Peracute spinal cord injury (SCI) will often produce examination findings that confound our conventional rules of neurolocalization. The Schiff–Sherrington posture (rigid extension of the thoracic limbs) occurs with damage to border cells in the thoracolumbar spinal cord segments that would normally ascend to inhibit thoracic limb extensor tone. This posture may be misinterpreted as indicating a cervical spinal lesion. Spinal shock is the loss of muscle tone and spinal reflexes caudal to the site of peracute SCI, even though the reflex arcs are physically intact. The initial hyporeflexia may be interpreted as a 'lower motor neuron sign', when the lesion is in fact of the upper motor neurons innervating that limb. In dogs and cats this phenomenon is short lived, with reflexes returning within hours (up to 2 hours for patellar reflexes and 12 hours for flexor reflexes). In most cases of vestibular syndrome the head tilt is towards the side of the lesion, however if the flocculonodular lobe of the cerebellum or the caudal cerebellar peduncle is affected then the head tilt will be towards the contralateral side, so-called paradoxical vestibular syndrome.

Non-neurological problems may affect function in a way that looks like a neurological abnormality or deficit on examination. Animals with bilateral lameness due to musculoskeletal or orthopaedic disease, such as bilateral cruciate ligament rupture, bilateral coxofemoral osteoarthritis, polyarthritis or iliopsoas muscle injury can appear 'ataxic' as the animal sways, shifting weight from lame limb to lame limb. Reluctance to bear weight on bilaterally painful limbs may also be interpreted as weakness. A musculoskeletal examination in combination with proprioceptive paw positioning while the patient is well supported is usually a reliable way to differentiate between neurological and bilateral orthopaedic conditions. Toxins or metabolic derangements may cause encephalopathic signs – signs produced tend to be diffuse/symmetrical in nature and often wax and wane. Conditions of the eye may cause apparent neuro-ophthalmological abnormalities. Mydriasis or a poorly responsive pupil may be caused by iris atrophy, glaucoma or cycloplegic drugs. Miosis may be caused by uveitis or corneal pain due to an ulcer.

KEY LEARNING OBJECTIVES

- Understand that true neurological abnormalities may be 'normal' for an individual and may be irrelevant to the clinical problem
- Recognize that certain neurological syndromes are exceptions to our conventional rules of neurolocalization and may confuse efforts to neurolocalize if not recognized
- Understand that many non-neurological problems may mimic neurological abnormalities on examination

MULTIPLE CHOICE QUESTIONS

1. Which of the following would not cause a mydriatic pupil?
 - (A) An oculomotor nerve lesion
 - (B) Glaucoma
 - (C) A corneal ulcer
 - (D) Iris atrophy
2. You examine a 4-year-old blue-eyed Himalayan cat as part of routine check-up and vaccination. It is otherwise healthy, but you notice fine side-to-side oscillatory movements of both its eyes. Which is the most likely cause?
 - (A) Pendular nystagmus due to congenital misrouting of visual pathways
 - (B) Otitis media/interna causing vestibular syndrome
 - (C) A thalamic infarct
 - (D) Cerebellar abiotrophy
3. You are examining a 6-year-old Staffordshire Bull Terrier that has just been hit by a car. It is lying in lateral recumbency. It has no observable voluntary movement in its pelvic limbs. Its thoracic limbs are rigidly extended compared to the pelvic limbs, but some voluntary movement is observed. Which is the most likely neurolocalization?
 - (A) Cerebellum
 - (B) C1–C5 spinal cord
 - (C) C6–T2 spinal cord
 - (D) T3–L3 spinal cord

Odd episodes: when is a seizure not a seizure?

Tom Cardy

Dogs and cats presenting with abnormal episodes provide some of the most challenging and frustrating cases for veterinary surgeons of all abilities. Veterinary surgeons are frequently presented with owners describing vague and often transient clinical signs that make it challenging to define the presenting problem precisely and construct a list of differential diagnoses. In veterinary

neurology these episodes can be defined as 'paroxysmal episodes' with a defined start, defined end and relatively short duration. There are relatively few causes of paroxysmal episodes in dogs and cats including: seizures, syncope, vestibular syndrome, paroxysmal dyskinesia (movement disorders), narcolepsy/cataplexy, neuromuscular disease and idiopathic head tremors.

