LOOKING AT BLINDNESS FROM NEUROLOGIST'S PERSPECTIVE

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LAURENT S GAROSI provides a detailed guide on the anatomy of the optic nerve and how to assess, evaluate and diagnose unilateral and bilateral vision loss

Anatomy of visual and PLR pathways

The optic nerve is not a "true" nerve, but an extension of the brain. It is part of the central visual pathway (involved in sensory visual perception) and the afferent component of the pupillary light reflex (PLR).

The visual pathway involves three consecutive neurons (Figure 1):

• **Neuron one.** This represents the bipolar cells of the retina and receives visual information from the neuroepithelial cell of the retina (for example, rods and cones).

• **Neuron two.** This corresponds to the ganglion cell of the retina. Its axons lie in the optic nerve and continue through the optic chiasm and proximal part of the optic tract of the opposite side (55 per cent decussation in man, 66 per cent decussation in felines, 75 per cent decussation in canines).

• **Neuron three.** Its cell body lies in the lateral geniculate nucleus in the diencephalon. Its axon projects to the visual cortex (mostly controlateral occipital cortex) in a band of fibres called the optic radiation.

The afferent part of the PLR shares some common pathways (up to the level of the optic tract) with the central visual pathways. While axons involved in vision reach conscious level after synapsing with the lateral geniculate nucleus, the axons involved in the PLR synapse with a third of neurons in the pretectal nucleus. Most of the axons arising from this nucleus decussate again and synapse on in the parasympathetic component of the oculomotor nucleus (ipsilateral to the stimulated eye) in the mesencephalon.

There are also neurons that do not decussate, and project to the oculomotor nucleus on the controlateral side of the stimulated eye. The proportion of axons that decussate is higher than those that do not. This explains why the direct response (constriction in the eye receiving the light stimulus) is greater than the consensual response (constriction in the eye not receiving the light stimulus).

The efferent part of the PLR is performed by oculomotor (CN III), causing constriction of the pupil. The PLR tests the integrity of the optic nerve and proximal part of the optic tract (decussation of the PLR fibres before the level of the lateral geniculate nucleus), but does not test the animal's vision.

The PLR should, therefore, be performed in all blind animals to determine the location of the lesion. The PLR can also be absent in the presence of primary or secondary iris disease (iris atrophy, iris inflammation and synechiation, leading to pupil fixation) and pharmacological blockade due to atropine or atropine-like drugs.

Methods for assessment of vision

Clinical evaluation of a suspected blind animal consists of obstacle course evaluation, testing the menace response in relation to the pupils' size and their response to light, and visual placing. If unilateral vision loss is suspected, each eye can be blindfolded in turn prior to completion of the obstacle course. A thorough neurological and eye examination should be conducted.

• Obstacle course evaluation

This test evaluates the ability of the animal to negotiate an obstacle course. For the most reliable information, the course should be evaluated binocularly and monocularly. The latter is, unfortunately, very difficult to practically apply in small animals, making this test a poorly sensitive method to assess subtle or unilateral visual impairment.

• Menace response test (Figure 2)

Eyelid closure, with or without head withdrawal, is produced by a threatening or unexpected image suddenly appearing in the near-visual field. This test is a cortically mediated eyelid closure and, therefore, represents a response (as compared to a reflex not involving the cortex). The menace response is a learned response that is present from about five to six weeks of age, depending not

only on the species, but also the breed.

The afferent arc of this response involves a number of neurons: the first in this arc is the bipolar cell of the retina. This receives impulses from the neuroepithelial cells of the retina (rods and cones). The second afferent neuron is the ganglion cells of the retina. Its axons lie in the optic nerve and continue through the optic chiasm and proximal part of the optic tract of the opposite side (55 per cent decussation in man, 66 per cent in cats, 75 per cent in dogs and 80 to 90 per cent in horses).

This second neuron synapses with neuron three in the lateral geniculate nucleus. The axons then project to the visual cortex (mostly occipital cortex) in a band of fibres called the optic radiation.

The efferent arc of this response is not well understood. The information generated in the optic cortex (contralateral to the eye stimulated) is forwarded to the motor cortex via association fibers. The cortico-bulbar pathways to the facial nerve nucleus (CN VII) then transmit the motor information. This response requires an intact facial nerve function. This function should be separately evaluated using the palpebral reflex.

There is some experimental and clinical evidence for a cerebellar involvement in the menace response efferent pathways: unilateral cerebellar lesions can lead to an ipsilateral menace response loss with retention of normal vision. The neuronal pathways through the cerebellum are, however, not known.

• **Visual placing reaction**

The visual placing response is tested by carrying a dog or a cat under its chest towards a table edge, but without letting the thoracic limbs touch the table. On approaching the surface, the animal will reach out to support itself on the table. This response requires intact visual and motor pathways.

Clinical evaluation

• **Combined results of menace response and pupillary light reflex** (Figure 3)

The optic nerve is the common component of the afferent pathways involved in vision, menace response, visual placing and the PLR. These tests use different integration centres within the brain and different efferent pathways. The integrity of the optic nerve can be determined by combining the results of these tests. The PLR tests the integrity of the optic nerve to the level of the lateral geniculate nucleus, but it does not test the animal’s vision. The PLR should, therefore, be performed in all blind animals to determine the location of the lesion.

Fundic examination and evaluation of the appearance of the optic disc are also important parts of the evaluation of the optic nerve.

• **Evaluation of signs of “brain disease”**

Suspicion of brain involvement can be based on historical findings (change in behaviour, epileptic seizures, change in mentation), as well as neurological examination findings. The latter should particularly focus on evaluating the following: change in mental status (depression, stupor, delirium); abnormal body posture at rest (head tilt, head turn); postural reaction; abnormal gait (ataxia with or without paresis, circling); and cranial nerve deficit.

