Proceedings of the 11th International Congress of the World Equine Veterinary Association

24 – 27 September 2009
Guarujá, SP, Brazil

Next Meeting:
Nov. 2 -6, 2011 - Hyderabad, India

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Emerging Equine Neurologic Syndromes:  
What Nature has taught us recently about  
Neurophysiology and Neuroanatomy

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Over the last 5 years or so we have learnt much about existing and new disorders in horses from documentation of careful clinical observations and interventions and from painstaking pathologic studies with especial emphasis on clinicopathologic correlates. Some of these disorders have been discussed elsewhere in this conference and this paper will highlight a few of these disorders through which we have added to our understanding of anatomy, physiology and clinicopathologic correlates – the building-blocks for advancing equine neurology.

Acquired cervical torticollis associated with *Parelaphostrongylus tenuis*

Several *Metastrongylid* nematodes are important causes of neurologic disease in wild and domestic ruminants in Europe, Asia, New Zealand and North America and likely occur wherever native Eurasian and American cervid populations exist worldwide and where suitable terrestrial molluscs can act as intermediate hosts. There are 2 genera of these so-called extrapulmonary lungworms affecting cervids - *Elaphostrongylus* and *Parelaphostrongylus* - that pass through the host CNS as part of their life cycle. Although they can cause neurologic disease in their primary hosts they tend to produce prominent neurologic infections, usually non-patent, in abnormal and aberrant hosts. One of the more common of these neurologic diseases involves *Parelaphostrongylus tenuis* that produces traumatic and inflammatory myeloencephalomeningitis in domestic ruminants, and recently in horses, showing syndromes relating to asymmetric spinal cord and brain stem involvement. The amazingly selective meanderings of these migratory nematode parasites are exemplified by the plight of *Parelaphostrongylus tenuis* in some horses that have been elegantly documented. This worm has caused an acute onset of a lateral deviation of the cervical vertebrae, or scoliosis, which remained static, and was the result of an exquisitely selective, continuous, cervicothoracic, inflammatory dorsal grey column lesion. Cutaneous hypalgesia to analgesia of the neck and/or thorax and mild unilateral ataxia and paresis but with no evidence of denervation atrophy in the epaxial muscles was observed in all affected horses. The scoliosis was clearly argued to be due to loss of afferent cervical proprioceptive inputs because of the dorsal gray column lesions with some white matter involvement accounting for ataxia and weakness [Figure 1].

![Figure 1. A continuous lesion in the dorsal gray column in the neck (blue star) causes a segmental interruption of the postural regulation of the muscle tone in epaxial muscles, resulting in scoliosis of the neck. In addition, it interrupts the proprioceptive and general somatic afferent input to higher centers resulting in ataxia and hypalgesia. If adjacent white matter is affected, ataxia in pelvic limbs may result.](image)

A moderate aseptic CSF pleocytosis with elevated protein and sometimes xanthochromia typically is detected in *Metastrongylid* nematode myelitis and encephalitis cases. Serologic immunotesting, such as western blot using DNA fragment antigens from *Parelaphostrongylus tenuis*, will be very useful...
in epidemiologic and clinical settings in future. These nematodes appear to be sensitive to various anthelmintics such as fenbendazole and ivermectin and such therapy has been successful when the cases have been treated soon after onset of clinical signs.

**Vestibular syndromes following cervical nerve root lesions**

A rather forgotten component to the vestibular system is proprioceptive input from the cranial cervical vertebrae, ligaments and muscles. Special proprioceptive inputs from the cranial cervical vertebral ligaments and muscles pass via at least the C1-3 dorsal spinal nerve roots to ascend the spinal cord via the spinovestibular tract [Figure 2] to the caudal vestibular nuclei. These nuclei receive no other afferent inputs. Lesions involving these cranial cervical nerves or the vestibulospinal input to the vestibular apparatus can result in signs of vestibular disease. This certainly can be seen with symmetric lesions of the dorsal nerve roots of C1-3 when loss of balance, eye deviation and head tilt have been seen.

![Figure 2: The spinovestibular pathway](image)

Cervical vertebral articular ultrasound and articular injection

Confirmation that apparent neck stiffness and pain, or thoracic limb lameness, is emanating from specific arthritic vertebral articulations requires radiographic and possibly scintigraphic evidence of active arthritis and positive relief being achieved from intra and peri articular injection of local anesthetic agent. The appropriate anatomy and ultrasonographically-guided method for achieving this is now elegantly described and should be applauded.

