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Review Article

An Iterative, Clinical Question-based Paradigm for Performing a Neuroanatomical Diagnosis During a Neuro-Ophthalmologic Evaluation in Dogs and Cats

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Abstract

Background: Assessment of appropriate innervation of the eye and integrated structures including the extraocular muscles is essential when animals present for vision, pupil, or ocular movement abnormalities. The complexity of neurologic influences on vision, pupil size and reactivity, and ocular motion can be challenging, however, for the clinician to systematically evaluate during this assessment. Following an iterative, clinical question-based evaluation process avoids omission of important clinical assessments and increases the likelihood of accuracy and thoroughness during neuroanatomical diagnosis of neurologic abnormalities involving the eyes and extraocular supporting structures. *Purpose:* Codification of a structured clinical assessment paradigm for evaluation of neuro-ophthalmological functions in animals. *Methods:* The clinical assessment paradigm presented in this review can be used as a read-do question prompt algorithm to ensure a consistent, stereotypic, and comprehensive evaluative process. *Results:* This approach contains prompted closed decision-forcing questions that are answered in a structured, iterative format. The individual questions posed ensure that the clinician examines all the important aspects of neurologic innervation of ocular functions. The aggregate of question prompts is balanced for efficiency and completeness focusing the clinician's clinical evaluative process in patients with vision, pupil, or ocular movement abnormalities. *Conclusions:* This structured approach can support an accurate neuroanatomical diagnosis when evaluating dogs and cats with abnormalities of vision, pupils, and ocular movement by avoiding omission of important clinical assessments. A similar approach can be modified or adapted for evaluation of other species based on their unique anatomy and physiology of the ocular structures and innervation.

Keywords

Eyes, Neuro-ophthalmology, Neurologic Assessment, Neuroanatomical Examination, Clinical Diagnosis

1. Background

Abnormalities of nervous innervation can affect vision, pupil size, pupil reactivity, and ocular movements [1-15]. Given the complexity of ocular anatomy and functionality, it is easy to become overwhelmed and overlook important clinical considerations when assessing potential neurologic abnormalities that affect ocular functions. Utilizing an iterative approach prompts the clinician to assess the various structural elements and neurologic functions of the eyes in a comprehensive manner to determine if a neurologic abnormality exists and equally, where that abnormality occurs within the nervous system

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(neuroanatomical localization). Following identification of neurologic abnormalities affecting the eye and where anatomically those abnormalities exist the clinician can formulate appropriate differential diagnoses lists, diagnostic testing plans, treatment options, and prognosis for affected animals.

This review includes a structured question/answer debiasing algorithm used during clinical case evaluation in animals presented for potential abnormalities of vision, pupils, and ocular movements. This approach contains prompted closed decision-forcing questions that are answered in a structured, iterative format. The aggregate of question prompts is balanced for efficiency and completeness. Closed questions are force-choice fixed responses (e.g. "Is this animal normal?"- "Yes/No") to direct clinical decision-making. This approach is functionally similar to a read-do checklist philosophy to ensure that critical steps in the clinical decision-making process are not forgotten [16-21]. Based on the answers, the clinician can make a neuroanatomical diagnosis as the primary step in generating a differential diagnosis list and a diagnostic plan. The questions are organized using a logical clinical thought process that expands clinical data collection through reflective internal questioning.

The main advantage of this approach is the explicit prompts for action which removes the need to spontaneously generate the clinical questions de novo from memory. This approach helps to avoid numerous cognitive errors associated with omission of important clinical details and result in medical errors.

The initial evaluation process begins with a general observational assessment of whether the animal has behavioral actions that suggest they have a visual acuity abnormality. Owners may prompt this evaluation by expressing this concern based on their personal assessments and intuitive understanding of their animal's normal behavior. Classically, "bumping into things" is a commonly articulated observation. In unfamiliar environments such as the hospital examination area, animals with visual acuity abnormalities may not move freely likely to avoid walking into an object such as a wall or chair. In other instances, owners do not articulate a potential visual acuity abnormality in their animal but rather present their animal for evaluation of other systemic pathophysiological concerns such as the classical "isn't doing right."