Anatomic diagnosis and differential diagnosis

The neurological examination aims to test the integrity of these various components of the nervous system and, if present, detect any functional deficit. Normal findings are as important as the abnormal in establishing the anatomic diagnosis. Based on the animal’s history and neurological examination, the clinician can determine if the animal suffers from a neurological disease. If so, attempts should be made to localise the lesion within the nervous system (anatomic diagnosis) prior to establishing a differential diagnosis list.

The interpretation of the neurological evaluation should begin by making a list of the abnormal results collected from the history and examination (for instance, menace response). Each of these abnormal findings should then be correlated to a specific region or to a specific pathway with the nervous system. An attempt should always be made to explain all the abnormal findings by a single lesion within one of the regions of the nervous system. If a single lesion cannot explain all the listed abnormal findings, the anatomical diagnosis is considered as multifocal or diffuse.

The second step is to form a differential diagnosis list. This is essential in choosing and interpreting any diagnostic test. Disease processes that can affect the nervous system are classically classified according to the pneumonic DAMNIT V (**D**egenerative - **A**nomalous - **M**etabolic - **N**eoplastic/ **N** utritional - **I**nflammatory/**I**nfectious/ **I**diopathic - **T**raumatic/**T**oxic - **V**ascular). Each of these disease processes has a typical signalment, onset and progression, as well as distribution within the nervous system.

• **Unilateral abnormal vision and PLR deficit** (Figure 4)

Concurrent loss of vision and PLR is suggestive of a lesion affecting the portions of the visual pathways that are common with the PLR pathways (such as ipsilateral ocular media and retina, ipsilateral optic nerve, and contro lateral optic tract up to the level of the lateral geniculate nucleus). Differentials for such a focal lesion include: lesions causing opacification of the ocular media (cornea, aqueous humour, lens and vitreous humour) or affecting the retina (I: retinitis/chorioretinitis; I, V and T: retinal detachment); the ipsilateral optic nerve (A: optic nerve hypoplasia; N: neoplasia of the optic nerve or neoplasia compressing the optic nerve; I: infectious or non-infectious optic neuritis; I: retrobulbar abscess/cellulites; T: trauma to the globe and orbit); or the contralateral optic tract up to the level of the lateral geniculate nucleus (T: hypotha lamic and

thalamic neoplasia; V: cerebrovascular accident).

• **Unilateral abnormal vision and intact PLR** (Figure 5)

Unilateral loss of vision with intact PLR suggests a lesion affecting the portions of the visual pathways that are not common with the PLR pathways (for instance, central portions of the visual pathway from the contralateral lateral geniculate nucleus to the contralateral visual cortex).

As a sole neurological finding, it is mainly seen with focal contralateral forebrain disease (**N**: primary or secondary brain tumour; **I**: inflammatory/ infectious CNS disease; **T**: head trauma; **V**: cerebrovascular accident).

• **Bilateral abnormal vision and PLR deficit** (Figures 6, 7 and 8) Bilateral loss of vision and PLR is suggestive of a lesion that bilaterally affects the portions of the visual pathways that are common with the PLR pathways (that is, bilateral ocular media and retina, bilateral optic nerve or optic tract up to the level of the lateral geniculate nucleus and focal optic chiasm).

Differentials include bilateral lesions causing:

• opacification of the ocular media (cornea, aqueous humour, lens and vitreous humour) or affecting retina (**D**: sudden acquired retinal degeneration; **A**: inherited retinal disorder - progressive retinal atrophy; **I**: retinitis/chorioretinitis; **I**, **V** and **T**: retinal detachment [Figure 6]);

• damage to the optic nerves (A: optic nerve hypoplasia; I: infectious or non-infectious optic neuritis [Figure 7]); or

• an optic chiasm or lesion affecting the optic tracts up to the level of the lateral geniculate nuclei ([Figure 8] **N**: neoplasia, such as meningioma, pituitary macroadenoma; **I**: inflammatory/ infectious CNS disease; **V**: ischaemic necrosis of the optic chiasm).

• **Bilateral abnormal vision and intact PLR** (Figure 9)

Bilateral loss of vision with intact PLR suggests a lesion affecting the portions of the visual pathways that are not common with the PLR pathways (that is, bilateral central portions of the visual pathway from the contralateral lateral geniculate nucleus to the contralateral visual cortex equals diffuse or multifocal forebrain lesion).

It should be noted that the PLR requires fewer intact axons than conscious perception of vision and, therefore, partial lesion of the proximal visual pathways (that is, retina and optic nerve) may cause loss of vision while sparing the PLR, creating the illusion of a more central lesion.

In general, other clinical signs of forebrain disorder would usually be expected in forebrain lesions

severe enough to cause visual deficits.

These would be more common with the diffuse or multifocal disease process, such as **A**: anomalous lesions (especially hydrocephalus); **M**: metabolic encephalopathy (especially hepatic encephalopathy and hypoglycaemia); **I**: inflammatory (granulomatous meningoencephalitis) or infectious (neosporosis, toxoplasmosis, distemper or bacterial) CNS disease; **T**: head trauma; **T**: toxins (especially lead poisoning); and **V**: cerebrovascular disease. They are also seen with space occupying lesions, such as **N**: primary and secondary brain tumour and brain haemorrhage.