Cervical (peri) articular injections have become a favored diagnostic and therapeutic tool at least in north America. Empiric injection of enlarged articular processes without substantive evidence that they are causing a clinical problem of neck pain alone, neck pain with ataxia and ataxia alone is to be discredited. This author has seen numerous patients with these syndromes where trauma to the neck has been suspected or not. Detailed and repeated cervical radiographs have failed to detect a cervical fracture but ultimately a serious fracture has been found at post mortem examination. On occasion, such injections and aggressive physiotherapy of the neck have been undertaken in such patients and under these circumstances both these treatment modalities must be deprecated.

Electrodiagnostics: magnetic motor evoked potentials and quantitative electromyography

Certainly the non-invasive and reasonably innocuous testing for magnetic motor evoked potentials now has received attention in large animal neurology and it appears to be a very sensitive and quite specific electrophysiologic test for disruption of somatic motor pathways in disease states. This and the additional use of more elaborate but error prone quantitative EMG investigations should allow more accurate identification of the presence and location of conduction blocks, and thus
functional lesions, in neurologic disease states such as wobblers and unusual hind limb gait abnormalities.

**Scandinavian knuckling horses**

A detailed synopsis of five outbreaks of a hind limb knuckling syndrome in horses has been presented and discussed 26 in which a total of 24 cases occurring in an at-risk population of 75 animals were affected by an interesting syndrome. Detailed clinical, video analysis, paraclinical and pathological investigations were undertaken on numerous affected cases, with only 3 surviving, one of which had recovered. Another 75 cases of idiopathic knuckling in horses in Norway have been described with this syndrome with no cause being determined but poor feed in the form of low quality baled silage being a frequent finding 27. Further outbreaks have been discussed on electronic listserves 28.

The clinical syndrome was one of varying degrees of bilateral sciatic nerve involvement, some horses showing more prominently signs of peroneal neuropathy and others showing more prominently signs of tibial neuropathy 27. Occasional cases had extensor weakness also indicating femoral nerve involvement. In all cases silage was fed along with poor quality hay. Several detailed post-mortem examinations have been undertaken and in many there was evidence of mild peripheral wallerian-like neuronal fiber degeneration on routine light microscopic sections. Some milder cases improve but it is not certain whether this recovery is complete to allow full performance. Indeed, the clinical syndrome, recovery of function and ultrastructural morphological lesions would be more indicative of a primary demyelinating lesion being operative 29. Peripheral neurotoxins of plant or non-biologic origin would be the most likely cause of these crippling syndromes 27, 29, 30.

**Idiopathic neuropathy with knuckling**

Japanese workers 31, 32 have studied three cases of a neuropathy present in a superficially described syndrome of growing horses referred to as knuckling. These patients sometimes dragged the toes of their forelimbs. The workers described widespread peripheral nerve wallerian degeneration, most prominent distally. The formation of bands of Büngner and regenerative axonal sprouts do make this pathological process consistent with a diffuse distal axonopathy. Neurogenic muscle atrophy with some regeneration was seen with fiber type grouping, particularly in distal muscles. The comment was made that "laryngeal paralysis was not observed in the present cases". Unfortunately, the basis for this statement was not given so that clinical evidence for the presence of a polyneuropathy was lacking.

The important issue here is that clinically, this appears to be the not uncommon syndrome of prominent upright posture with tendency to flex the carpi and fetlocks that occurs during weight support as an acquired syndrome in weanlings and yearlings. The presence or absence of evidence for a neuropathy causing this relatively common syndrome needs further investigation.

**Equine Motor Neuron Disease [EMND]**

Acquired equine motor neuron disease [EMND] is a fascinating neuromuscular disorder of horses that does not appear to have existed prior to 1982 and was first described by the late John Cummings and co-workers from Cornell University in 1990 33. Hundreds if not thousands of horses now have been definitively diagnosed with EMND in North America and from around the world.

