to 2017 (97% vs 76%, respectively). Similar findings were noted for patients with atrial fibrillation who received oral anticoagulants on discharge (90% vs 50%) and patients discharged on antihypertensives (95% vs 80%).

Conclusion Use of a clinical support platform in managing acute stroke is an intervention that improves stroke care.

REFERENCES

- Urimubenshi G, Langhorne P, Cadilhac DA, Kagwiza JN, Wu O. Association between patient outcomes and key performance indicators of stroke care quality: A systematic review and meta-analysis. *European Stroke Journal* 2017;2(4):287–307. <https://doi.org/10.1177/2396987317735426>
- Sandercock P, Gubitz G, Foley P. Antiplatelet therapy for acute ischaemic stroke. *Cochrane Database Syst Rev* 2003;2: CD000029. Google Scholar
- Kwan J, Sandercock P. In-hospital care pathways for stroke. *Cochrane Database Syst Rev* 2004;4: CD002924. Google Scholar
- Saxena R, Koudstaal PJ. Anticoagulants for preventing stroke in patients with nonrheumatic atrial fibrillation and a history of stroke or transient ischemic attack. *Cochrane Database Syst Rev* 2004;4: CD000187. Google Scholar
- Goyal M, Menon BK, van Zwam WH. Endovascular thrombectomy after large-vessel ischaemic stroke: a meta-analysis of individual patient data from five randomised trials. *Lancet* 2016;387:1723–1731. Google Scholar | Crossref | Medline | ISI
- Middleton S, McDuff P, Ward J. Implementation of evidence-based treatment protocols to manage fever, hyperglycaemia, and swallowing dysfunction in acute stroke (QASC): a cluster randomised controlled trial. *Lancet* 2011;378:1699–1706. Google Scholar | Crossref | Medline | ISI

037 FRONTOTEMPORAL DEMENTIA OR FRONTAL VARIANT ALZHEIMER'S DISEASE? A CASE SERIES

^{1,2}Alice Powell*, ³David Foxe, ³Glenda M Halliday, ³Olivier Piguet, ³John R Hodges, ^{1,3}James R Burrell. ¹Concord Repatriation General Hospital, Sydney, NSW, Australia; ²Brain and Mind Centre, University of Sydney, Redfern, NSW, Australia; ³Brain and Mind Centre, The University of Sydney, Sydney, NSW, Australia

10.1136/jnnp-2019-anzan.34

Introduction Accurate prediction of the underlying neuropathology in behavioural variant frontotemporal dementia (bvFTD) is essential for future targeted therapy trials and prognostication. Alzheimer's disease (AD) pathology has been reported in a significant proportion of patients with clinical bvFTD. We sought to determine whether detailed clinical and neuroradiological assessment was sufficient to distinguish bvFTD with AD pathology from bvFTD with frontotemporal lobar degeneration (FTLD).

Methods Two patients with clinically diagnosed probable bvFTD but AD pathology at autopsy, were identified. The clinical, neuropsychological and imaging features of these patients were compared with those of ten patients with clinically probable bvFTD and proven FTLD pathology (tau, TDP-43, FUS).

Results Both patients with AD pathology presented with behavioural symptoms typical of bvFTD as well as memory impairment. Executive function, memory and visuospatial skills were impaired in both pathologic groups. Language skills were relatively spared in those with AD pathology. Neuropsychiatric symptoms were frequent in both groups but significant depression and anxiety were seen only in those with FTLD pathology. Dementia severity and caregiver burden were similar. The degree or topographical distribution of atrophy on MRI did not differ.

Conclusions Alzheimer's pathology may cause bvFTD symptoms which are otherwise indistinguishable to those caused by FTLD pathology. While there may be subtle differences in patterns of cognitive deficits, standard neuropsychological testing is insufficient to discern the underlying pathology.

Similarly, structural imaging cannot be used to reliably identify AD pathology. Better access to amyloid biomarkers may be needed to more accurately define bvFTD caused by AD pathology.