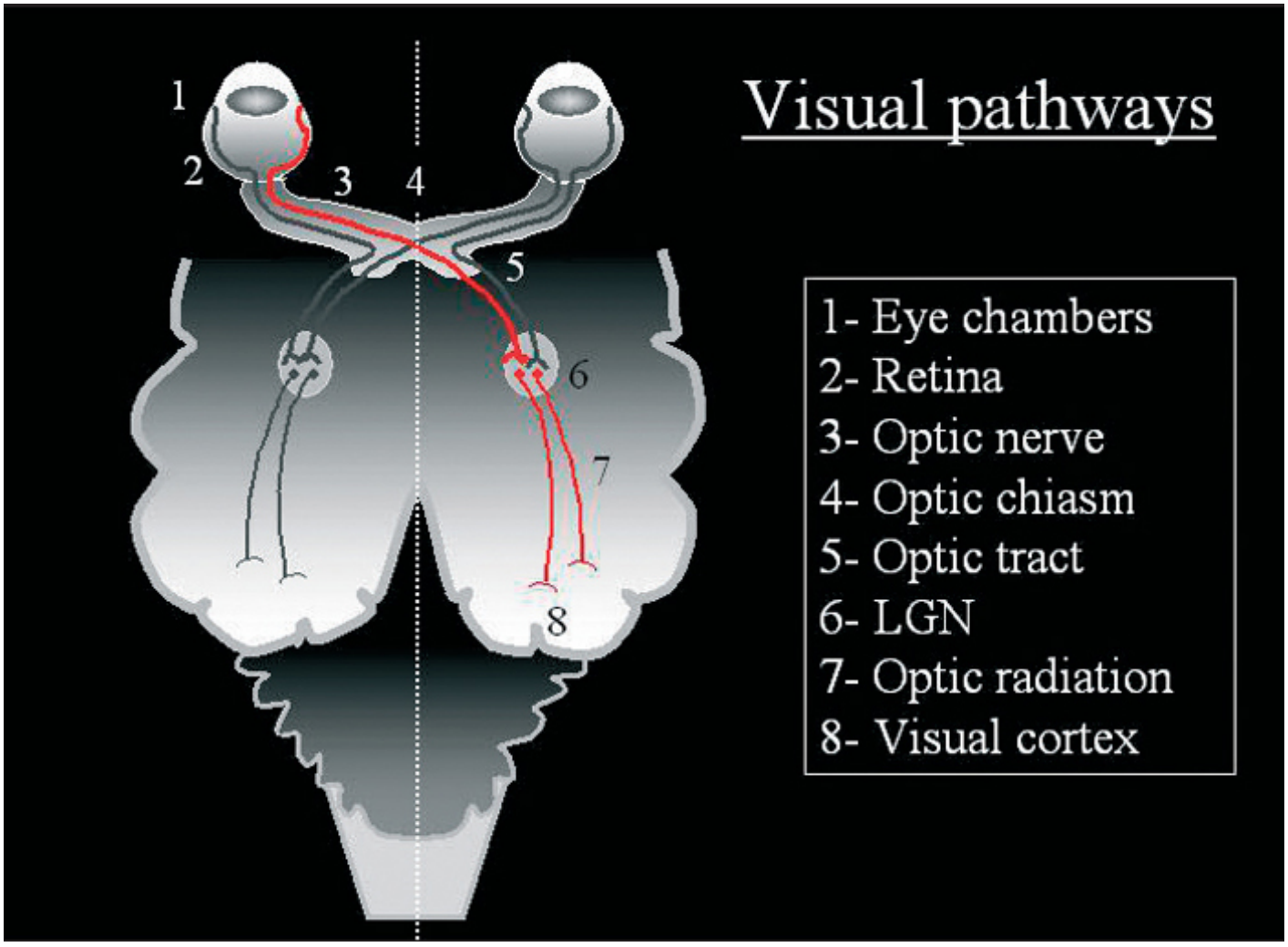

Figure 1. The visual pathway, which involves three consecutive neurons.