The clinical syndrome expressed depends on the stage of the disease 34-36. Weight loss in the face a good to increased appetite, increased recumbency and slight resting muscle tremors are consistent findings in early cases. The weight loss often precedes the onset of trembling by several weeks 7. Many animals display an extended tail head position the appears to be due to selective involvement of dorsal sacrococcygeal muscles that are postural muscles containing a high proportion of Type 1 fibers. Atrophy is followed by fibrous contracture leading to an elevated tail position. A short-strided gait is commonly seen that can show a rapid placement of the foot at the end of the protraction phase akin to that seen with fibrotic myopathy. This also may well be due to fibrous contracture of affected muscles that in this case are caudal thigh muscles involved in stifle flexion and/or hip extension. Ophthalmic examination reveals
varying degrees of a mosaic pattern with dark brown to yellow brown pigment deposited in the tapetal zone, coupled with a horizontal band of pigment at the junction of the tapetum and nontapetum [lipofuscinosis] \(^{37,38}\). A clinical truism for the syndrome is that **affected horses move better than they stand**. Relative occurrence of the more frequently occurring signs of EMND are given in Table 1. Overall study of this disease has given us a better understanding of syndromes of diffuse weakness in horses and particularly weakness involving Type 1, postural, slow twitch muscles.

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Occurrence %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle loss</td>
<td>100</td>
</tr>
<tr>
<td>Muscle trembling</td>
<td>95</td>
</tr>
<tr>
<td>Increased lying down</td>
<td>93</td>
</tr>
<tr>
<td>Excellent appetite</td>
<td>90</td>
</tr>
<tr>
<td>Shifting weight</td>
<td>90</td>
</tr>
<tr>
<td>Extended tail head</td>
<td>76</td>
</tr>
<tr>
<td>Lowered head posture</td>
<td>62</td>
</tr>
<tr>
<td>Retinal lesions</td>
<td>18</td>
</tr>
</tbody>
</table>

Definitive diagnosis is dependent on the postmortem demonstration of degeneration and loss of cell bodies in the ventral horn of the spinal cord and in motor nuclei in the brain stem with the exception of those supplying cranial nerves III, IV and VI. Cell bodies swell, lose the Nissl substance, become chromatolytic and have accumulations of neurofilaments in perikarya and proximal axons before dying and being removed. Concomitantly there is degeneration of axons in the ventral roots and peripheral nerves and neurogenic muscle atrophy that predominantly affects type 1 fibers \(^{33,40-42}\). Of clinical interest is that approximately 30% of lower motor neurons need to be affected in the spinal cord before there are clinical signs evident \(^{43}\).

In experimental cases and in naturally-occurring cases of EMND in North America and in Europe, vitamin E deficiency has been the only consistent nutritional or toxic-related abnormality determined \(^{34}\). Upon diagnosis of EMND it is reasonable to supplement with vitamin E and added intakes of 4,000-6,000IU per horse per day have been recommended. Green forages are by far the major and best source of vitamin E and until recently, many grain-based concentrates had less vitamin E than the NRC recommendations for horses of 50 IU/kg. Horse feeds with increased amounts of vitamin E have become available and increased owner awareness of the possibility of vitamin E deficiency and increased use of vitamin E supplements may partially explain the apparent decline in the frequency of reported cases of EMND. Further studies are necessary to explain the development of hypovitaminosis E and EMND in horses that have adequate access to pasture. Genetic factors for alterations in amount or activity of the alpha-tocopherol transfer protein, such as the 744 del A mutation of the alpha-TTP gene operative in humans with some forms of hypovitaminosis E \(^{44,45}\), will need exploration in this regard.

**Equine Polysaccharide Storage Myopathy [EPSM]**

Equine polysaccharide storage myopathy [EPSM] is an autosomal recessive disorder in Quarter horse and related breeds and can result in rather exceptional susceptibility to recurrent exertional rhabdomyolysis \(^{46-51}\). Recently, there has been an association of the disease with a particular microsatellite marker \(^{52}\). However, the morphologic diagnostic criteria for making the diagnosis are variable \(^{46,47,53-56}\). The disease EPSM thus refers to the clinical syndrome of muscle disease, particularly rhabdomyolysis, with amylose-resistant, sarcolemmal inclusions of acid mucopolysaccharides evident on muscle biopsy sample. In distinction, where there are clinical signs of myopathy but histologic evidence of no or mild myopathic changes with excess, aggregates or cores of sarcoplasmic, mostly amylose-sensitive polysaccharide [glycogen], then a distinguishing term such as **polysaccharide-associated myopathy** should be used. The former is seen particularly as a likely autosomal recessive trait in Quarter Horses and related breeds and in several other breeds including draft horses, whereas excess or
unusual glycogen storage is seen in association with clinicopathologic myopathic syndromes in a wide variety of breeds and may be present, histologically, in upwards of 50% of horses.