038 TREMOR: A CLINICAL AND NEUROPHYSIOLOGICAL STUDY

^{2,1}Alessandro Fois*, ^{2,1}Neil Mahant, ^{2,1}Steve Vucic, ^{2,1}Victor SC Fung. ¹Movement Disorders Unit, Westmead Hospital, Westmead, NSW, Australia; ²Sydney Medical School, the University of Sydney, Sydney, NSW, Australia

10.1136/jnnp-2019-anzan.35

Introduction Tremor is a common clinical problem seen in a number of diseases. Robust classification and diagnosis of tremor remains controversial due to overlap in clinical features and lack of established biomarkers. This hampers effective research including therapeutic trials. We present our research protocol for a cross-sectional study which aims to find more robust methods of tremor classification and diagnosis.

Methods Adults with upper limb tremor of varying aetiologies, diagnosed using current clinical criteria (including essential tremor, Parkinsonian tremor, and dystonic tremor), and age-matched controls are eligible for recruitment. Participants undergo a clinical and neurophysiological assessment, including accelerometry, surface electromyography, long-latency stretch reflexes, temporal discrimination, and tonic vibration reflexes. Data will be analysed using a cluster analysis to identify robust tremor syndromes and biomarkers associated with them. We aim to recruit 100 participants prior to analysis.

Results At time of writing, 13 participants with upper limb tremor have been studied (6 with essential tremor, 5 with dystonic tremor, and 2 with indeterminate tremor; mean age 66 years, range 18–85). Participants tolerated the clinical and neurophysiological studies well with 100% completion rate after recruitment. With current rates of recruitment we anticipate completion of recruitment and commencement of data analysis in October 2019.

Conclusions Our protocol aims to identify robust tremor phenotypes and biomarkers for them. This will allow patients with tremor to be classified into more biologically homogeneous diagnostic categories, aiding future research into the mechanism of tremor and more rational clinical trial design.

039 ESTIMATING THE HEALTH AND ECONOMIC BURDEN OF INVESTIGATING TUMEFACTIVE DEMYELINATION COMPARED TO CONVENTIONAL MULTIPLE SCLEROSIS

¹Matthew Silsby*, ²Pedro S Lopez, ^{3,2}Judy Spies, ^{1,2}Stephen Reddel, ²Jane Frith, ²Joshua Barton, ^{3,2}Heidi Beadnall, ^{3,2}Michael Barnett, ^{1,2}Todd Hardy. ¹Neurology, Concord Hospital, Concord, NSW, Australia; ²Sydney Neurology, Brain and Mind Centre, Camperdown, NSW, Australia; ³Neurology, Royal Prince Alfred Hospital, Sydney, NSW, Australia

10.1136/jnnp-2019-anzan.36

Introduction Tumefactive demyelinating lesions, defined as demyelinating lesions > 2cm in diameter, occur most commonly in association with multiple sclerosis (MS), and can pose a diagnostic challenge. The aim of this study was to

estimate the cost and morbidity associated with the diagnostic investigation of patients with tumefactive demyelination (TD) compared to patients with conventional relapsing-remitting MS.

Methods Retrospective review of patients seen between 2013–2018 in clinics at the Brain and Mind Centre, Sydney; a centre with tertiary referral expertise in MS. Records were searched for the terms ‘tumefactive’ and ‘pseudotumour’. All patients diagnosed with TD were included and paired with a patient of similar age diagnosed in the same year with MS according to 2010 McDonald criteria.

Results There were 31 patients with TD and 31 patients with conventional relapsing remitting MS. The estimated cost of investigating TD was more than 7.5 times higher per patient than in MS (\$18,300 AUD vs \$2,418 AUD, $p<0.001$). Brain biopsy was performed in 6/31 TD patients and 0/31 MS patients and was the cause of more adverse outcomes in the TD versus MS group. More patients in the TD group were admitted to hospital (22/31 vs 10/31) and ICU admissions only occurred in the TD group (10/22 vs 0/10).

Conclusion The cost and adverse outcomes associated with investigating TD are higher than in conventional MS. Improvements in the diagnosis of TD have the potential to improve health and economic outcomes.