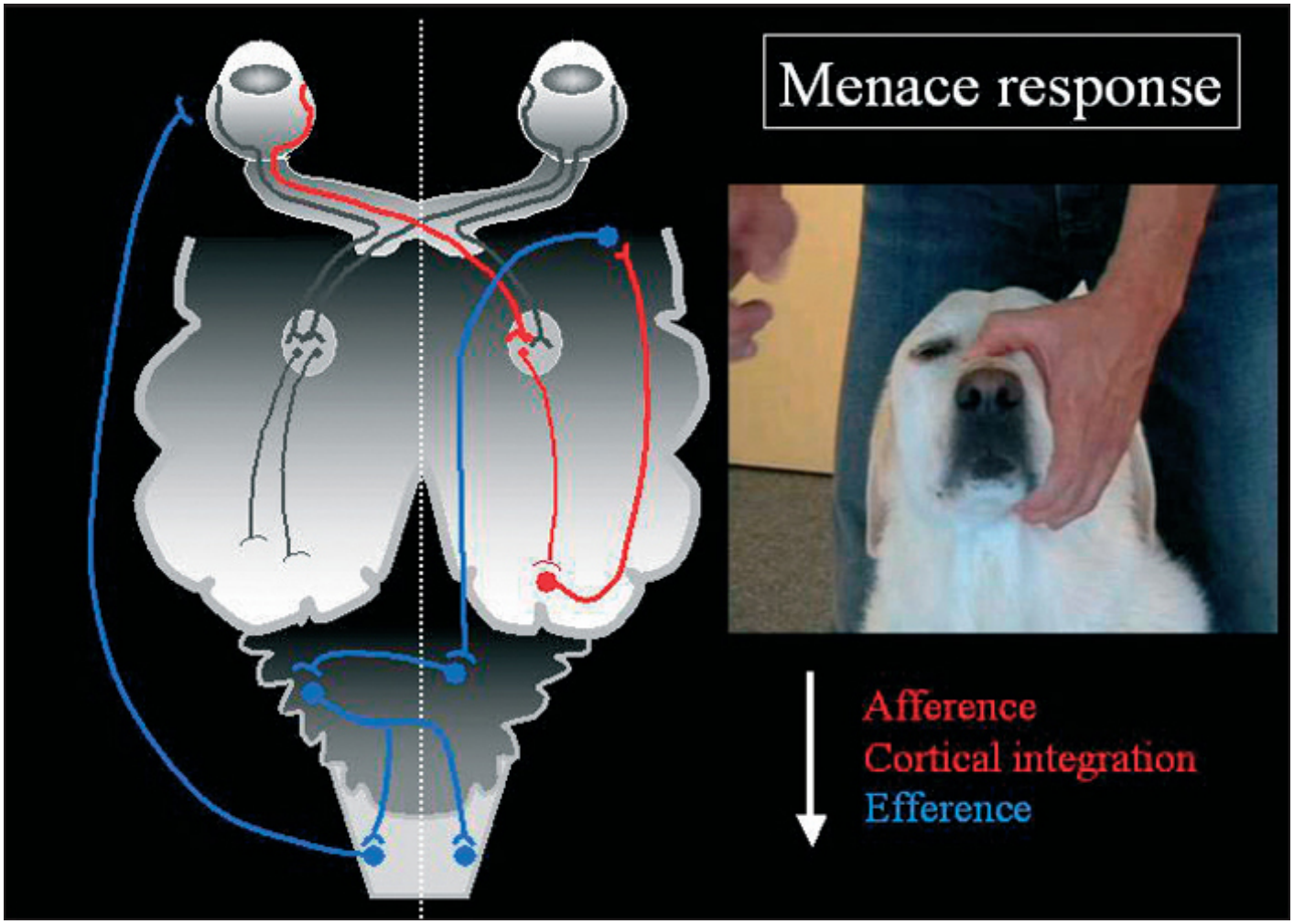

Figure 2. The menace response test.

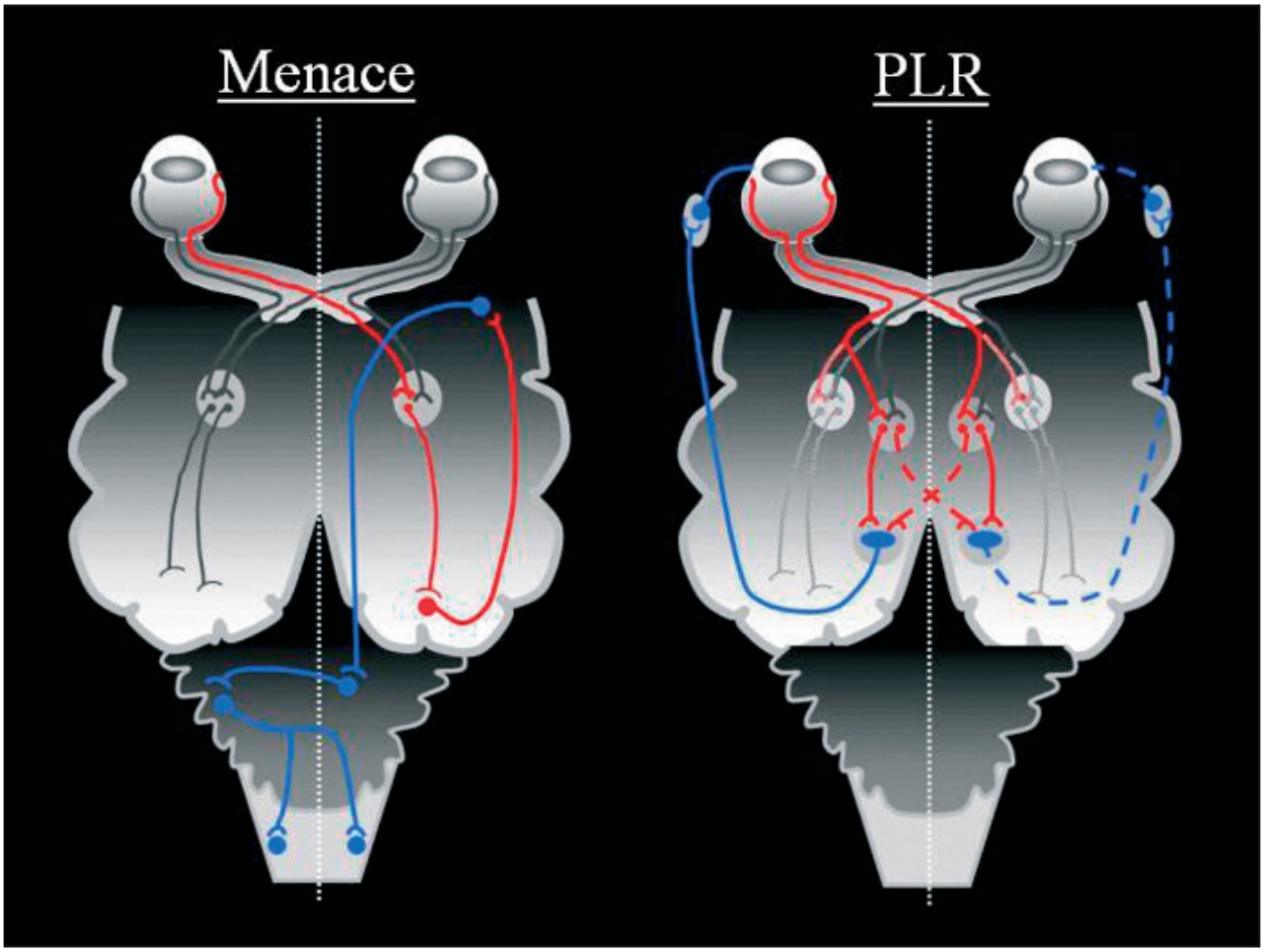

Figure 3. The combined results of the menace response test and pupillary light reflex.

