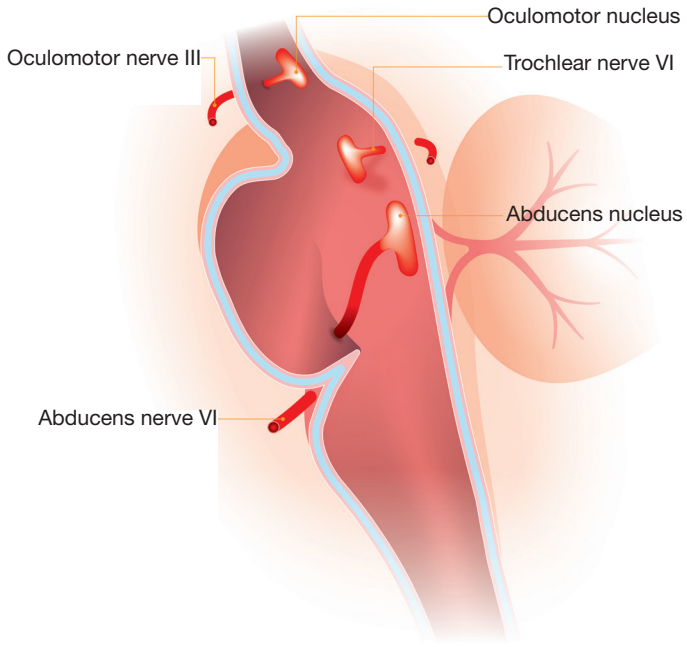

Figure 4. Neuroanatomical positions of cranial nerves III, IV and VI in the brainstem.

Table 2. Clinical neuroanatomical correlation of cranial nerve lesions

Cranial nerve	Nucleus location	Clinical features of lesions at the nuclei	Fascicular pathway	Clinical features of lesions along the fascicular pathway	Subarachnoid space	Clinical features of lesions in the subarachnoid space
Cranial nerve III	Midbrain at level of superior colliculus	Ipsilateral cranial nerve III palsy plus ■ Bilateral ptosis+/- contralateral superior rectus weakness	Caudal to rostral: ■ Medial longitudinal fasciculus ■ Red nucleus interpeduncular fossa ■ Lateral to the posterior communicating artery	Ipsilateral cranial nerve III palsy plus Superior cerebellar peduncle: Nothnagel's syndrome: cerebellar ataxia Red nucleus: Benedikt's syndrome: contralateral limb tremor Cerebral peduncle: Weber's syndrome: contralateral limb hemiplegia	Lateral to posterior communicating artery	Compressive lesions: pupil involving cranial nerve III palsy Ischaemic lesions: non-pupil involving cranial nerve III palsy
Cranial nerve IV	Midbrain at level of inferior colliculus	Contralateral cranial nerve IV palsy plus ■ Ipsilateral internuclear ophthalmoplegia ■ Relative afferent pupillary defect ■ Ipsilateral Horner's syndrome ■ Ipsilateral intention tremor or ataxia	Exits brainstem dorsally	Contralateral cranial nerve IV palsy plus ■ Ipsilateral internuclear ophthalmoplegia ■ Relative afferent pupillary defect ■ Ipsilateral Horner's syndrome ■ Ipsilateral intention tremor or ataxia	Travels between cerebellar and posterior cerebral arteries	Trauma: high susceptibility of unilateral or bilateral cranial nerve IV palsy, because of the long intracranial course
Cranial nerve VI	Lower pons, anterior to fourth ventricle and level of facial colliculus	Ipsilateral cranial nerve VI palsy plus ■ Ipsilateral horizontal gaze palsy ■ Ipsilateral internuclear ophthalmoplegia ■ Foville syndrome: ipsilateral cranial nerve V (loss of facial pain and temperature sensation), VII (facial weakness) and VIII (hearing) ■ Contralateral hemiparesis ■ Ipsilateral Horner's syndrome	Within pons: through medial lemniscus and corticospinal tract Exits at pontomedullary junction	Ipsilateral cranial nerve VI palsy plus ■ Foville syndrome ■ Raymond syndrome: contralateral hemiparesis and facial paresis ■ Millard–Gubler syndrome: ipsilateral cranial nerve VII palsy and contralateral hemiplegia	Transverses between pons and clivus, and crosses over petrous apex of temporal bone	False localising sign (see text)

