neuropathy is a recognized part of FM, surprisingly little is known about large fibre neuropathy. The present study investigates the sensory and motor axonal properties using novel nerve excitability testing (NET) to seek a better understanding of the pathogenesis of this painful disorder.

Methods 25 FM patients were recruited from the Wan Fang Hospital in Taiwan who fulfilled the American College of Rheumatology diagnostic criteria. NCS, pain scores, blood tests and NET were performed in all patients and patients with factors that may confound the results of NET were excluded. Control data were obtained from age and gender-matched healthy controls (HC) who had no neurological deficits or known pain disorders.

Results The FM group showed an increase in superexcitability (p<0.05), subexcitability (p<0.05) and over-shoot during hyperpolarizing threshold electrotonus (p<0.05) in the sensory excitability profiles in contrast to HC. However, motor nerve excitability profiles showed no significant difference.

Conclusions Alterations in the sensory axonal parameters can be detected while NCS is normal, these findings are compatible with the concept that the sensory system is mainly involved in the pathogenesis of FM. Results implied probable hypofunction of the paranodal fast K+ channel in the sensory axons, known to be associated with the generation of pain. Our study highlights the advantage of NET over NCS, in the early detection of axonal dysfunction and may provide further understandings of future therapeutic treatment.

REFERENCES

Abstracts

118 CASE SERIES: SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD IN VITAMIN B12-REPLETE RECREATIONAL NITROUS OXIDE ABUSERS

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Introduction Within a few years of its discovery in late 18th century, nitrous oxide was being used recreationally for its pleasurable effects. It remains in widespread use as an inhaled stimulant today, and can be legally acquired in bulk quantities with relative ease. In the body prolonged exposure to nitrous oxide leads to the oxidation of vitamin B12, rendering it unusable in key enzymatic reactions necessary for myelin synthesis. Over time this qualitative deficiency leads to a central demyelination syndrome that characteristically develops despite normal serum vitamin B12 levels and, with continued exposure to nitrous oxide, resists treatment with vitamin B12 supplementation.

Method Nitrous oxide abusers presenting with a central demyelination syndrome were enrolled in this case series. Serum levels of vitamin B12, active B12, folate and homocysteine were measured. Nitrous oxide exposure was discontinued, and all patients were treated in accordance with evidence-based guidelines.

Results Eight patients presented with predominantly moderate-to-severe clinical deficits. The majority were vitamin B12 replete. In most cases individuals had actively engaged in prolonged vitamin B12 supplementation in an attempt to circumvent the harmful pathophysiology, of which they were loosely aware. Following treatment and rehabilitation several patients were discharged into full-time care, and most had significant residual disability at follow-up.

Conclusions This case series not only illustrates the tragic consequences of abuse of this widely available and legally procured stimulant, but also highlights the futility of...