Accurately classifying the paroxysmal episode is dependent on taking a thorough and systematic history that includes: a description of the event, the clinical status between episodes, asking if there is impairment or loss of consciousness, presence or absence of autonomic signs, description of muscle tone and lateralization of presenting clinical signs. Where possible videos of the events should be reviewed with the owner.

Delivered by BSAVA to:

Guest (guest)

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In this lecture we will use case examples and videos to review common paroxysmal episode presentations. Particular attention will be given to seizures, syncope, vestibular syndrome and paroxysmal dyskinesia. We will investigate the diagnostic approach to these conditions, including genetic testing for certain breed-associated conditions. The cases will review the appropriate use of pharmacological or dietary treatments and highlight how the provision of accurate information to owners is critical in ensuring the optimal management of these varied conditions.

KEY LEARNING OBJECTIVES

- Describe approaches to dogs and cats presenting with 'paroxysmal episodes' and understand how adopting a problem-based clinical reasoning approach can improve diagnosis of seizures
- Understand how factors from the animal's signalment and presenting clinical signs can aid in classifying paroxysmal episodes accurately
- Be familiar with the optimal diagnostic approaches and management strategies for animals with commonly presenting paroxysmal episodes

MULTIPLE CHOICE QUESTIONS

1. How can syncope best be described?
 - (A) Dystonia and inability to stand/walk in conscious individuals
 - (B) Severe ataxia, with head tilt, nystagmus and positional strabismus
 - (C) Rapid and reversible loss of consciousness and muscle tone due to insufficient perfusion of oxygen to the brain
 - (D) Severe alteration in consciousness, motor activity, autonomic function, sensation, cognition due to hypersynchronous cortical electrical activity
2. Which genetic mutation has been associated with episodic falling syndrome in Cavalier King Charles Spaniels?
 - (A) Epm2B
 - (B) KCNJ10
 - (C) LGI2
 - (D) BCAN
3. Idiopathic head tremor in English Bulldogs will spontaneously resolve in what percentage of cases?
 - (A) 50%
 - (B) 25%
 - (C) 100%
 - (D) 0%

Emergency seizure management: dos and don'ts

Tom Cardy

Status epilepticus (SE) is a relatively common emergency in small animal veterinary medicine that requires immediate and aggressive treatment. If untreated SE can result in life-threatening complications involving the brain and systemic organs. SE often requires rapid and comprehensive patient assessment, treatment with multiple anticonvulsant agents, and intensive supportive care. The lecture describes the aetiopathogenesis and diagnosis of SE along with treatment algorithms to help achieve rapid and effective cessation of the seizures and a successful recovery.

SE is defined as continuous seizure activity, greater than 5 minutes in duration, or more than one discrete seizure without regaining a normal level of consciousness between seizures. Potential causes of SE include idiopathic epilepsy, structural epilepsy or reactive seizures secondary to metabolic disturbances or toxin ingestion. The initial 30 minutes of SE is referred to as the 'compensatory phase' as the brain increases cerebral blood flow to compensate for increased oxygen and glucose demands. Body temperature can elevate markedly due to seizure activity and sympathetic activation, and lactic acidosis, hypoglycaemia and electrolyte imbalances can develop secondary to increased anaerobic metabolism.

After approximately 30 minutes the brain's regulatory mechanisms begin to fail and the patient enters a 'decompensatory phase' with systemic hypotension, decreased cerebral blood flow, disruption of the blood-brain barrier, cerebral oedema and ultimately neuronal cell death. After approximately 60 minutes severe and potentially fatal systemic complications can occur with cardiac arrhythmias, increased intracranial pressure and multiple organ failure. Without prompt and aggressive treatment death can occur.

The goals in the management of SE are fourfold: stop seizures, systemically stabilize the patient and protect the brain, treat the underlying causes and prevent further seizures. Initial investigations should be the same as any acute neurological case with a thorough history, physical examination and neurological examination. All patients should ideally have a minimum database performed with particular attention to electrolytes and glucose levels. First-line treatments typically include one or more of: benzodiazepines, levetiracetam, phenobarbital or intravenous anaesthesia. Care must be taken to ensure the patient is systemically supported with a focus on airways, breathing and circulation. The level of intervention is patient dependent but can often be intense and hands-on. With an appropriate response to initial management and stabilization, the outcomes in the management of SE patients can be good.

KEY LEARNING OBJECTIVES

- Define status epilepticus and understand the systemic and neurophysiology that results in clinical signs