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SODIUM OXYBATE AS A TREATMENT FOR DBS-RESISTANT, ALCOHOL-RESPONSIVE POST-ANOXIC MYOCLONUS

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Myoclonic jerks are sudden shock-like involuntary movements, either manifesting as a brief contraction of a group of muscles (positive) or cessation of muscle activity (negative), driven by aberrant activity in one of the cortex, subcortical regions, brainstem or spinal cord. Its aetiology is variable and can occur physiologically, in a primary myoclonic syndrome (myoclonus-dystonia), or as part of an epilepsy (Progressive myoclonic epilepsy) or neurodegenerative syndrome (Alzheimer's disease, multiple systems atrophy). Myoclonus can also occur secondary to hypoxic brain injury either in an acute (myoclonic status epilepticus) or a chronic form, eponymously titled Lance-Adams syndrome (LAS).

LAS is characterised by a non-progressive generalised myoclonus with added seizures and ataxia. There is no curative management strategy, only symptomatic relief. A multidisciplinary approach involving medical, physiotherapy, speech and occupational therapy achieves the best holistic outcomes for patients. Case studies suggest certain antiepileptic agents may alleviate symptoms, but combinations are often required, and the most effective options have sedative side effects. A subset of cases have marked alcohol responsiveness that can lead to dependence.

A number of experimental treatment options have been proposed including deep brain stimulation (DBS) and sodium oxybate, a sodium salt of gamma-Hydroxybutyrate. In what follows, a patient with an eleven year history of the LAS is presented and the experimental therapeutics explored, concluding with a recent trial of sodium oxybate. Sodium oxybate was well tolerated and produced improvements in the patient's symptoms and perceived disability. This case highlights the difficulties of managing chronic myoclonic conditions and suggests sodium oxybate may be a useful treatment option in these patients.