the age. Although the disease typically affects patients of middle-age (median age of onset is 39 years) it is important to remember that 15–20% of patients may present to paediatricians (<16 years) or elderly care physicians (>65 years). It should be suspected when the MRI spine demonstrates a longitudinally extensive spinal cord lesion, extending for three or more vertebral segments, primarily involving the central cord gray matter. The syndrome is rare therefore there are no randomised controlled trials to guide treatment. Relapses are severely disabling, therefore prevention with immunosuppression is the most important aspect of management.

3195 A CASE REPORT OF ACUTE DISSEMINATED ENCEPHALOMYELITIS WITH PROMINENT MENINGEAL INVOLVEMENT AND BENEFIT FROM DELAYED PLASMA EXCHANGE THERAPY

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Background We report a diagnostically challenging case of acute disseminated encephalomyelitis with prominent meningeal involvement on neuraxial imaging. This case demonstrates the utility of plasma exchange, even in a delayed setting, for functional recovery.

Case A 62-year-old male traveller from Vietnam presented with three days of fever, myalgia and confusion. His condition deteriorated rapidly despite empirical treatment for infective meningoencephalitis, with flaccid quadriparesis and hypoxic respiratory failure requiring invasive ventilation. His CSF was floridly inflammatory with no positive microbiology and equivocal oligoclonal bands. Neuraxial MRI showed multiple periventricular deep white matter lesions, some exhibiting diffusion restriction and faint contrast-enhancement, a prominent C5/6 contrast-enhancing cord lesion, and extensive leptomeningeal enhancement and thickening, which progressed over two weeks with more numerous contrast-enhancing brainstem and cord lesions. The aetiology remained elusive despite extensive CSF and blood studies, bone marrow biopsy, nontargeted meningeal biopsy, and imaging including whole-body FDG-PET. He received empirical anti-tuberculosis antimicrobials, and pulse methylprednisolone followed by intravenous corticosteroids. He regained upper limbs' function but remained paraplegic. Serial imaging six weeks later demonstrated persistent gadolinium-enhancing, FDG-avid cord lesions. Therapeutic plasma exchange was performed despite the delay since presentation, and resulted in significant partial recovery in lower limbs' strength with markedly decreased neuraxial inflammation on imaging.

Conclusion Early prominent meningeal involvement posed significant diagnostic and management challenges in this case of acute disseminated encephalomyelitis. It also demonstrates that even in the delayed setting, where imaging evidence of neuraxial inflammation persists, therapeutic plasma exchange may provide benefit.

3197 EPILEPSY MANAGEMENT IN PRISONERS IN NEW SOUTH WALES, AUSTRALIA: SYSTEM OUTCOMES IMPROVEMENT IN OUTPATIENTS REVIEW

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Background In NSW, prisoners with epilepsy are reviewed at the Comprehensive Epilepsy Service at Prince of Wales Hospital (POWH).

Ongoing care of prisoners is hampered by lack of access to prisoners' medical records.

Many attributes of the normal history taking, physical examination and investigations are impacted because of the prisoner's privacy and prison security concerns.

Methods: Review of the literature on epilepsy, prison regulations and health care of prisoners.

Results All prisoners have a full medical assessment on entry.

The prison population is heterogeneous in – age, gender, location and security arrangements.

13,000 prisoners are housed in 39 facilities throughout NSW – from maximum to minimum security.

There is an over-representation of indigenous, less-educated and lower socio-economic individuals within prisons.

Based on self-reported data, there is a high prevalence of drug dependence, chronic co-morbid medical and psychiatric illnesses.

Innovations in Norway reduced recidivism to 20%, suggesting an effective intervention.

Incarceration significantly impacts prisoners' families with ramifications, especially after release.

Research into prisoners is limited by prison rules and legislated restrictions.

Conclusion Research into prisoners' health fails to match that within the community.

Short incarceration periods allows a small window of opportunity to monitor and improve prisoner health care.

Recidivism follows lack of intervention during incarceration.

Family health is under-researched - potentially affecting both family and prisoners post incarceration.

Outpatient hospital care requires greater access to medical records, held within the prison; hampering timely response during hospital outpatient appointments.