EPSM is thus one cause of exertional rhabdomyolysis and glycogen-associated myopathy probably is also. Signs of a hypometric, short-stride gait, reluctance to move, thoracolumbar lordotic and kyphotic postures and several movement disorders can be seen in association with these disorders. It does appear however that glycogen-associated myopathy is not the cause of most cases of the common postural and movement disorder known as shivers in draft horse and many other breeds. Our search for common factors in the cause of this group of ubiquitous and enigmatic postural and movement disorders continues.

The clinical syndromes can likely be ameliorated in many cases with management and dietary modification to include consistent and appropriate levels of exercise and addition of <20% lipid as a source of energy and reduction of soluble carbohydrates in the animals’ diet. Dantrolene sodium may help in altering membrane Ca++ flux to reduce the outcome of myolysis in these disorders.

Hyperkalemic Periodic Paralysis [HYPP]

Young-adult (2 to 3 years), mostly male Quarterhorse and Quarterhorse-related breeds have been reported to be affected with this autosomal dominant disease. Homozygous animals are more severely affected than heterozygotes. The owner notices intermittent episodes of muscle trembling over the body or face, sometimes with intermittent projection of the nictitating membrane, that may lead to involuntary recumbency. Other premonitory signs include yawning, lowering of the neck, swaying and disinterest in food and water. During a mild episode the horse is alert, appears distracted and reluctant to move, and may stumble as if weak. In a fulminant episode fasciculations or muscle tremor, particularly involving the flank, shoulders and neck and sometimes the face, progress to staggering, buckling, marked muscle spasms and paraplegia may precede involuntary recumbency. A severe episode, perhaps following forced exercise or KCl provocation, results in severe tremor and tetany of many muscles with recumbency and sweating. This is followed by a state of flaccidity, possibly with depressed spinal reflexes. Attempts to move the patient result in further tremor and tetany, although the horse remains alert. An episode may last several minutes to hours, typically less than an hour, with full and usually rapid recovery occurring. Between episodes, affected, well-muscled Quarterhorses appear essentially normal. Most owners notice stridor at some time or other in affected horses. Exercise and rest following exercise may precipitate episodes which can occur daily or monthly and stressors such as transportation, weaning and anesthesia.

The clinical syndrome in this inherited channelopathy is distinct but does closely resemble the acquired channelopathy induced by lolitrem-B in perennial ryegrass staggers.

HYPP is an autosomal dominant inherited channelopathy that is co-segregated with the alpha subunit of the equine adult sodium channel gene for which there is a single base pair substitution and accurate testing procedures are in place. The frequency of Quarterhorses positive for the heterozygote form of the mutation was estimated to be 4.4% in 1996. Interestingly, Quarterhorse show judges appear to favor the muscular phenotype of IMPRESSIVE, the sire that founded the lineage of affected progeny; a factor to confound attempts at testing for and removing the gene from the equine DNA pool!

Stiff horse syndrome

Recently, a stiff horse syndrome similar to stiff person syndrome in people has been recorded. Clinical signs appear to wax and wane and range from mild muscle stiffness to sudden and often violent muscle contractions. Generally there is an insidious onset. Between episodes the horse may appear normal although if present, the generalized muscle stiffness may persist.

A stiff person syndrome has been recognized in humans for some time. It is characterized by muscle rigidity and episodic and often violent muscle cramps. This rare condition is associated with antibodies produced against the enzyme glutamic acid decarboxylase (GAD) which is responsible for...
converting gamma-aminobutyric acid [GABA] into its active form. GABA is one of the most important inhibitory central neurotransmitters and a reduction in GABA activity can lead to continuous contraction of both agonist and antagonist muscle groups resulting in spasms. Occasional cases of SPS following other diseases such as west Nile fever possibly are due to cross reactivity of antibodies to WNV and to effector targets such as GAD 70. Variations on the SPS that are associated with muscle cramping restricted to one limb such as the stiff leg syndrome also occur. Recently, a similar syndrome has been seen in horses and is termed the stiff horse syndrome [SHS] 71.