040

ACUTE CEREBELLAR ATAXIA FOLLOWING EPSTEIN-BARR VIRUS INFECTION

¹Stephanie L Barnes*, ^{2,3,4}Bruce J Brew. ¹Department of Neurology, St Vincent's Hospital, Darlinghurst, NSW, Australia; ²Departments of Neurology and Immunology, St Vincent's Hospital, Darlinghurst, NSW, Australia; ³Peter Duncan Neurosciences Research Unit, St Vincent's Centre for Applied Medical Research, Darlinghurst, NSW, Australia; ⁴St Vincent's Clinical School, Faculty of Medicine, University of NSW and University of Notre Dame Sydney, Sydney, NSW, Australia

10.1136/jnnp-2019-anzan.37

Introduction Infectious aetiologies such as acute Epstein-Barr virus (EBV) infection are in the differential diagnosis for acute cerebellar ataxia (ACA). This syndrome remains exceptionally rare and not well characterised in adults.^{e.g. 1 2}

Methods A retrospective case review of a patient diagnosed with ACA following EBV infection with implications for pathogenesis and treatment.

Results A 29-year-old Caucasian male presented with a three day history of ACA. Seven days prior he was diagnosed with infectious mononucleosis; bloodwork was consistent with acute EBV infection. These symptoms improved rapidly with oral prednisolone. He took no regular medications, drank alcohol moderately and had no significant family history.

On examination, he was afebrile, ataxic and mildly dysarthric. Sensory examination was normal, particularly proprioception. Romberg's test was negative. Remaining neurological and general examination was normal.

Bloodwork showed mild liver dysfunction and positive ANA (titre 1/320, homogenous and speckled patterns). Immune screen was otherwise negative. Antineuronal antibody panel was negative in serum and CSF. CSF glucose was 3.1 mmol/L, protein 751 mg/L, albumin 523 mg/L, neopterin 24

nmol/L and B₂ microglobulin 1.1 mg/L. The sample was acellular with negative EBV PCR (<500 copies/mL). Other infective serology and PCRs were also negative. MRI brain with gadolinium showed no abnormality.

The patient received supportive care and was neurologically normal within three months.

Conclusions ACA related to EBV is rare in adults. This report is important because it documents an adult case, other ACA causes have been rigorously excluded, resolution without antiviral therapy is detailed, and investigations support an immune-mediated pathogenesis.

REFERENCES

1. McCarthy CL, McColgan P, Martin P. Acute cerebellar ataxia due to Epstein-Barr virus. *Pract Neurol* 2012;**12**:238–240.
2. Lascelles RG, Longson M, Johnson PJ, Chiang A. Infectious mononucleosis presenting as acute cerebellar syndrome. *Lancet* 1973;**2**:707.

042

RESPIRATORY FUNCTION AND COGNITIVE PROFILE IN MOTOR NEURON DISEASE

¹William Huynh*, ²Lara E Sharplin, ¹Jashelle Caga, ¹Elizabeth Highton-Williamson, ¹Matthew C Kiernan. ¹Brain and Mind Centre, Camperdown, NSW, Australia; ²Faculty of Medicine, The University of Notre Dame, Sydney, NSW, Australia

10.1136/jnnp-2019-anzan.38

Introduction Motor neuron disease (MND) is increasingly recognised as a multisystems disorder with 30–50% of patients having mild to moderate cognitive impairment. Mechanisms of cognitive dysfunction in MND are multifactorial but chronic hypoxia secondary to respiratory dysfunction may contribute to cognitive decline in patients.

Objectives The current study aimed to identify the relationship between respiratory function in MND patients and the presence and degree of cognitive impairment.

Methods MND patients were prospectively recruited from a multidisciplinary MND clinic. Patients meeting the criteria for frontotemporal dementia were excluded. Baseline clinical assessments including respiratory function as assessed by spirometry were recorded with FVC $\leq 75\%$ considered to have reduced respiratory function. Cognitive testing was performed utilising the Addenbrooke's Cognitive Examination (ACE).

Results From a cohort of 100 MND patients 48% were categorised as having impaired respiratory function whilst 52% had normal function. Compared to the group with normal respiratory function (ACE: 86.83 ± 1.5), patients with respiratory dysfunction had significantly reduced cognitive function (ACE: 90.68 ± 0.89 , $P=0.025$). Subscores demonstrated significant differences between the groups with respect to domains in memory, attention with a trend observed in fluency. There was a significant correlation between FVC and ACE scores as well as between FVC and memory and fluency subscores ($P<0.01$).

Conclusion MND patients with respiratory compromise were more likely to develop reduced cognitive function. In addition to improving physical function, it remains plausible that non-invasive ventilation may alter the progression of cognitive impairment in MND patients, thereby potentially improving their overall quality of life and carer burden.