

compared to alteplase in the AIS population. The model structure combines a decision tree for the first 90 days post index stroke, where the 7 modified Rankin Scale (mRS) states are informed by the AcT trial, and a Markov model for the remainder of the lifetime horizon. Cost and utility values were derived from the literature and public sources. Canadian health care system and hospital perspectives were used. Results: This economic analysis demonstrates that tenecteplase is dominant compared to alteplase, providing more quality-adjusted life years at lower costs. Conclusions: Adding tenecteplase to hospital formularies for AIS would generate savings for the health care system while providing more benefits.

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Mapping motor pathways with transcranial magnetic stimulation to predict functional outcomes in non-human primate models of chronic stroke

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Background: Ischemic stroke often results in long-term motor impairments due to disrupted corticospinal pathways. Transcranial magnetic stimulation (TMS) motor mapping is a non-invasive technique used to assess corticospinal integrity by measuring motor evoked potentials (MEPs). This study investigates whether MEP amplitudes can predict impairment severity and functional performance in chronic stroke. Methods: Four non-human primates (NHPs) with chronic stroke (> six months) following transient right middle cerebral artery occlusion underwent TMS motor mapping using neuronavigation under ketamine anesthesia. Single pulses of TMS (50-70% of maximum stimulator output) were applied to the affected and contralesional primary motor cortices to elicit MEPs and assess cortical excitability. Intramuscular electromyography recorded muscle responses from the biceps, extensor digitorum longus, and abductor pollicis brevis. Neurological dysfunction was evaluated daily for three weeks using the NHP Stroke Scale, NHP Upper Extremity Motor Dysfunction Scale, and the primate Rankin Scale. Results: MEPs were present in NHP1, NHP3, and NHP4 but absent in NHP2. Stronger MEPs correlated with lower impairment severity and better functional performance, while NHP2 exhibited higher impairment and poorer performance. Conclusions: MEP presence and strength can serve as biomarkers of motor recovery potential, highlighting their role in assessing corticospinal integrity and functional outcomes.

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Epilepsy Absence of children, A Guinean cohort of 69 cases

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Background: Absence epilepsy is a common epilepsy syndrome in children. This can have a negative impact on the cognitive abilities of preschool and school-age children. The objective was to study in the Guinean context, the epidemiological, clinical, electrophysiological, therapeutic and evolutionary aspects of this syndrome. Methods: The study included all children diagnosed with absence epilepsy based on evidence obtained from history, clinical, and electroencephalogram. Results: The cohort was made up of 41 girls and 28 boys with a sex ratio (F/M) equal to 1.46. The mean age was 8 ± 2 years with extremes of 2 and 14 years. The simple absences were observed in 42.02% of cases. The components : tonic was associated in 11.59%, clonic in 10.14%, atonic in 13.04%, automatisms in 15.94% and vegetative in 7.25%. EEG was typical in 75.36%. As monotherapy, sodium valproate was used in 92.75% and ethosuximide in 2.9%. The evolution was marked by a remission of seizures in 85.51%. During follow-up, the appearance of tonic-clonic convulsions was noted in 4.3%, myoclonus in 2.9%, a combination of myoclonus and tonic-clonic convulsions noted in 4.3%. Conclusions: Effective and efficient collaboration between stakeholders is essential for the best overall management of this syndrome with serious cognitive repercussions in children.

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SYNGAP-1 developmental and epileptic encephalopathy: utility of corpus callosotomy and neuromodulation

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Background: Pathogenic variants in *SYNGAP1* cause developmental and epileptic encephalopathy (DEE) and intellectual disability. Seizures are medically refractory and there is limited evidence on the use of corpus callosotomy (CC) and vagal nerve