2. Clinical Assessment 1

Are the ocular structures (externally) normal? (Figure 1)

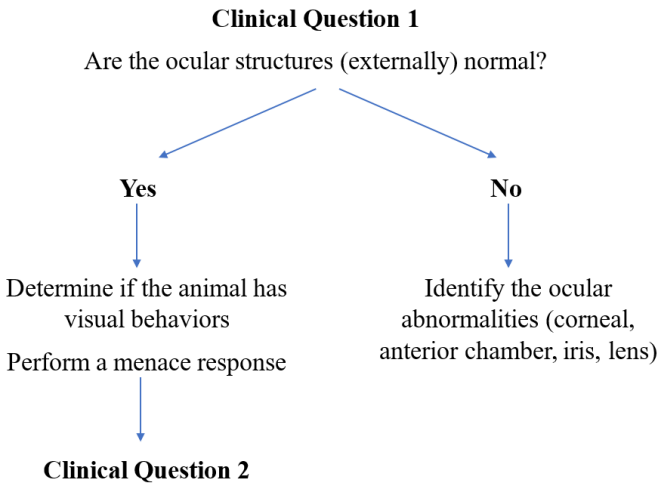


Figure 1. *Clinical Question 1 determines the integrity of the ocular structure.*

Animals with abnormalities of vision or pupils may have a primary ocular problem independent of a nervous system abnormality. Therefore, an appropriate ophthalmologic assessment is performed to eliminate primary ocular abnormalities as the cause of the vision and/or pupillary dysfunction. If the ocular structures are abnormal, performance of an ocular assessment of the cornea, anterior chamber, iris, lens, and retina to identify abnormalities that will impede light stimulation from activating the nervous system components associated with visual and pupillary functions is imperative. If the animal's ocular structures appear externally normal, the next assessment is to determine whether the animal has vision by performing a menace response assessment (see Clinical Assessment 2).

Importantly, some structural ocular abnormalities may have a neurologic cause. For example, normal corneal innervation is necessary for maintenance of the integrity of the corneal epithelium [1-15]. The cornea is innervated by the ophthalmic branch of the trigeminal nerve (Cranial Nerve (CN) V). If corneal innervation is abnormal, the corneal structure will become disrupted, cloudy, and a corneal ulcer can form. This ulcer most commonly is centrally located in the corneal [15].

The corneal alteration resulting from lack of appropriate corneal innervation is known as neurotropic keratitis. Tear production can also decrease when corneal sensation is abnormal as afferent sensory inputs through the ophthalmic branch of CN V are important for reflex tear production. In this instance, tear production is decreased but usually not absent. Concurrently, the ipsilateral nasal mucosa may also become dry and crusted. A decreased palpebral reflex may occur as well given that CN V provides the sensory input for this reflex. In this scenario, further evaluations of trigeminal nerve functions are indicated such as facial sensation as well as temporalis and masseter muscle size and action.

3. Clinical Assessment 2

Does the animal have a normal menace response? (Figure 2). After determining that the ocular structures are normal, next assess the menace response.

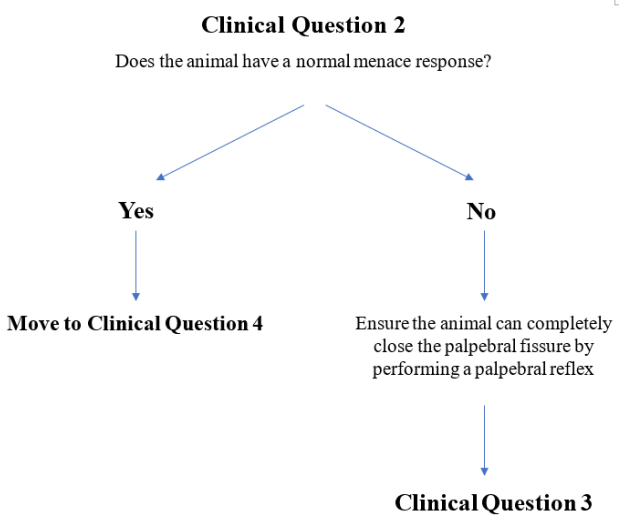


Figure 2. *Clinical Question 2 assesses the menace response.*

Visual recognition and integration are complex events within the nervous system [15]. The menace response is a simple, albeit somewhat rudimentary clinical assessment of visual function. When the menace response is positive this response suggests that the animal's nervous system has processed a visual stimulus and responded appropriately through the action of closing the palpebral fissure. Anatomical areas within the nervous system necessary for performance of the menace response include the ocular structures, the optic nerve, the central projection pathways to the visual cortex (occipital), cortical association and projection pathways coordinating the response within the intracranial nervous system structures (including the cerebellum), and finally, motor neurons necessary to complete the muscle activation resulting in closure of the palpebral fissure [1, 7, 8, 15].

