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

- Understand the aetiology and epidemiology of status epilepticus in dogs and cats
- Learn how to stabilize patients with status epilepticus and the range of first-line treatments available to stop seizures effectively

MULTIPLE CHOICE QUESTIONS

1. How can status epilepticus be defined?

- (A) Continuous seizure activity, greater than 5 minutes in duration, or more than one discrete seizure without regaining a normal level of consciousness between seizures
- (B) Two or more seizures in a 24-hour period
- (C) Continuous seizure activity, greater than 2 minutes in duration, or more than one discrete seizure without regaining a normal level of consciousness between seizures
- (D) Hypersynchronous neuronal electrical activity in the cerebral cortex

2. Which of the following can occur during the compensatory response to status epilepticus?

- (A) Decreased cerebral blood flow, hypotension and hypoglycaemia
- (B) Increased cerebral blood flow, systemic hypertension and tachycardia
- (C) Increased cerebral blood flow, systemic hypotension and tachycardia
- (D) Decreased cerebral blood flow, hypertension and hyperglycaemia

3. Which of the following are key drugs used in the emergency stabilization of status epilepticus?

- (A) Benzodiazepines, levetiracetam, potassium bromide
- (B) Benzodiazepines, levetiracetam, phenobarbital
- (C) Ketamine, levetiracetam, phenobarbital
- (D) Ketamine, levetiracetam, potassium bromide

Spinal emergencies: when to refer and when not to

Patrick Kenny

Referral from general to specialist practice is generally required for advanced imaging and surgical treatments. The decision to refer is going to be based on the initial presumptive diagnosis, the tentative prognosis based on the presumptive diagnosis and severity of neurological signs, as well as owner factors (financial limitations, geographic location and ability to transport to a referral centre).

Patients that have been witnessed to have an external trauma are usually not a diagnostic challenge. If no trauma was observed or evidenced on examination, other causes of acute myelopathy (such as intervertebral disc herniation, ischaemic myelopathy, meningomyelitis or spinal neoplasia) should be considered. A combination of patient data (signalment), historical data (onset and progression of clinical signs) and examination data (neurolocalization, degree of symmetry of neurological signs and the presence or absence of spinal pain) are helpful in formulating and ranking a list of differential diagnoses. Patients that have suffered an external trauma, or if there is a possibility or suspicion of a spinal fracture or luxation, should be immobilized on a spinal board immediately.

Prognosis after an acute spinal cord injury is going to be determined by the cause and the severity of

neurological signs prior to institution of (appropriate) therapy. For most causes of acute spinal cord injury (spinal fracture/location, intervertebral disc herniation, ischaemic myelopathy), the presence or absence of deep pain perception (DPP) is the most important predictor of recovery. As such it must be properly tested — using an unequivocally noxious stimulus (forceps on periosteum) and looking for a behavioural response that this has been perceived by the animal as pain. It should be tested prior to administering any analgesic medications that may blunt this response and result in the clinician giving the patient an inappropriately poor prognosis.

The majority of dogs with intact DPP will return to ambulation if their acute spinal cord injury is non-compressive or is rapidly surgically decompressed. Approximately 50% of DPP-negative dogs with intervertebral disc herniation that have surgery within the first 24 hours will return to ambulation, with prognosis diminishing rapidly as time progresses. Very few patients that were DPP negative after acute spinal fracture luxation have reportedly regained ambulation, urinary and faecal continence even after spinal stabilization. Owners should be informed of these prognoses before transport.

KEY LEARNING OBJECTIVES

- Understand that any rapidly progressing or non-ambulatory patient should be considered an emergency
- Recognize that prognosis may be time dependent so make your plan early — don't wait to offer referral if necessary
- Understand that patients with polytrauma and/or systemic abnormalities such as shock may need stabilization before transport

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