Clinical signs appear to wax and wane and range from mild muscle stiffness to sudden and often prominent muscle contractions. Generally there is an insidious onset. Exercise intolerance associated with mild to moderate muscle stiffness may be the only initial clinical sign. This may easily be attributed to a primary myopathy, with pain on muscle palpation, although serum muscle enzyme concentrations remain in the normal range. Components of the syndrome bear resemblance to such disorders as tetanus, equine motor neuron disease, hyperkalemic periodic paralysis, exertional myopathies and especially the acquired channelopathies associated with the mycotoxices such as perennial ryegrass staggers.

The most useful diagnostic test is detection of antibodies against GAD in serum and cerebrospinal fluid and although some cases have had high anti-GAD titers several strongly suspected cases have been negative on this test. It may be necessary to liaise with a human hospital for analyzing for GAD antibodies in the obtained samples. The test relies on cross-reaction with human antigens. CSF can also be tested for EPM.

The overall message really is that with the array of enigmatic movement and postural disorders encountered in equine neurology that appear to be variations on the themes of stringhalt, shivering and claudication 72-74, a broad approach to delving into possible etiologic mechanisms should be taken that includes the possibility of immune associated neurotransmitter derangements such as SPS.

**Grass sickness**

Grass sickness or equine dysautonomia has been described since the early 20th century and since then has had quite a devastating effect on equine populations in parts of Western Europe 75-78. Horses of all breeds as well as nondomestic equidae and camelids can be affected and dogs, cats, rabbits and hares are affected by similar dysautonomies 77, 79-84. Most often, this disease occurs in 3 to 8-year-old horses that are kept outside during late spring and summer, although cases occur year round and rarely in stalled animals. The disease occurs commonly in Northern and Western Europe, particularly in Scotland and England 77, 78, 81. More recently it has been recorded as an epizootic in Hungary where 15 out of 55 one to three year old horses succumbed to the disease over one summer with only three surviving 85. An identical equine dysautonomia known as Mal Seco occurs in at least Argentina and Chile in South America and grass sickness appears to now occur in the horse in North America 86-88.

Per-acute colic with gastrointestinal stasis and rupture, anorexia with mild signs of colic and ileus, to chronic cachexia covers the general spectrum of syndromes seen with this very unusual disease. Moderate tachycardia, indifference to food, difficulty swallowing, excessive salivation, depressed gastrointestinal sounds, abdominal distension and usually mild colic are very often present to varying degrees. Muscular tremor and patchy sweating may be primary signs or may reflect the dehydration, electrolyte imbalances and colic that occur. Posturing with all feet close together as a weak patient does, ptosis and especially rhinitis sica are very distinctive signs when present.

No definitive clinical diagnostic test exists. As the clinical signs of Horner’s syndrome are symmetric, ptosis can be difficult to detect. However, a recently described aid to clinical diagnosis is the observance of a rapid reversal of ptosis with 0.5ml of 0.5% phenylephrin eye drops 89. The resulting marked difference in degree of ptosis and particularly in eye lash angle can be spectacular.

**Atypical myopathy**

Several hundred cases of highly fatal, atypical myopathy or myoglobinuria have been reported in young, adult, grazing horses mostly from Europe but also North America and Australasia 90-93. Horses
may be found dead or more often showing various signs of reluctance to move, stiff and short strides, apparent sedation and fine muscle tremors. They quickly become laterally recumbent and urine becomes dark with myoglobin staining although more sub-acute cases do occur. Tachypnea and even difficult respiration can mimic primary pulmonary disease with an accompanying tachycardia. Substantial cytosolic muscle enzyme release into the circulation occurs and along with confirmation of myoglobinuria forms the basis of the clinical diagnosis. Symptomatic fluid and analgesic therapy with attentive nursing care for severely ill and often recumbent patient is called for. The lethality of the disease is around 90%.