The menace response is performed by the clinician making a rapid hand movement toward the eye being evaluated (i.e., the uncovered eye) while the opposite eye is covered. When making such a movement towards the animal's eye, it is important that the clinician does not touch the eye or surrounding structures (e.g., vibrissae) or create any excessive air currents which stimulate touch receptors in or around the eye independent of visual recognition. The normal response to such an intuitively threatening gesture is for the animal to close the palpebral fissure in the eye stimulated. This response occurs through both visual integration of light stimuli and ultimately activation of the facial nerve (CN VII) with resultant contraction of the orbicularis oculi muscle.

While a common test of visual function, performance of the menace response is challenging in animals that are head-shy, resist restraint, or constantly moving. Additionally, there are some animals, either due to a heightened state of fear or pain, that do not demonstrate a menace response even when visual. Lightly touching the medial or lateral canthus by following through one of the hand movements toward the eye and then performing the menace response may elicit a more consistent response in some of these patients. Very young animals may not have a menace response as this is considered a learned response [1-15].

An abnormal menace response usually indicates an abnormality either with the ocular structures, the peripheral and central nerves associated with vision, or innervation to, and appropriate muscle action of, the orbicularis oculi muscle through the motor functions of the facial nerve (CN VII). When the menace response is decreased or absent it is essential to complete the ophthalmic examination with an examination of the retina (e.g., a fundic examination), lens, posterior chamber, and optic nerve. Clues found during retinal examination may indicate pathologic abnormalities such as hypertension, coagulopathies, and inflammation/infection processes that may concurrently be affecting the nervous system.

If there is still uncertainty about whether the animal is visual after performing the menace response, additional tests of vision are indicated. Visual function, for example, can be assessed subjectively by observing whether the animal bumps into structures in the examination space or by having the animal walk through an unfamiliar area with obstacles placed in its path. It is important to remember that non-visual dogs may accommodate visual deficiencies through utilization of other senses to assess the environment. Rolling an object in front of an animal, therefore, is not a good test of vision, as hearing may be activated with the animal orienting through this sense rather than visual stimulation. Having the patient follow a light stimulus (such as a laser pointer light), object (such as a tennis ball or toy), or dropping cotton balls are alternative tests, however, animals may inconsistently respond to these visual stimuli. An animal that does not respond to these stimuli should not, therefore, be immediately assessed as having a visual abnormality. Motivating the animal to navigate through an unfamiliar obstacle course (maze test) is often the best test of vision but does require extra set-up and performance time.

Soft, non-injurious structures placed as obstacles will be less traumatic to the animal if it does have a visual deficit and subsequently contacts these obstacles while navigating the course. If vision is suspected to be absent in only a single eye, blindfolding the normal eye will help in determining vision in the questionable eye. Be careful, however, as animals with neurologic deficits may become more unsteady and/or fall with blindfolding.

One interesting nuance to menace response assessment occurs in animals with cerebellar disease. Animals with cerebellar disease will have normal vision and normal CN VII function but may not have a normal menace response. With unilateral cerebellar disease, an animal will have a menace response deficit in the eye ipsilateral to the cerebellar lesion. A pupillary dilation may also be present [22]. In addition, there are often signs of cerebellar system dysfunction such as incoordination, ataxia, hypermetria, head tilting, and falling which support this localization.

Animals that have normal visual function but cannot close the palpebral fissure because of CN VII dysfunction usually retract the eye away from the stimulus even when unable to close the palpebral fissure. An elevation of the nictitating membrane is often noted concurrently during eye retraction which provides a clinical clue that the animal was able to see the stimulus however not respond appropriately. In these instances, the clinician needs to assess the functionality of CN VII which is performed in Clinical Assessment 3.

4. Clinical Assessment 3

Can the animal blink (i.e., appropriately close the palpebral fissure)? Does the animal have a normal palpebral reflex? (Figure 3).

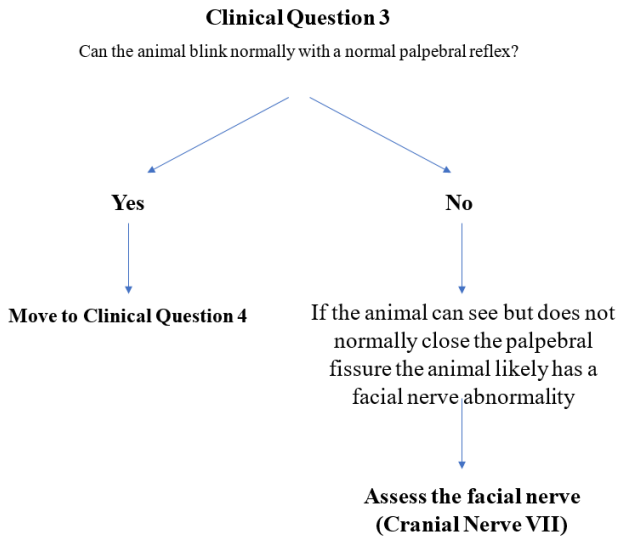


Figure 3. *Clinical Question 3 assesses the ability of the animal to close the palpebral fissure if the menace response is decreased or absent.*