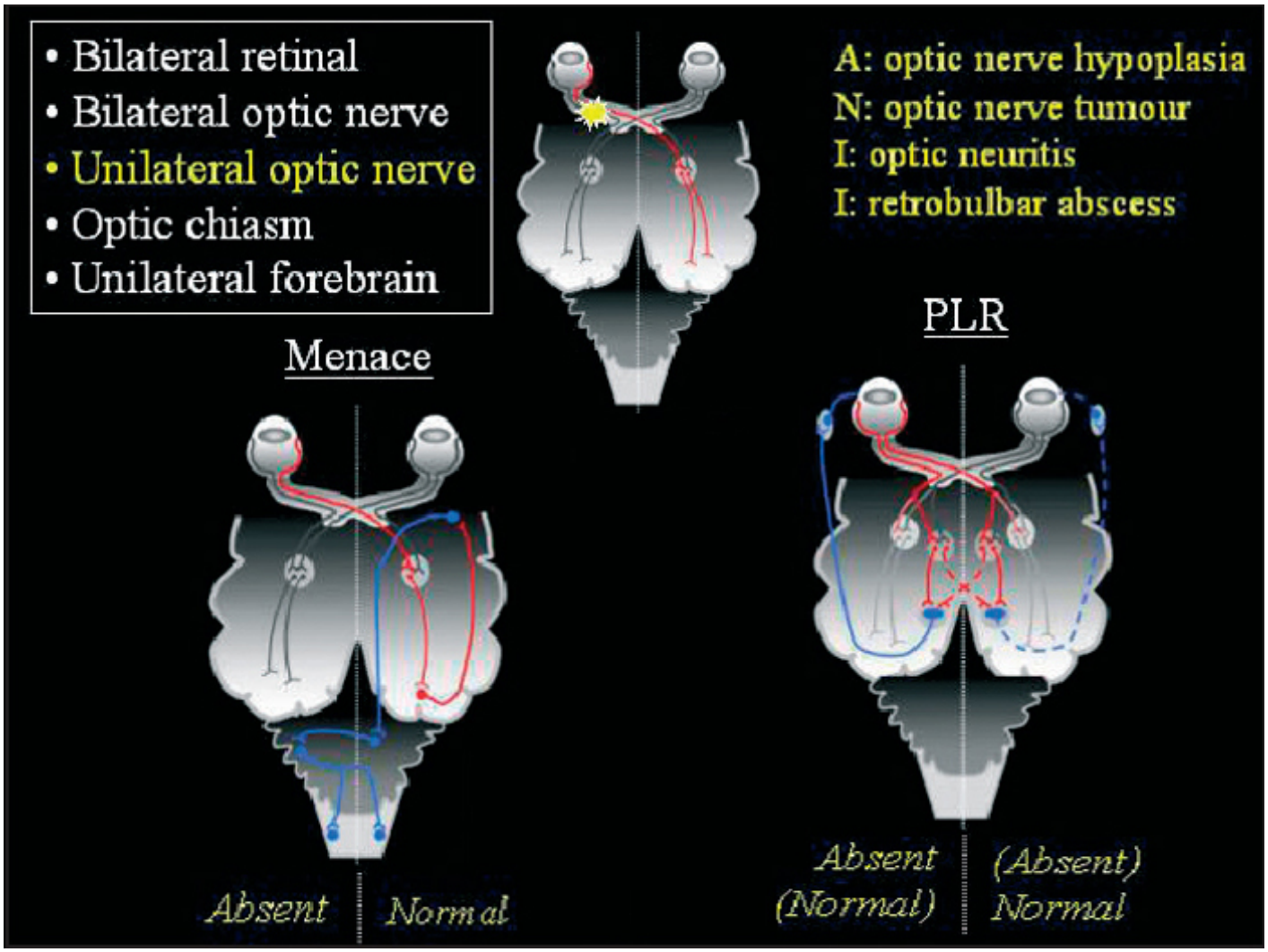

Figure 4. Unilateral abnormal vision and PLR deficit is suggestive of a lesion affecting the portions of the visual pathways that are common with the PLR pathways.

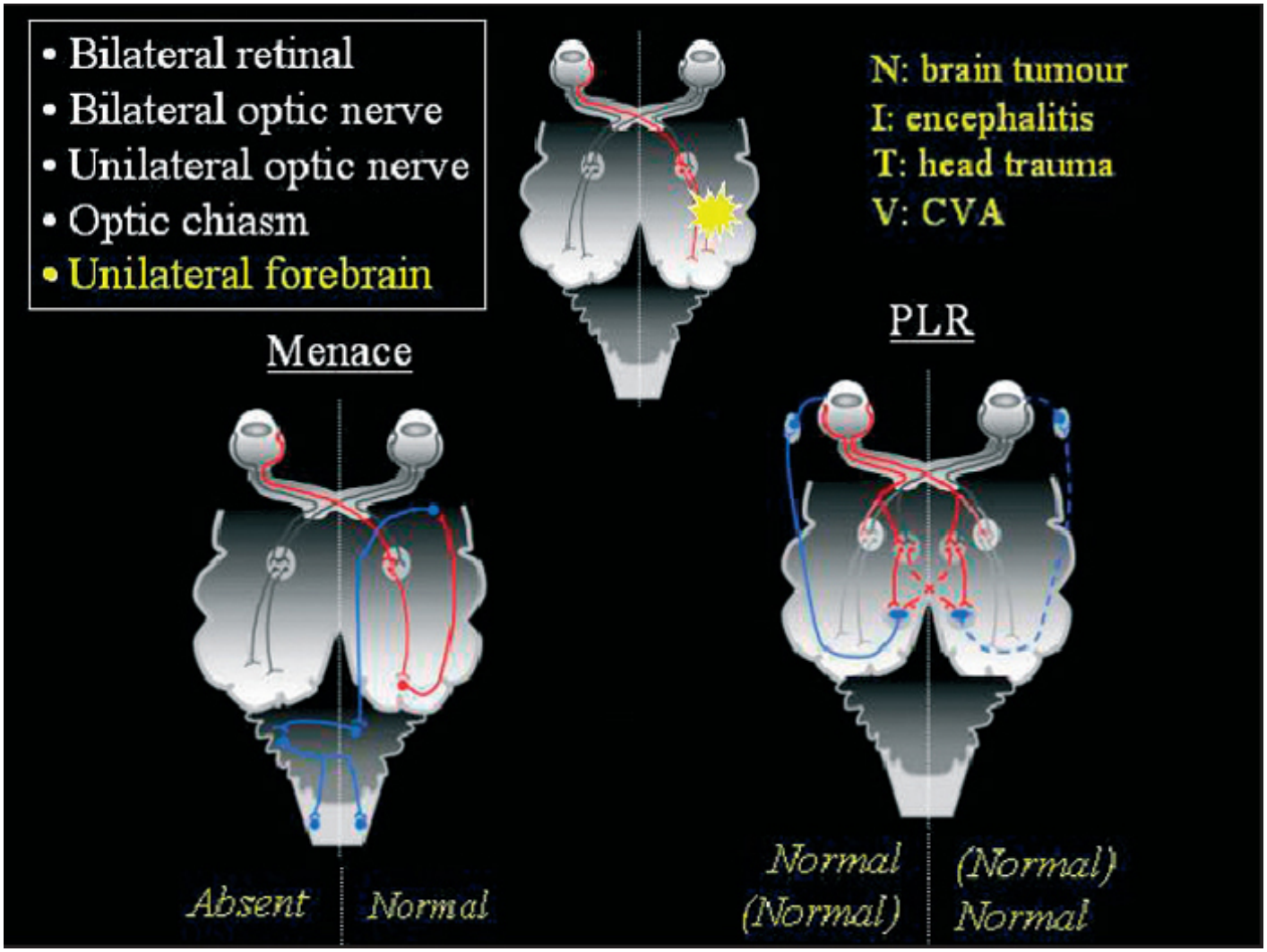
• Bilateral retinal
• Bilateral optic nerve
• Unilateral optic nerve
• Optic chiasm
• Unilateral forebrain
N: brain tumour
I: encephalitis
T: head trauma
V: CVA
Menace
PLR
Absent
Normal
Normal
(Normal)
(Normal)
Normal