Outbreaks do occur, usually in the colder months, and can occur repeatedly on a property. Access to trees and inclement weather appear to be risk factors for the disease. Plant, bacterial and fungal toxins have all been considered as possibilities but the cause or causes remain completely unknown. Preliminary results from one group of investigators suggest that *Clostridium sordellii* and *Clostridium bifermentans* producing lethal toxin may play a role in what they term pasture myodystrophy. Possibly additional factors such as antioxidant deficient or toxic states inducing mitochondrial degeneration could be operative but vitamin E and selenium deficiency are not shown to be consistently present in affected patients and at risk animals.

Clinicians suspecting cases are urged to log on to the atypical myopathy alert site and complete the appropriate forms in an effort to unravel the epidemiology of this dastard disease:
dominique.votion@ulg.ac.be; http://www.ivis.org/reviews/rev/votion/chapter.asp?LA=1#forms

**Complex regional pain syndrome**

Complex regional pain syndrome in humans is a debilitating clinical pattern of signs falling within the spectrum of neuropathic pain disorders, characterized by a chronic, severe, burning pain sensation and having other strict diagnostic criteria. There is usually an area of intense allodynia [pain in response to non-painful stimuli], hyperalgesia [increased responsiveness to painful stimuli], objective evidence of local autonomic dysregulation [vasomotor disturbance, increased or decreased skin temperature, sweating abnormalities, and edema] and dermal changes [e.g. hyperpigmentation, altered hair growth, inflammatory skin lesions]. The spectrum of clinical presentations is exemplified by the various historical terms used to describe this debilitating syndrome including reflex sympathetic dystrophy, causalgia, algodystrophy, Sudeck’s atrophy and post-traumatic vasomotor syndrome. Cases of complex regional pain syndrome have been reported in horses in which the localized profound allodynia has been so debilitating as to prompt decisions for euthanasia. Interestingly, sometimes following a perineural anesthetic block is performed in the limb of a horse there is a regional strip of reflex vasodilation and sweating proximal and distal to the injection site. The regional distribution of this sympathetic response outside the resulting analgesic area is similar to that seen around the trigger point of horses having complex regional pain syndrome.

**Fluphenazine toxicity,**

The long acting phenothiazine drugs perphenazine and fluphenazine are used in equine reproductive disorders and as long acting anxioytics. As in humans treated for psychiatric disorders, a few treated horses have shown toxic effects of movement disorders perhaps representing extra pyramidal dysfunction akin to a Parkinson-like syndrome. What is seen is fluctuant somnolence and compulsive activity that can consist of adopting abnormal postures and displaying incredibly frantic, repetitive movements. Thus an affected horse may stand fixated by an object and appear to watch unapparent images move, stand with the forelimbs placed well forward and the head flexed between the knees, and perform tonic repetitive movements such as incessant pawing with one limb, head swinging and pseudo-rubbing on objects. Recumbency without somnolence and seizure-like activity also occurs. Anticonvulsant and narcotic drugs are indicated to help control the signs and possibly anticholinergic drugs such as benztrpine and diphenhydramine can be considered.
**Granulomatous meningoencephalomyelitis [GME]**

A collection of cases demonstrating disseminated, multifocal, granulomatous lesions are documented in large animal internal medicine and dermatology literature and are referred to by several terms including idiopathic generalized granulomatous disease, eosinophilic granulomatous disease, equine sarcoidosis, multisystem granulomatosis, systemic granulomatous disease and equine histiocytic dermatitis; the former definition being preferable for many reasons 104-108. In one case of idiopathic systemic granulomatous disease in an aged mare there was a multifocal granulomatous encephalomeningitis and neurologic signs following a short febrile respiratory consisted of rapid progression of somnolence, head pressing, excitement, seizures and recumbency 109. Subacute leukoencephalitis and necrosis was accompanied by necrotizing vasculitis and large perivascular accumulations of lymphocytes, macrophages, some neutrophils and multinucleated giant cells. Several other very similar isolated cases have been seen in horses by the author and have been documented 110.

In general, it is accepted that equine idiopathic generalized granulomatous disease is a programmed immune response to chronic environmental antigens 107, 111. On the other hand, with further cytologic specification some of the apparently inflammatory meningoencephalitides may more closely resemble diffuse lymphoma, meningeal sarcoma or microglioma 111, 112. Drawing a likeness between this idiopathic generalized granulomatous disease in horses to *Vicia villosa* [hairy vetch, fodder vetch] toxicity 113-116 is enticing. This is particularly so as firstly these plants contain numerous phytolectins that bind lymphocytes 117 and secondly non-suppurative, granulomatous leukoencephalitis has been detected histologically in one horse with this disease 114.