Blinking ability can be assessed by simply observing natural blinking (which occurs spontaneously and multiple times per minute) and by performing the palpebral reflex [1-15]. When the touch receptors surrounding the eye are activated, the sensory nerves of the trigeminal nerve (ophthalmic branch medially; maxillary branch laterally) are activated which subsequently activates the motor components of the CN VII to complete the blinking movement. As the skin surrounding the palpebral fissure is touched, the animal should close the palpebral fissure rapidly and completely. If the animal partially closes the palpebral fissure, lightly touching the dorsal eyelid margins usually increases the strength of closure.

If the animal does not have a menace response but can close the palpebral fissure during the palpebral reflex and has normal pupils (both size, symmetry, and reactivity) then the animal likely has a central nervous system cause for blindness. In this instance the clinician needs to perform additional evaluations to detect signs of intracranial disease.

Animals with abnormal CN VII function will not be able to close the palpebral fissure completely when this reflex is activated. In these instances, during performance of the menace response, the eye may be retracted in the orbit from the menacing gesture suggesting the afferent (visual) components of this response are normal. Often, when the eye is retracted in the globe, the nictitating membrane will be simultaneously and rapidly elevated over the eye. Similarly, when the palpebral margins are touched, the animal may retract the eye in the orbit but be unable to close the lids. Again, the nictitating membrane will correspondingly be dynamically elevated.

If the animal is unable to close the palpebral fissure during both the menace response and the palpebral reflex test there is likely an abnormality of CN VII or orbicularis oculi muscle function. If the animal closes the palpebral fissure with the menace response but not during the palpebral reflex, this indicates a sensory (afferent) abnormality involving the ophthalmic or maxillary branch of the trigeminal nerve (CN V).

In addition to an inability to close the palpebral fissure, animals with facial nerve disease often have associated abnormalities of position and movement of the lips, ears, and eyelids. The parasympathetic division of the facial nerve is responsible for lacrimal gland secretion, and keratoconjunctivitis sicca (KCS) may also be seen with facial nerve disease [1-15]. Corneal ulceration may also occur due to an inability to protect the eye through eyelid closure.

5. Clinical Assessment 4

Are the pupils normal in appearance, size, shape, and reactivity to degrees of light stimulation? (Figure 4).

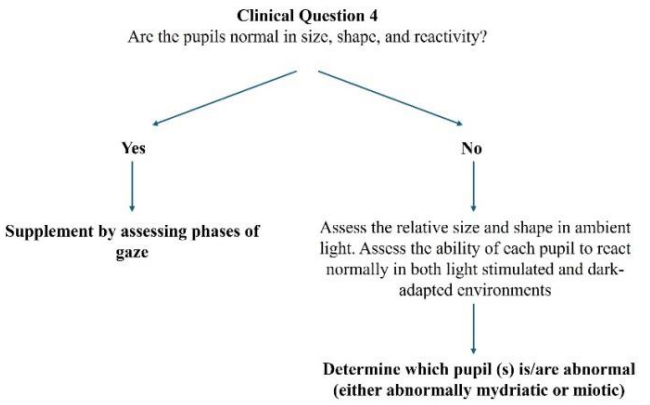


Figure 4. *Clinical Question 4 assesses the functionality of pupil innervation and the iris muscle.*

Following evaluation of visual function, the next assessment is to determine if each pupil is normal size, too large (mydriatic), or too small (miotic) relative to ambient environmental light and how the pupil size changes with differing degrees of light stimulation. Retro-illumination aids in determining the pupillary margins especially when the adjacent iris is darker in color. With bright ambient light environments, the pupils should be smaller (i.e., constricted or miotic) in both eyes equally. In darker ambient light environments, the pupils should be relatively larger (i.e., dilated or mydriatic) in both eyes. In traditional examination room lighting the pupils are commonly mid-range and symmetric in size.

Following assessment of pupillary size and shape, a pupillary light reflex (PLR) is performed to determine of the reactivity of the pupils to light stimulation. For performance of the PLR, a light stimulus is directed independently into each eye. It is important when performing this assessment to use a bright, consistent light source such as a Finoff transilluminator. If a relatively weak light source is used, a poor, slow, or inconsistent pupil response may occur lending confusion to the appropriateness of the reflex.