Figure 5. Unilateral abnormal vision and intact PLR suggests a lesion affecting the portions of the visual pathways that are not common with the PLR pathways.

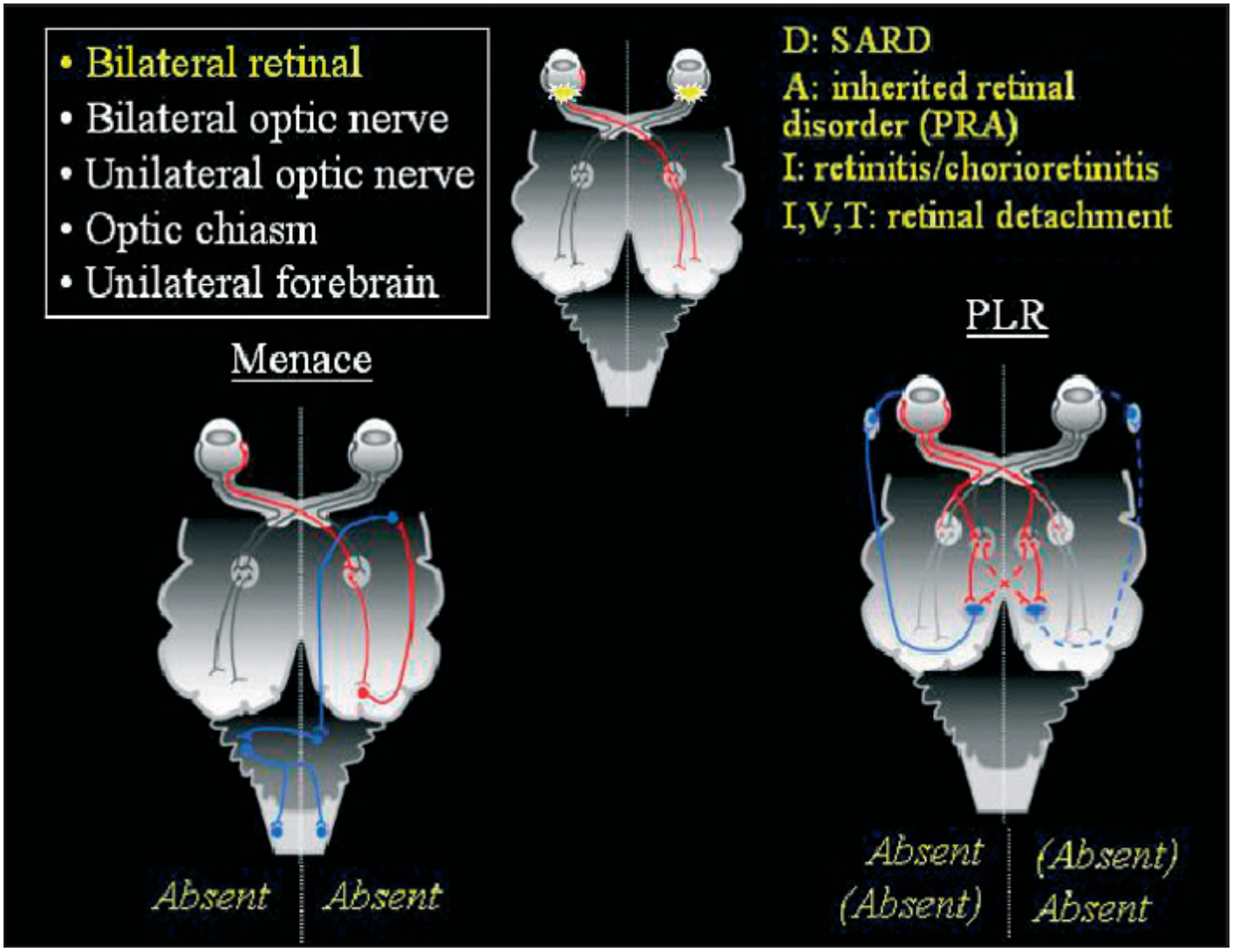

Figure 6. Bilateral lesions causing opacification of the ocular media or affecting retina.

