related to visuospatial impairment($p=0.02$) but not to global measures of cognition, motor severity, age or disease duration. Factor analysis confirmed a unique relationship between color discrimination, visual hallucinations and visuospatial function.

Conclusion Our results suggest that color impairments may be a specific biomarker of visual hallucinations and associated visuo-perceptual deficits in evolving Lewy body disorders rather than dementia per se and thus providing insight into a shared pathophysiological substrate.

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TRAINING IN HEADACHE IN AUSTRALIA, NEW ZEALAND AND ASIA

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10.1136/jnnp-2019-anzan.53

Introduction Migraine is the leading cause of age-adjusted neurological disability in Australia, but little is known about headache training in our region. We aimed to assess the quantity of teaching in headache subjects during undergraduate and postgraduate years.

Method This is a cross-sectional survey study where questionnaires were sent to 137 delegates from Australia, New Zealand and Asia, prior to the Headache Master School in Sydney in August 2018. The Main outcome measured are recalled number of hours of teaching in undergraduate year and postgraduate years in: 1) Migraine; 2) Trigeminal autonomic cephalalgias (TACs); 3) Asthma; 4) Myasthenia gravis (MG).

Results The questionnaire response rate was 73% (100 of 137), of which 29 delegates were within 10 years of completing their undergraduate degree and 98 were neurologists. In undergraduate training, there was much greater quantity of teaching in asthma than migraine ($Z=5.007$, $p<0.000$) despite both being high-prevalent (asthma 11%, migraine 15–20%) conditions. Similarly, for diseases of medium-to-low prevalence, there was less training in TACs (1/1000), compared to MG (1.2/10,000) ($Z=6.196$, $p<0.000$). These major differences in training were also seen in postgraduate years even though overall headache teaching was greater in postgraduate than undergraduate training ($p<0.000$).

Conclusions Despite the high prevalence and morbidity of headache disorders, they receive less attention in training than conditions with similar prevalence. We propose that headache training opportunities should be improved in our region, particularly in the undergraduate course and preceptorships or fellowships in postgraduate years.

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ALCOHOL ASSOCIATED TOXIC MYELOPATHY WITH NORMAL HEPATIC FUNCTION

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10.1136/jnnp-2019-anzan.54

Introduction Alcohol related neurological dysfunction affects the central and peripheral nervous system. We present a rare consequence of excessive alcohol consumption. A 42-year-old female consuming 2 bottles of wine daily for the preceding 5 years presented with 12 months of progressive gait disturbance. Examination revealed a spastic paraparesis with prominent dorsal column signs. Extensive work up found no alternate cause for her presentation. The final diagnosis was direct alcohol mediated toxic myelopathy. We reviewed the current literature on this rare condition.

Methods We searched PubMed/OVID databases with the terms 'alcohol AND myelopathy'.

Results There is a paucity of research on this clinical entity. Sage *et al.*¹ described 5 well-nourished alcoholics presenting with a progressive myelopathy. As with our patient, routine laboratory tests, including nutritional screen, were normal. CT Myelogram revealed no structural cause. CSF was normal. In contrast, a myelopathic syndrome can occur in alcoholic hepatic failure, attributed to portosystemic shunting of blood, with resultant hyper-ammonaemia and demyelination of corticospinal tracts.^{2–4}

Conclusion Alcohol associated toxic myelopathy is a rare entity, distinct from hepatic myelopathy. It is thought to result from direct toxic effects of ethanol on corticospinal and large myelinated sensory tracts. The sensory involvement is unique for this entity, which distinguishes it from hepatic myelopathy, where corticospinal involvement predominates. It is prudent to look for other potentially treatable secondary causes of myelopathy. Abstinence from alcohol results in modest improvement in symptoms. Minimal literature exists on this clinical entity and further research is required.

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HEADACHE WITH PERIORBITAL ECCHYMOSIS; A RARE CLINICAL ENTITY

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10.1136/jnnp-2019-anzan.55

Objectives Primary headache disorders are common with migraine and tension headache accounting for the vast majority of cases. A smaller proportion suffer from trigeminal autonomic cephalgia (TAC). We present a 23-year-old Caucasian female who described characteristic, episodic headaches starting with a dull retro-orbital/bi-frontal pressure evolving, over the course of 1 minute, to experience florid periorbital ecchymosis. While this phenomenon has been described in the literature, the characteristics of our case are unique and noteworthy of reporting.