When light is directed in one eye, both pupils will constrict. The constriction of the light stimulated pupil is referred to as a direct pupillary light response. Constriction of the pupil contralateral to the stimulated eye is referred to as the consensual or indirect pupillary response. Depending upon the intensity of the light stimulation, and the astuteness of the examiner, the pupil directly stimulated by the light may constrict slightly faster and to a slightly greater extent (1-2 mm smaller) compared to the consensually stimulated pupil. This is referred to as a dynamic contraction anisocoria and is normal in animals.

The pupil is innervated by both parasympathetic and sympathetic systems (referred to as dual reciprocal innervation) [1-15, 23]. When stimulated, parasympathetic system activation constricts the pupil; sympathetic system activation dilates the pupil. Parasympathetic nerves to the eye travels with the oculomotor nerve (CN III). Sympathetic innervation is through central pathways in the brain and spinal cord innervating pre-ganglionic and post-ganglionic sympathetic nerves innervating the eye.

In addition to the PLR, when a bright light is directed into an eye, a blink response commonly occurs [15]. This is referred to as the dazzle reflex and is an alternative test to assess light-stimulated reflex activity and is especially useful when corneal/anterior chamber disease is present. This is a subcortically mediated reflex as is the PLR.

When assessing pupils, it is important to determine that the iris muscle is normal and there is no restriction to its movement such as a synechia (Clinical Question 1). Mechanical abnormalities of the iris can result in abnormalities of pupil size, shape, and movement [15]. Iris atrophy, for example, is a common cause for unequal pupil size, relatively dilated pupils, or abnormally shaped pupils (dyscoria) especially in older dogs.

Unequal pupil size is known as *anisocoria*. If anisocoria is present, the subsequent step is to determine which pupil (s) is/are abnormal. That is, is one pupil relatively too large and the other normal; is one pupil relatively too small and the other normal; or are both pupils abnormal (i.e., abnormally mydriatic or abnormally miotic)? In some situations when one pupil is miotic the opposite eye will adapt and the pupil slightly dilate. In other instances, when one pupil is mydriatic the opposite eye will adapt with pupillary constriction. Finally, with some visual pathway deficits the ipsilateral pupil may slightly dilate resulting in a subtle anisocoria.

A miotic pupil in traditional examination room ambient light with a normal iris muscle that constricts with light stimulation and does not dilate in a darker environment likely has sympathetic denervation [1-15, 24-27]. Other clues to sympathetic denervation (referred to as Horner's syndrome) include enophthalmos, elevation of the nictitating membrane, and ptosis.

A mydriatic pupil in traditional examination room ambient light with a normal iris muscle that does not constrict with light stimulation and remains dilated in a darker environment (with previously determined normal vision) likely has parasympathetic denervation. Stimulation through the parasympathetic division of CN III results in pupillary constriction during performance of the PLR. An abnormality of the autonomic functionality of CN III will result in a dilated pupil referred to as internal ophthalmoplegia. Animals may have abnormalities of one or both (autonomic and motor) functions of CN III.

As parasympathetic nerves to the pupil travel with CN III (oculomotor nerve), next assess the animal for motor functionality of CN III by observing for a resting strabismus or failure of medial gaze. In a forward gaze position, abnormalities of oculomotor nerve (CN III) can produce a lateral and ventral strabismus (ventrolateral). Ptosis ("droopy" upper eyelid) may result due to paralysis of the levator palpebrae muscle.

Once it is determined the type of pupillary abnormality, an appropriate differential diagnosis can be formulated. This list includes commonly noted abnormalities as well as uncommon entities such as Adie's pupil (idiopathic abnormal parasympathetic pupillary innervation or iris muscle dysfunction) and

Pourfour du Petit syndrome (sympathetic system hyperactivity) [15, 28-30].

6. Clinical Assessment 5

Are the eyes positioned normally in the orbit? (Figure 5).