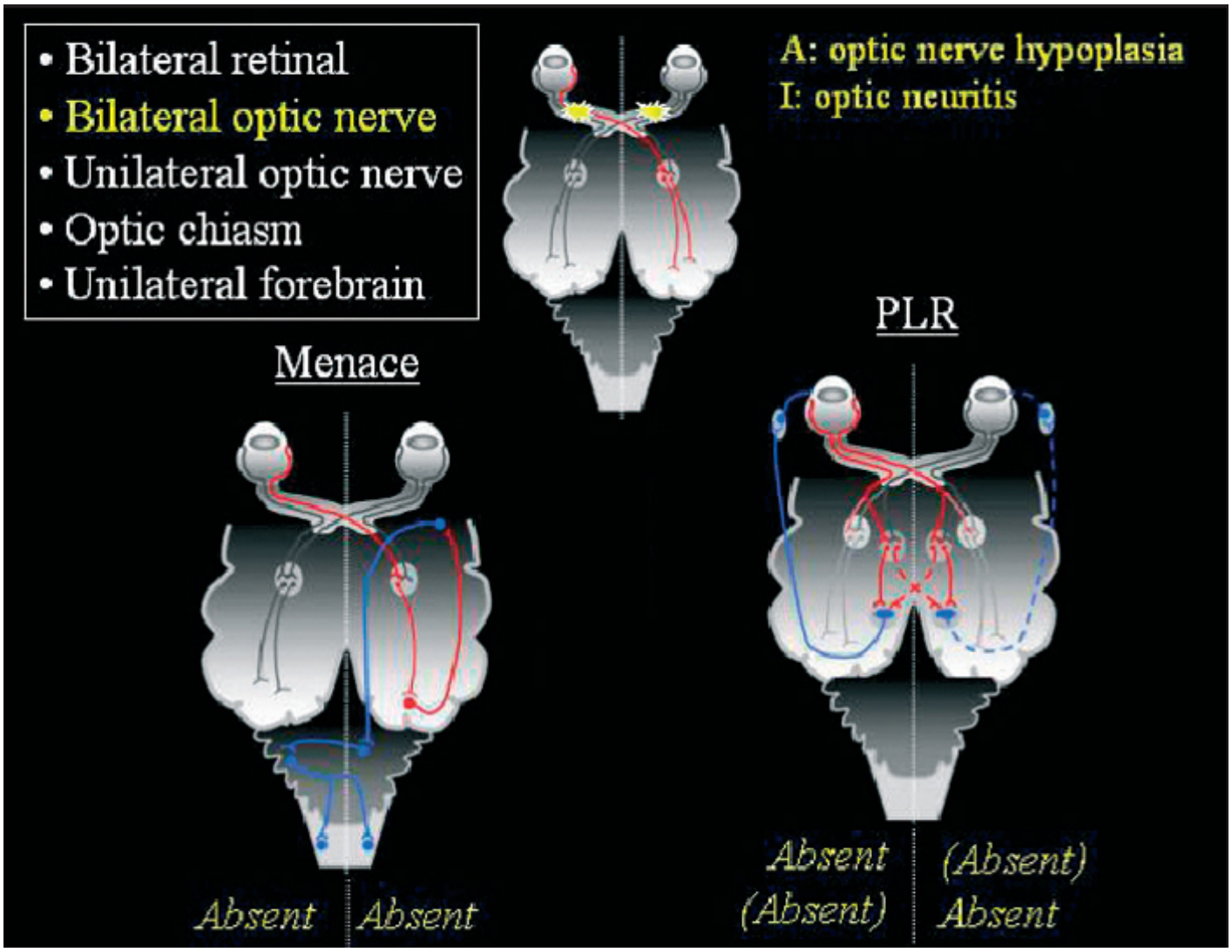

Figure 7. Here, the effect of bilateral lesions on the optic nerve is depicted.