**Sclerosing panencephalitis**

Sclerosing panencephalitis occurs in at least two specific diseases in humans, namely subacute sclerosing panencephalitis associated with modified measles virus DNA and progressive multifocal leukoencephalopathy associated with a papovavirus. A novel case of sclerosing panencephalitis has been documented in a mature horse showing progressive multifocal asymmetric signs including blindness, hypermetric ataxia and multiple asymmetric cranial nerve dysfunction over c.12 weeks 118. Lymphocytic encephalitis, necrosis, demyelination and prominent fibrillary and gemistocytic astrogliosis dominated the lesions widespread throughout the neuropil and meninges of the brain. Although a diffuse astrocytoma could result in some of the cellular characteristics it was concluded that this was primarily a panencephalitis. A search for viral DNA in future cases is warranted.

**Anatomy of the sphenopalatine sinus**

Disorders of the equine sphenopalatine sinus, including empyema and neoplasia, have been reported to cause damage to cranial nerves II and V. However, the clinical anatomy of these sinuses has only recently been well described in horses 119. There is much variation between individual horses in sphenopalatine sinus anatomy. The sphenoidal sinuses are small in young horses and become larger and more complex with age. Variation in the extent that the sphenopalatine sinus extended into the basisphenoid bone also occurs. The septum dividing left and right sphenoidal sinuses is frequently not midline, but always intact. The sphenoidal and palatine sinuses communicate in most horses. In such cases, what could accurately be termed the (combined) sphenopalatine sinuses usually drains directly into the caudal maxillary sinuses. Additionally, in 5 out of 16 cases, some compartments of the sphenoidal sinus also drain into the ethmoidal sinus. The dorsal and lateral walls of the sphenoidal sinus are very thin and directly adjacent to cranial nerves II, III, IV, V and VI and major blood vessels. Thus many combinations of cranial nerves and blood vessels can be damaged with disorders involving the sphenopalatine sinus with degrees of blindness, strabismus, anisocoria and facial analgesia being seen.

**Lateral digital myotenectomy for stringhalt.**

Stringhalt, also known as springhalt and Hahnentritt (“rooster kick”), is an anciently recorded disease that is characterized by a sudden, apparently involuntary, exaggerated flexion of one or both hind
limbs during attempted movement. The hind limb motion may be as mild as a slightly excessive flexion to violent movements during which the fetlock or toe will contact the abdomen, thorax and occasionally the elbow with attempted strides leading to a peculiar bunny hopping and plunging gait.

The form that usually occurs as outbreaks is seen in Australia, New Zealand, United States, Chile, and Japan and thus will be referred to as bilateral, plant-associated stringhalt. Usually there is symmetrical or slightly asymmetrical involvement of the pelvic limbs, with prominent distal muscle atrophy in severe cases. The thoracic limbs are also affected in severe cases with knuckling of the forelimb fetlocks, prominent extension of more proximal joints and atrophy of the distal musculature, in association with prominent stringhalt in both hind limbs. Bilateral stringhalt has been associated with exposure to several plants notably *Hypochoeris radicata*, *Taraxicom officinal* and *Malva parviflora* [mallow weed]. These are related species of flat weeds, *Taraxicom officinal* being the common dandelion. It is interesting that size and age may be predisposing factors in at least bilateral stringhalt, in so far as older and taller horses tend to become affected in preference to smaller horses such as ponies and native Chilean breeds.

The pathological lesions present represent a distal axonopathy preferentially affecting large diameter axons in long nerves. This explains the muscle atrophy but there must also be selective involvement of γ-efferent fibers to account for the movement disorder with abnormal input via the 1a-afferent fibers to the α-efferent neurons resulting in inappropriate firing of lateral digital extensor and other muscles.

Although palliative, removing a section of the myotendinous region of the lateral digital extensor muscle relieves the syndrome quite spectacularly in many cases.