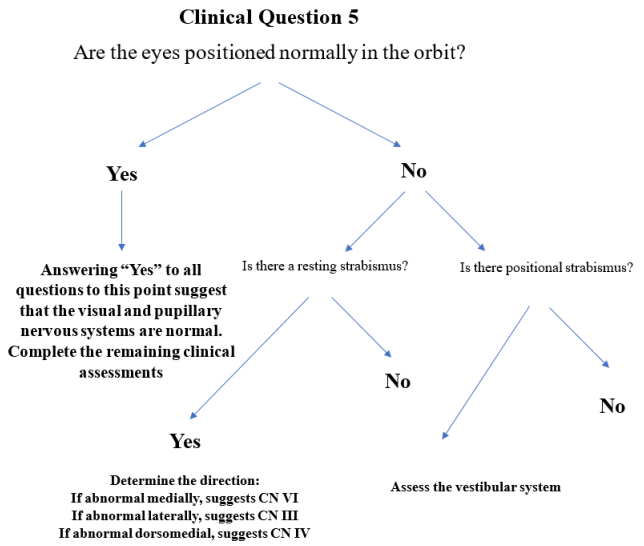


Figure 5. *Clinical Question 5 assesses ocular position based on extraocular muscle functionality.*

Based on innervation patterns of the extraocular muscles the eye can be directed in multiple orientations. When activated, for example, the motor activities of CN III move the eye primarily in a medial (sometimes dorsal and ventral) direction and elevates the palpebral fissure based on its innervation of associated extraocular muscles. The Trochlear nerve (Cranial Nerve IV) rotates the eye medially [1-15]. The Abducens nerve (Cranial Nerve VI) moves the eye in a lateral direction and retracts the eye in the orbit. These latter functionalities are not always as clinically obvious unless they are directly activated during gaze testing.

When these extraocular muscles have abnormal innervation, the eye may deviate from a normal position. Abnormal eye position is termed *strabismus.* A strabismus may be present in the normal forward gaze position (i.e., "at rest" or "resting") or occur dynamically with changing head position (i.e., a "positional strabismus") [1-15]. In a forward gaze position, abnormalities of oculomotor nerve generally produce a ventrolateral strabismus (ventrolateral). *Ptosis* (droopy upper eyelid) may result due to paralysis of the levator palpebrae muscle.

Abnormalities of the trochlear nucleus or nerve should cause a contralateral dorsomedial strabismus however this is not always evident in all species [15]. In species with a round pupil, no strabismus is seen externally from the examiner's perspective. Ophthalmologic examination of the retina with a direct ophthalmoscope, however, reveals the superior retinal vein to be deviated laterally. If indirect visualization is used, the superior retinal vein will be directed medially. As cats have a vertical pupil, the dorsal aspect of the vertical pupil may be deviated laterally with such abnormality. Isolated abnormalities of this nerve, however, are extremely rare in small animals. Disease of CN VI can result in a medial strabismus. The eyeball cannot abduct fully and may not be retractable within the orbit. A medial strabismus has also been recognized with central thalamic abnormalities in dogs [31]. In general, isolated abnormalities of CN III, IV, and VI are clinically rare.

Siamese and other breeds of cats can have strabismus (esotropia) not due to an abnormality of innervation of the extraocular nerves but rather to congenitally abnormal visual projection pathways [15]. This eye position is an adaptation of eye position to abnormal visual information projection pathways. Siamese and similarly affected cats have increased numbers of decussating or crossing visual projections from the retina to the thalamus. The eye is deviated to result in improved visual acuity. There may be a concurrent "pendular" nystagmus or ocular tremor with the eyes moving rapidly from side to side usually when the animal's head is stationary.

A *positional strabismus* is most easily evoked by extending the head dorsally so that the gaze is directed dorsally toward the ceiling [1-15]. As the head is moved to this extended dorsal position, the vestibular receptors in the ear become activated. Vestibular information is projected cranially through the medial longitudinal fasciculus in the brain stem to influence the functions of the cranial nerves (III, IV, and VI) to the extraocular muscles. These nerves are either excited or inhibited to move the eyes to the new direction of gaze (i.e., dorsally).

Normally as the dog's head is extended dorsally, the eyes will remain positioned in the orbit so that the gaze is directed forward (or in this position, dorsally). If one or both eyes' position deviates from this direction, this is considered a positional strabismus. Most commonly, the abnormal eye(s) will deviate comparatively ventrally and is most easily appreciated by observing the more prominent dorsal sclera of the affected eye. As there is no strabismus with a normal forward gaze position, a positional strabismus implies that the vestibular input to CNs III, IV and VI is abnormal. This clinical abnormality is usually associated with an ipsilateral vestibular abnormality, most commonly in the peripheral, but sometimes the central (brain stem), vestibular system.

7. Clinical Assessment 6

Do the eyes move normally? (Figure 6).