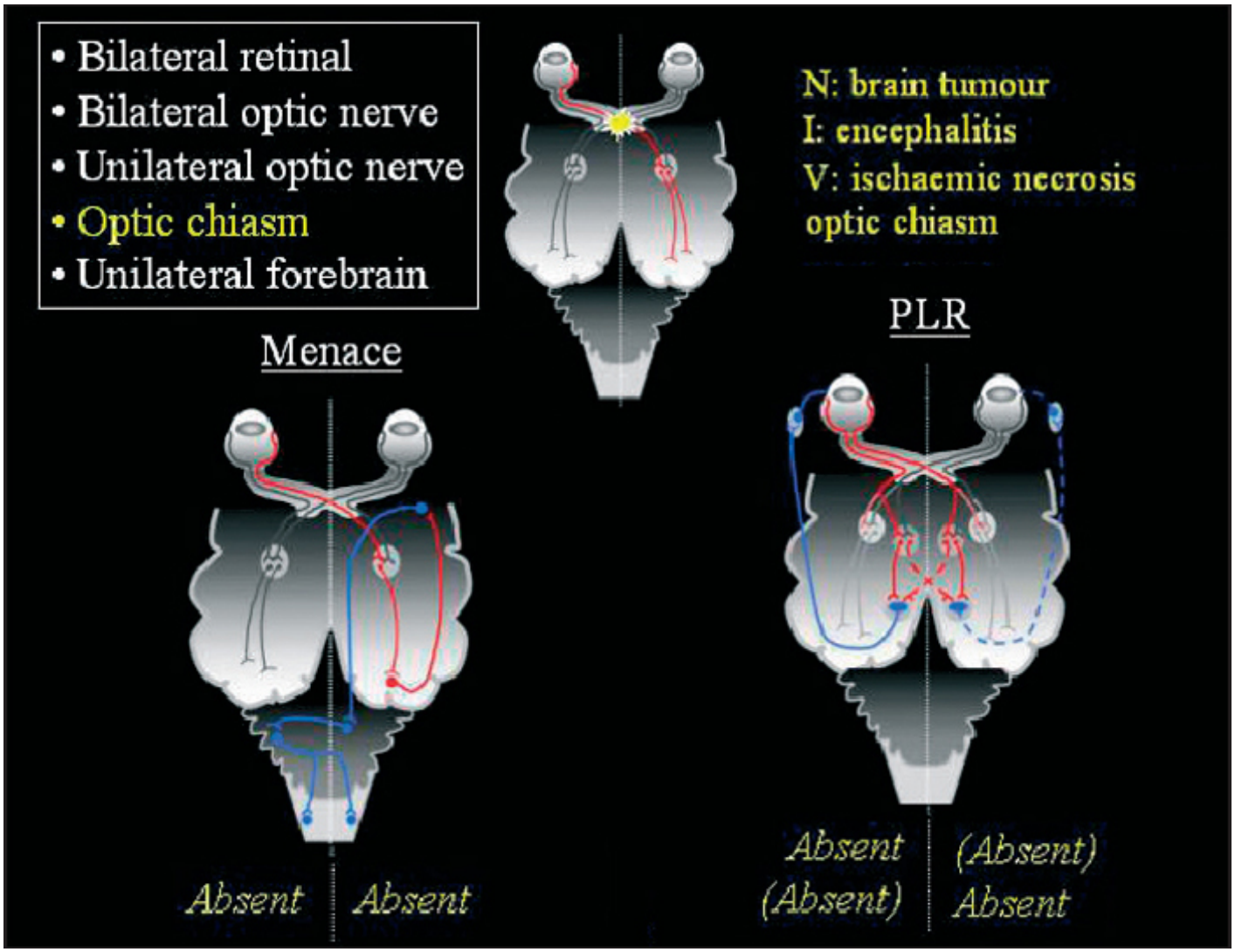

Figure 8. An optic chiasm affecting optic tracts to the level of the lateral geniculate nuclei.

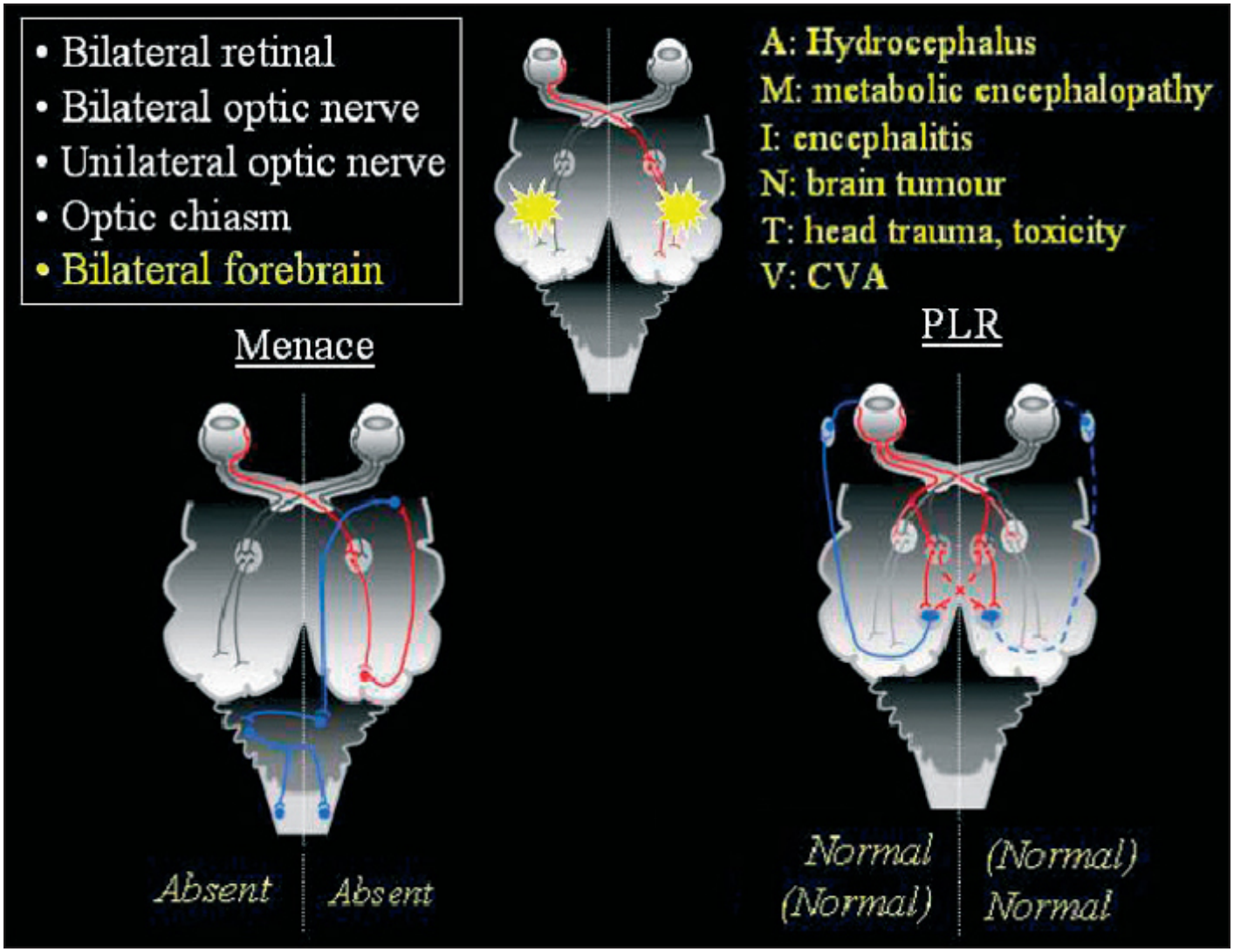

Figure 9. This diagram shows an example of bilateral abnormal vision and intact PLR.