3199 ABDOMINAL PARAGANGLIOMA MIMICKING IDIOPATHIC INTRACRANIAL HYPERTENSION

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An otherwise healthy male in his twenties suffering from a four-month history of headaches, four-day history of visual disturbance and hypertension was referred to the emergency department by his general practitioner. The patient remained persistently hypertensive following admission, with systolic

blood pressures exceeding 200 mm Hg, and ophthalmological examination revealed changes consistent with those of hypertensive retinopathy (Grade IV). Computed tomography (CT) and subsequent magnetic resonance imaging of the brain were conducted, both demonstrating findings suggestive of idiopathic intracranial hypertension (IIH). Collectively, however, the clinical picture was more consistent with 'hypertensive emergency' than IIH, particularly given the discordance of the patient's phenotype with that typically seen in IIH. This prompted investigation into the cause of secondary hypertension, which revealed elevated plasma normetanephrines. A mass suspicious for a para-aortic paraganglioma was subsequently demonstrated on CT. Surgical excision of the mass, which was histopathologically consistent with a paraganglioma, resulted in rapid resolution of the hypertension and symptoms. This case illustrates (i) the importance of exploring for secondary causes of hypertension in individuals presenting with a suspected hypertensive emergency, particularly young individuals; (ii) emphasises the lack of specificity of neuroimaging findings traditionally associated with IIH; and (iii) reveals that abdominal paragangliomas can radiologically mimic idiopathic intracranial hypertension, manifesting with these same neuroimaging findings. This case will be discussed in relation to published information on idiopathic ICH.

3200 SIMULTANEOUS ONSET OF CENTRAL AND PERIPHERAL DYSFUNCTION: AN INTERESTING CASE OF NITROUS OXIDE INDUCED ACUTE MYELONEUROPATHY AND THALAMIC CHANGES

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Background Recreational Nitrous oxide (N_2O) use has seen a rise in Australia in recent years. Chronic N_2O misuse leading to adverse neurological sequelae such as myeloneuropathy are well described in literature. We report a case of chronic N_2O inhalation causing cognitive dysfunction and thalamic changes on MRI-Brain along with concurrent acute myeloneuropathy.
Objective To describe a case of chronic N_2O use induced cognitive dysfunction with thalamic changes on MRI-Brain.
Case A 24-year-old male with no past medical history presented with 2 weeks of progressive lower limb weakness, paraesthesia, ataxia, forgetfulness, and low mood on a background of heavy N_2O (8g) and cocaine (0.5–2g) inhalation every day for 4 months.

Neurological examination showed mild short term memory loss, hyporeflexia and sensorimotor impairment predominantly affecting bilateral distal lower limbs.

Investigations revealed an elevated methylmalonic acid level of 23umol/L. CSF studies showed mildly elevated proteins (564mg/L) with no oligoclonal bands. Nerve Conduction Studies showed evidence of a motor predominant axonal polyneuropathy. MRI-Spine showed cervical myelopathic changes from C2-C7 vertebral level. MRI-Brain showed right thalamic hyperintensity on FLAIR images without corresponding contrast enhancement or diffusion abnormality.

Patient was treated with intramuscular hydroxocobalamin and improved with neurorehabilitation.
Conclusion N_2O can cause cognitive dysfunction in addition to myeloneuropathy. N_2O related neurotoxicity to the brain has not been well documented. Clinicians should be vigilant in the search of clinical and radiological features in patients with suspected N_2O misuse.

3201 CASE OF HIRAYAMA DISEASE WITH UNUSUAL NEUROPHYSIOLOGY

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Background Hirayama disease is a cervical myelopathy, characterized by painless progressive unilateral or bilateral asymmetric amyotrophy of C7-T1 myotomes, with sparing of the brachioradialis and proximal upper limb muscles, without objective sensory or lower limb involvement. Clinical progression is usually self-limited. Patients have characteristic abnormal forward-shifting of the posterior dura detected only on dedicated neck flexion cervical spine MRI.
Case C.D. is a 15-year-old, active male, with no past medical history, presenting with a 2 year history of progressive distal right upper limb weakness, which has plateaued over the past 6 months.

Examination reveals right distal upper limb wasting and weakness (oblique amyotrophy), with preservation of proximal strength and normal sensory examination.

Initial nerve conduction studies (NCS) of the right upper limb showed prolonged distal motor latencies (DMLs), reduced motor amplitudes and absent f-waves, with normal sensory studies. Left upper limb studies were normal.

C.D. was treated with IVIG for possible multifocal motor neuropathy, without clinical improvement. Repeat NCS revealed normalisation of distal motor latencies and f-wave latencies, but impersistent f-waves. MRI cervical spine with neck flexion showed widening of dorsal epidural space and mild focal atrophy of right hemicord C5-C7.

IVIG has since been ceased, and NCS and motor function remains stable.
Discussion C.D.'s clinical features are most in keeping with Hirayama disease. We present atypical neurophysiology findings for Hirayama disease, so as not to misdiagnose this condition, and have patients on ineffective treatments.

3202 MILLS SYNDROME: PROGRESSIVE HEMIPARETIC PRESENTATIONS OF AMYOTROPHIC LATERAL SCLEROSIS (ALS)

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Background Mills syndrome is a relatively slowly-progressive, neurodegenerative motor neuron disorder, first described in 1900. A distinctively unilateral disease course remains the hallmark of Mills syndrome.