Table 3. The C-VITAMIN mnemonic for causes of cranial nerve palsies
Congenital
Vascular
Infectious
Trauma
Autoimmune
Metabolic
Idiopathic or iatrogenic
Neoplastic

involvement, neuroimaging is still warranted given the variability in presentation of cranial nerve III palsy caused by aneurysm (Margolin, 2020). An idiopathic isolated cranial nerve III palsy can only be determined as a diagnosis of exclusion (Modi and Arsiwalla, 2021).

Cranial nerve IV palsy

Cranial nerve IV innervates the superior oblique muscle. With cranial nerve IV palsy, there is hypertropia of the paretic eye (Figure 2), with the hypertropia being worse in contralateral gaze and ipsilateral head tilt, resulting in vertical diplopia. Patients with a cranial nerve IV palsy often have a head tilt to the contralateral side (Danchaivijitr and Kennard, 2004).

Causes of cranial nerve IV palsy include microvascular insult, demyelinating disease, closed head trauma and congenital causes. Investigations for an acute cranial nerve IV palsy depend on the presenting factors. Patients over the age of 50 years with an acute isolated cranial nerve IV palsy likely have a microvascular cause, so neuroimaging is not required and optimisation of cardiovascular risk factors is important. However, if the patient has brainstem symptoms (Figure 4) or is under 50 years of age with no history of a congenital cranial nerve IV palsy, then urgent neuroimaging and a more extensive work up are warranted (Margolin, 2020).

Cranial nerve VI palsy

Cranial nerve VI innervates the lateral rectus muscle of the eye. With cranial nerve VI palsy, the paretic eye has limitation of abduction. An esodeviation in the primary position of gaze (Figure 2), which is incomitant and increased when the patient is looking towards the affected side and for distance fixation, results in horizontal diplopia mainly for distance. Cranial nerve VI palsy is mostly caused by a microvascular insult, but haemorrhage, trauma, intracranial neoplasm, systemic neurological disease and idiopathic causes are all possible (Elder et al, 2016; Kim et al, 2018). Cranial nerve VI has a unique anatomy and its intracranial course over the petrous apex of the temporal bones and within Dorello's canal means it is sensitive to changes in intracranial pressure (Danchaivijitr and Kennard, 2004). Symmetrical or asymmetrical bilateral cranial nerve IV palsy could be related to raised intracranial pressure (a false localising sign). In the presence of a false localising sign, it is important to perform a fundoscopy to identify any papilloedema (optic disc swelling as a result of raised intracranial pressure). Children who present with an acute esodeviation (without any previous history of strabismus), abduction deficit and evidence of papilloedema on fundoscopy should be urgently referred to paediatrics because there is a significant risk of this being caused by a neoplasm (Graham and Mohseni, 2021).

Cavernous sinus syndrome

The cavernous sinuses are located in the middle cranial fossa either side of the sella turcica of the sphenoid bone, with the temporal bone laterally. They contain the internal carotid artery, sympathetic nerves, cranial nerves III, IV and VI, the ophthalmic and maxillary divisions of cranial nerve V and drainage of the ophthalmic veins (Figure 5) (Graham and Mohseni, 2021).