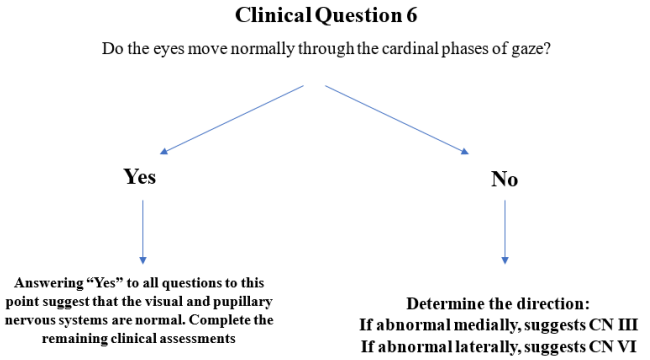


Figure 6. *Clinical Question 6 assesses ocular motor functionality.*

As the motor function component of CN III is responsible for eye movement, primarily in a medial direction, poor ability to move the eye medially may be seen with a deficit of this nerve. For this assessment, the animal's head can be held in a stationary position while simultaneously creating some stimulus for the animal to look in various directions, not allowing it to move the head, but only the eyes. For example, the animal's head can be stabilized by an assistant, and a sound stimulus (speaking or clapping of the hands) can be created slightly outside the limits of the animal's lateral visual field of the contralateral eye to be tested. Most often this kind of stimulus will result in animals directing their gaze in the direction of the stimulus. An animal with CN III motor dysfunction may not be able to move the affected eye in a medial direction due to loss of innervation of the medial rectus muscle. This is referred to as an *external ophthalmoplegia*. If this occurs with concurrent parasympathetic dysfunction (that is, a dilated pupil that does not constrict when stimulated) this is *complete ophthalmoplegia*.

In other instances, there may not be detectable strabismus during the normal forward gaze, however, during active gaze the animal may not move the eye in a medial direction. If either of these situations is present and the animal has a dilated, poorly responsive (to light stimulation) pupil, this indicates that both the motor and the parasympathetic components of cranial nerve III are affected.

8. Clinical Assessment 7

Is there a nystagmus or other spontaneous ocular movement abnormality? (Figure 7).

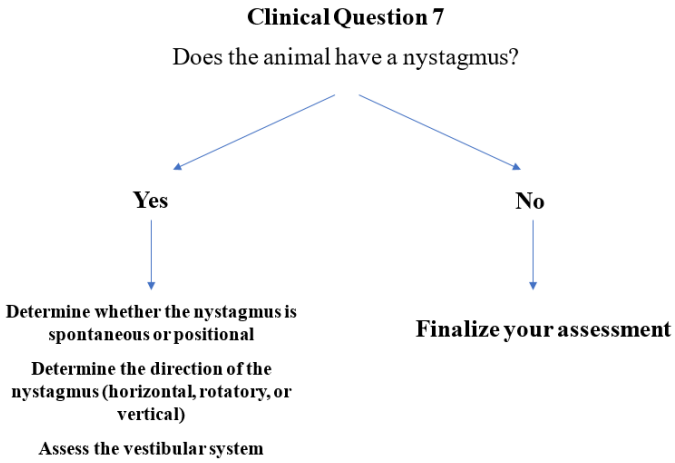


Figure 7. *Clinical Question 7 assesses abnormal movement of the eyes.*

Spontaneous *nystagmus*, a characteristic eye movement with a quick and slow phase, is commonly associated with vestibular dysfunction. A nystagmus-type movement can be induced normally (oculovestibular response) by turning the head from side to side. The fast phase of eye movement is in the direction that the head is moved. This slow drift and quick reset during sideways movement of the head is normal. In animals with bilateral peripheral vestibular disease, the oculovestibular response is absent as the peripheral receptors are not activated during head movements. When the vestibular system is dysfunctional, the eyes tend to spontaneously drift in the direction of the abnormality (slow phase), and, through a brain stem reset mechanism, the eyes are quickly returned to their initial location (fast phase).

Abnormal nystagmus can occur spontaneously (present at rest) or with abnormal head positions (i.e., *positional nystagmus*). This latter nystagmus is only present when the head is placed in an abnormal orientation by the examiner. A positional nystagmus is most easily elicited by placing the animal upside down on its dorsum. If an abnormal nystagmus is identified, the direction of the nystagmus is described in relation to the horizontal axis through the palpebral fissure. With a horizontal nystagmus, eye movement is in the direction of this axis. A vertical nystagmus is in the direction perpendicular to this axis. With a rotatory nystagmus, the eye moves around the parasagittal axis in either a clockwise or counterclockwise direction.

The presence of an abnormal nystagmus should prompt further evaluations primarily of the vestibular system. There are other types of nystagmus such as a conversion retraction nystagmus and upper eye retraction (Collier's sign) that can occur with abnormalities if the dorsal midbrain (Parinaud's syndrome) [32-35].

