followed. Further research is required for risk stratification of RCVS to guide the early implantation of intraarterial vasodilation to avoid permanent neurological injury.

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3138 EFFECTIVENESS OF CLADRIBINE COMPARED TO FINGOLIMOD, NATALIZUMAB, OCRELIZUMAB AND ALEMTUZUMAB IN RELAPSING-REMITTING MULTIPLE SCLEROSIS

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Background/Objectives Cladribine reduces the frequency of relapses by 57% and disability worsening by 33% compared to placebo in relapsing-remitting multiple sclerosis (RRMS). No head-to-head comparisons of cladribine to other potent immunotherapies are available.

Methods Patients with RRMS who were treated with cladribine, fingolimod, natalizumab, ocrelizumab or alemtuzumab from 01/2018 were identified in the global MSBase cohort study and 2 UK centres. Included patients were followed for >=6 months and had a minimum of 3 disability assessments. Patients were matched using propensity score. Four pairwise analyses compared annualised relapse rates (ARR), and disability outcomes. All subsequent events were analysed, regardless of changes in treatment status.

Results The eligible cohorts consisted of 853(fingolimod), 464 (natalizumab), 1131(ocrelizumab), 123(alemtuzumab), or 493 (cladribine) patients. Cladribine was associated with a lower ARR than fingolimod (ARR 0.07 vs 0.12,p=0.006), and a higher ARR than natalizumab (0.10 vs 0.06,p=0.03), ocrelizumab (0.09 vs 0.05,p=0.008), and alemtuzumab (0.17 vs 0.04, p<0.001). Compared to cladribine, the risk of disability worsening did not differ in patients treated with fingolimod (HR1.08, 95%CI 0.47–2.47) or alemtuzumab (0.73, 0.26–2.07), but was lower for patients treated with natalizumab (0.35, 0.13–0.94) and ocrelizumab (0.45, 0.26–0.78).

Conclusion Cladribine is an effective therapy, associated with low absolute ARR. While cladribine effectiveness on relapses is superior to fingolimod, alemtuzumab is superior to cladribine in reducing relapses. Natalizumab and ocrelizumab are superior to cladribine in reducing both relapses and disability accrual. Cladribine can thus be viewed as a step-up in effectiveness from fingolimod, but less effective than the most potent intravenous MS therapies.

3141 DETERMINING ETHNIC DIFFERENCES IN NERVE CONDUCTION VELOCITY

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Background/Objectives Accurate normative data is essential for interpretation of nerve conduction studies (NCS), though the impact of patient ethnicity is unclear. Anecdotal experience suggests nerve conduction is faster in East Asians (EA) than in Caucasians. The present study tested this hypothesis.

Methods This is a retrospective audit of patients presenting to Westmead Hospital Neurophysiology between March 2020 and December 2022. Patients were included if amplitudes were within lab normative values; the median, ulnar, tibial, and sural nerves were assessed. Ethnic data was collected from recorded demographic information.

Results 200 patients were included: 84 East Asian and 116 Caucasian. There was no difference in median age (EA 55 [47–64], Caucasian 54 [43–64] years, P=0.135), male to female ratio was 2:3. The F wave latencies were significantly faster in East Asians after controlling for height (Ulnar: EA 23.0±7.9ms, Caucasian 27.5±2.0ms, P=0.005; Median: EA 22.2±7.6ms, Caucasian 26.3±1.8ms, P=0.013; Tibial: EA 47.4±4.8ms, Caucasian 51.7±5.4ms, P=0.041). Distal motor latencies were faster for ulnar nerves only (EA 2.5±0.4ms, Caucasian 2.7±0.4ms, P=0.009). Upper limb sensory latencies were faster in East Asians (Ulnar: EA 2.0±0.2ms, Caucasian 2.1±0.3ms, P=0.018; Median: EA 2.4±0.2ms, Caucasian 2.6 ±0.3, P=0.020), while only the sural nerve demonstrated significantly faster conduction velocities (EA 51.5±6.1m/s, Caucasian 48.0±4.8m/s, P=0.003). Motor velocities were not significantly different.

Conclusion The present study supports the hypothesis that nerve conduction is faster in East Asians, as measured by F waves and distal latencies. This has potential implications for normal values in neurophysiology laboratories. Prospective studies are planned to further explore these potential differences.

3144 CONSENSUS TO CLINIC: ENHANCING DIAGNOSTICS FOR POSTERIOR CORTICAL ATROPHY

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Background/Objectives Nearly thirty thousand Australians under the age of 65 live with dementia. Posterior Cortical Atrophy (PCA) is one form of young onset atypical dementia, often caused by Alzheimer disease. Due to its rarity, atypical phenotype and young onset, the diagnosis is often missed for years. A consensus expert opinion diagnostic framework was created in 2017 to reduce delays to diagnosis. We aimed to