Methods We reviewed the literature surrounding this rare entity by using PubMed/OVID databases and the search terms 'Headache AND ecchymosis'.

Results Case reports exist in older patients¹⁻³, where the headache is side locked and associated with other autonomic characteristics such as periorbital oedema, conjunctival injection and tearing. Our case is a young female with only ecchymosis in a unilateral and/or bilateral manner and no other autonomic or indeed migraine features. The patient underwent vascular/cranial imaging and blood tests to exclude haematological, autoimmune, vasculitic causes for this presentation which were unrewarding.

Conclusion Variations on this clinical entity are described;¹⁻⁴ we hope this report may bring attention to this fascinating phenomenon. The pathophysiological process is likely to be similar to those implicated in TACs, namely activation of the trigemino-neurovascular system and facial autonomic pathways. The release of neuromediators such as CGRP, VIP and Substance P cause blood vessel fragility resulting in diapedesis. Optimal treatment regimens are unknown but various agents have been trialled. Our patient declined treatment and continues to be followed.

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PARKINSON'S DISEASE AND THE GASTROINTESTINAL MICROBIOME: CLINICOPATHOLOGICAL CORRELATIONS AND CONTROVERSIES

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10.1136/jnnp-2019-anzan.55

Introduction There has been a recent surge in interest around the gastrointestinal microbiome (GM) and its association with Parkinson's disease (PD). The GM mediates interactions between the brain and the gut via the 'microbiota-gut-brain-axis'. Compelling studies suggest that a shift in GM composition may play an important role in the pathogenesis and progression of PD.

Methods We conducted a literature review exploring the pathological association between the GM, α -synuclein spread and intestinal inflammation in PD. We also summarised patterns and correlations of gut microflora seen in clinical studies of the GM in PD.

Results To date 14 mainly cross-sectional studies from 7 countries have reported GM alterations in PD. All studies described significant alterations between PD and healthy control groups across multiple bacterial families, genera and

species. Several studies suggested that putative 'pro-inflammatory' bacteria were significantly more abundant, while putative beneficial bacteria were less abundant in PD. Various complex microbiota-gut-brain-axis interactions have been proposed due to alterations in the GM, inferred by changes in gut mucosal integrity and permeability, short-chain-fatty-acid metabolism, oxidative stress and inflammation.

Conclusions Across the recent GM studies in PD, alterations in bacterial taxa have been repeatedly associated with various clinicopathological features, endorsing a plausible biological link between the GM and PD. Mechanisms involved in the pathogenesis of PD due to GM changes are complex and require ongoing study.

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PRESENCE OF ANTI-MYELIN OLIGODENDROCYTE GLYCOPROTEIN ANTIBODIES IN THE SERUM OF TWO PATIENTS FOLLOWING ALEMTUZUMAB THERAPY FOR SUSPECTED MULTIPLE SCLEROSIS

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10.1136/jnnp-2019-anzan.57

Introduction Myelin oligodendrocyte glycoprotein (MOG) antibody mediated disease is an autoimmune demyelinating disorder which can resemble multiple sclerosis (MS).^{1 2} Thus, this condition can be misdiagnosed and treated as MS.³ We present the clinical trajectory of two cases initially diagnosed as MS, treated with Alemtuzumab followed by clinical and radiological deterioration. Both were subsequently found to have anti MOG antibody in their serum.

Methods This is a retrospective case study based on a medical record search of neuroimmunology clinics in two teaching hospitals in Victoria. We searched for patients treated with Alemtuzumab who subsequently tested positive for MOG antibody.

Results We found two young women who fulfilled the eligibility criteria. One patient presented with dizziness and vertigo, the other with unilateral optic neuritis. Both had supratentorial MRI lesions and were both diagnosed as having MS. Both patients experienced multiple relapses while on treatment for MS. Hence, they were commenced on Alemtuzumab therapy. Unexpectedly, both patients experienced a decline in their clinical status with worsening of expanded disability status scale (EDSS) and an increasing lesion load on MRI brain. Their serum anti MOG antibodies were then found to be positive. Subsequently, patients were treated with rituximab and plasma exchange with a favorable response.

Conclusions These two cases demonstrate that Alemtuzumab is ineffective and in fact can worsen cases of anti-MOG antibody associated encephalomyelitis. This highlights the importance of anti MOG antibody testing when patients diagnosed with MS do not respond to Alemtuzumab and in those patients presenting with atypical features of MS.

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