Common neuroanatomical diagnoses in animals with neurological abnormalities of vision, pupillary reactivity, or ocular movement

While each individual case may have a unique constellation of clinical features, some clinical presentations are more commonly encountered in clinical practice. These common clinical scenarios are described in Table 1. These descriptions include the constellation of clinical signs and the associated neuroanatomical diagnosis.

Table 1. *Common clinical presentations.*

Clinical Scenario	Neuroanatomical Localization
An animal has an absent vision, an absent menace response, a normal palpebral reflex, and normal pupillary light reflex.	In this case, the animal likely has a central nervous system cause of the menace abnormality in the intracranial rostrotentorial (forebrain) nervous system.
An animal has an absent menace response with a decreased or absent palpebral reflex and normal pupillary light reflex. The animal retracts the globe, and the nictitating membrane is elevated during the menacing gesture and when the periocular skin or hair is touched by the examiner.	In this case, the animal likely has an abnormality of the ipsilateral facial nerve (CN VII). The retraction of the stimulated eye suggests visual recognition of the stimulus but without the ability to close the palpebral fissure.
An animal has a resting anisocoria. In normal ambient light one pupil is normal (or miotic) and the other pupil is larger than normal (mydriatic). The menace response and palpebral reflex are normal. When performing the pupillary light reflex, light directed in the normal sized pupil (i.e., the direct response) results in that pupil constricting however the opposite pupil (i.e., the indirect response) does not change in size. When light is directed into the relatively larger pupil no change in that pupil occurs while the opposite pupil (i.e., the relatively normal-sized pupil) constricts.	After determining that the iris muscle is normal, this constellation of clinical signs indicates an abnormality of the parasympathetic component of the oculomotor nerve (CN III). Abnormalities of ocular position (strabismus) and/or failure to move the eye medially during the cardinal phases of gaze are additional supporting evidence of an oculomotor nerve abnormality.
An animal has bilaterally dilated pupils. The menace response and palpebral reflex are normal. When performing the pupillary light reflex, light directed in either eye does result in pupillary constriction. The animal blinks, however, when a bright light is directed into either eye.	This constellation of clinical signs indicates either a bilateral iris muscle abnormality (such as iris atrophy) or a bilateral abnormality of the parasympathetic component of the oculomotor nerve (CN III).
An animal has bilaterally dilated pupils. The menace response is absent in both eyes while the palpebral reflex in each eye is normal. When performing the pupillary light reflex, light directed in either eye does not result in pupillary constriction. The animal also does not blink when a bright light is directed into either eye.	This constellation of clinical signs could indicate either bilateral ocular disease including retinal disease, bilateral optic nerve disease (such as optic neuritis) or an optic chiasm abnormality.
An animal has resting anisocoria. In normal ambient light one pupil is normal and the other pupil is smaller than normal (miotic). The menace response and palpebral reflex are normal in both eyes. When performing the pupillary light reflex, light directed in the normal sized pupil (direct response) results in that pupil constricting and the opposite side (relatively smaller) pupil also constricting. When light is directed in the relatively smaller pupil, both the smaller and the normal pupil constrict. In a dark environment, the smaller pupil does not dilate appropriately while the relatively normal size pupil does dilate.	After determining that the iris muscle is normal, this constellation of clinical signs indicates an abnormality of the sympathetic innervation in the relatively miotic pupil. Additional signs of sympathetic denervation (e.g., elevated nictitating membrane, ptosis, and exophthalmos) support this conclusion.

9. Conclusion

While vision, pupil, and ocular movement abnormalities can be challenging to localize, using an iterative questioning paradigm for clinical evaluation focuses the clinician's attention in determining where the abnormalities are occurring within the nervous system in affected animals. The questions presented in this review provide the necessary prompts

throughout the clinical examination process for completeness of the neuro-ophthalmological assessment. By answering these posed questions, a thorough assessment can be achieved resulting in an accurate neuroanatomical diagnosis. Once this neuroanatomical diagnosis is rendered, an appropriate differential diagnosis list can be created followed by justified diagnostic testing to ultimately determine the cause of the animal's impairment.

Abbreviations

CN	Cranial Nerve
PLR	Pupillary Light Reflex

Author Contributions

Rodney Bagley: Conceptualization, Data curation, Formal Analysis, Investigation, Methodology, Project administration, Software, Supervision, Validation, Visualization, Writing – original draft, Writing – review & editing

Conflicts of Interest

The author declares no conflicts of interest.

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