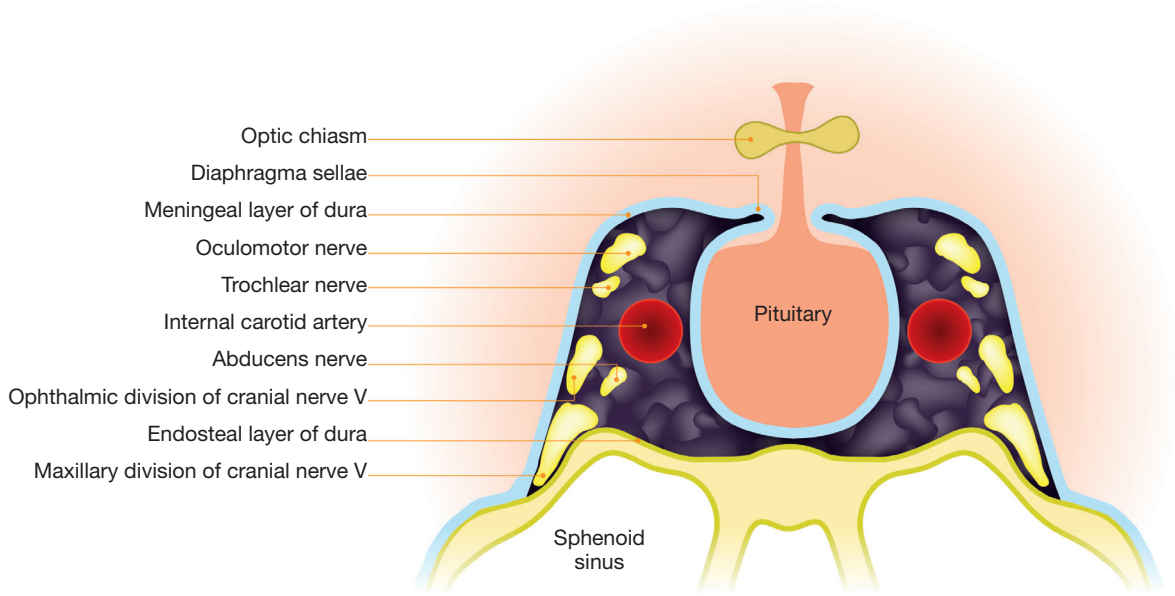

Figure 5. Cavernous sinus and its anatomical structures.

Cavernous sinus syndrome includes any disease that involves the cavernous sinus. The most common causes are tumours, including meningiomas, nasopharyngeal carcinomas, lymphoma and metastases. Vascular causes such as carotid-cavernous fistulas and infectious causes such as cavernous sinus thrombosis are also important causes of cavernous sinus syndrome (Plewa et al, 2021).

Symptoms suggestive of cavernous sinus syndrome include diplopia as a result of the involvement of cranial nerve III, IV and VI causing marked ophthalmoplegia, headache (reported in 90% of patients), ptosis, proptosis, chemosis, facial sensory loss, visual loss, fever, facial asymmetry, hearing loss and seizure (Kuybu and Dossani, 2022). These symptoms are similar to those for orbital inflammatory conditions, such as orbital apex syndrome and superior orbital fissure syndrome, with profound vision loss being a discriminating factor in favour of orbital apex syndrome. Any combination of these symptoms should alert the clinician to request urgent neuroimaging.

Conclusions

History and examination are essential when assessing a patient with diplopia. Diplopia is usually caused by dysfunction of the extraocular muscles, whether it be the nerves that supply them or the actual muscles themselves. The differential diagnoses for this dysfunction vary. By understanding the clinical presentation, important features of the history and examination, as well as the corresponding anatomical correlations, the cause of diplopia can be accurately determined. Time-sensitive diagnosis is particularly important, as life-threatening conditions can present with diplopia. In the acute setting, the patient's diplopia can be alleviated with conservative measures such as occlusion, or prisms once they are assessed by a strabismus specialist. Strabismus surgery is an option once there is stabilisation of the ocular motility measurements.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

Key points

- Diplopia is the perception of seeing two of a single object.
- Delineating the characteristics of the diplopia perceived by the patient will guide further examination and investigation.
- Binocular diplopia is a result of disparity of the stimulation of the retina between the two eyes.
- The most common cause of binocular diplopia is strabismus, which can be paretic or restrictive, comitant or incomitant, manifest or latent.
- Central and peripheral cranial nerve palsies may be differentiated by the presence of associated signs and symptoms, which aids neuroanatomical localisation of the lesion.
- Acute diplopia is treated conservatively with prisms or occlusion, while strabismus surgery may be an option once there is stability of ocular motility measurements.

Curriculum checklist

This article addresses the following requirements from the general internal medicine training curriculum.

- Managing an acute specialty-related take
- Managing patients in an outpatient clinic, ambulatory or community setting, including management of long-term conditions
- Is focused on patient safety and delivers effective quality improvement in patient care.

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