**Temporohyoid osteoarthropathy,**

Temporohyoid osteoarthropathy [THO] with proliferative osteopathy involving the temporal bone, temporohyoid joint and hyoid bone, is reported only in adult horses and may be sub-clinical or may result in evidence of difficulty chewing or more often neurologic syndromes, notably various combinations of facial and vestibulochoclear nerve dysfunction. Some of the cases have bilateral disease as determined by endoscopic and radioimaging studies, although the clinical signs are most often unilateral. The cause of temporohyoid osteoarthropathy is unclear although to this author a traumatic origin is most plausible in most cases with chronic otitis media/interna accounting for a select few cases. Regardless of the etiology of the osteoarthritis, clinical signs can occur from either the osteoarthritis itself or from fractures of the adjacent temporal bone and rarely basilar bones, due to partial or complete fusion of the joint.

Physical examination findings may include difficulty chewing, pain on external palpation of the parotid area, head shaking and behavioural problems especially when being ridden. Once the joint is partly fused, sudden forced head jerking, falling, teeth floating, nasogastric intubation and sudden prolonged vocalization can cause periarticular fractures of the petrous temporal bone resulting in combinations of an abrupt onset of facial and vestibular nerve dysfunction.

Endoscopic examination of the guttural pouch is probably superior to plain radiographic imaging in confirming the presence of the disease by revealing enlargement of the proximal stylohyoid bone due to osteoarthritis when compared to the opposite side. In acute or progressive cases having ill-defined endoscopic and plain radiographic imaging findings, gamma scintigraphy should be considered as a diagnostic aid. Monitoring brain stem auditory evoked potential recordings is a very non-invasive procedure that may assist in refining the prognosis in individual cases and following progress of the disease.

Several cases have improved with the passage of time only to show further signs relative to facial and vestibular nerve dysfunction in weeks to months time. These would seem to be ideal candidates for unilateral surgical disunion of the hyoid apparatus. Initial surgical disunion of the hyoid apparatus was performed by removal of a mid-shaft portion of the stylohyoid bone. To reduce the temporary...
difficulties in swallowing encountered and to reduce the possibility of other real and potential complications of this surgery the technique of ceratohyoidectomy was proposed and used with success \textsuperscript{135, 136}.

Except with major cranial fractures and if eye problems cannot be resolved \textsuperscript{135}, the outlook for survival with residual neurologic deficits is quite good. Of 33 cases of temporohyoid osteoarthropathy\textsuperscript{132}, 20 cases survived for which there were longer term follow up details. Of these, 70\% returned to previous level of use although more than 50\% of the 20 horses still had evidence of facial nerve deficits and/or vestibular dysfunction. Thus, in spite of some optimistic suggestions, if full athletic performance without neurologic dysfunction is required then the prognosis with or without surgical intervention has to be fair to guarded for these cases.

Cases of THO have given us a better insight into the ability of horses to accommodate to vestibular dysfunction and to survive with degrees of facial paralysis.

**Post-anesthetic cerebral necrosis**

An newly defined unexpected complication of apparently routine general anesthesia in some mature horses is diffuse and severe cerebral necrosis resulting in signs of diffuse encephalopathy immediately or some hours to days after recovery from anesthesia \textsuperscript{139, 140}. There is cerebral edema and laminar neuronal cortical necrosis, and with time gliosis and small perivascular mononuclear cuffs, most prominent in the watershed zones between major vascular supplies in the occipitoparietal lobes. These lesions are associated with generalized signs predominantly consisting of somnolence to dementia, central blindness, wandering compulsively, pushing against objects and ataxia. One patient with this tentative diagnosis that recovered showed prominent muzzle and ear twitching \textsuperscript{141}, very reminiscent of patients suffering from bacterial meningitis and from West Nile viral meningoencephalitis.

There certainly must be a compromise to cerebral circulation or metabolism and circumstantially it is related to the general anesthetic procedure, but no consistent perturbations predominate \textsuperscript{139, 142}.

**Concluding Comments**

Being a biology watcher does have its rewards in equine neurology. Over the last 5-10 years by making accurate observations, recording of findings and publishing clinical results we have a better insight into the pathophysiology of diseases, the application of better therapy and improved accuracy of diagnosis and prognosis. Hopefully this discourse on some of the interesting aspects of several equine neurologic diseases will stimulate others to share their experiences also.

**References on request**