Whereas, S-100b levels ≥1.03 mcg/L on the 3rd day had sensitivity 57.8% (CI = 0.4544–0.6939), specificity 95.6% (CI = 0.8782–0.9909), PPV 93.2% (CI = 0.8134–9857) for poor neurological outcome.

The improved survival rate in HG patients was not observed, which was also the case in the previous trial published in 2013.[6] There was a tendency for the serum levels of both proteins to be higher in HG patients. Lower NSE levels have been reported earlier in HG patients but in those studies more patients had favorable neurological outcome.[7] The authors consider it unlikely that the kinetics of the two proteins were changed by TH. There is increasing evidence that resuscitated patients with NSE concentration much higher than the cut-off level can survive with moderate or good neurological outcome. Hemolysis or several forms of cancer may influence levels of NSE; similarly, S-100b is released from tissues other than brains such as adipocytes, chondrocytes and several forms of cancer of central nervous system and melanoma.

The authors conclude that TH has no influence on NSE and S-100b serum levels in comatose CA survivors. The increase in both the proteins indicate poor neurological outcome; hence, their measurement is an additional tool for making prognosis on comatose CA survivors. However, at present it is not possible to recommend reliable threshold protein concentration, further investigations in this field are warranted.

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Akeju O, Pavone KJ, Westover MB, Vazquez R, Prerau MJ, Harrell PG, *et al.* A Comparison of Propofol- and Dexmedetomidine-induced electroencephalogram dynamics using spectral and coherence analysis. Anesthesiology 2014;121:978-89.

Electroencephalogram pattern observed during sedation with dexmedetomidine appear similar to those observed during general anaesthesia with propofol. However, these drugs have different molecular mechanisms and behavioural properties and are likely accompanied by different neural circuit dynamics. Whether the differing clinical effects of these drugs can be distinguished by their electroencephalogram signature is unclear.

The authors hypothesized that propofol-induced slow oscillations would have lower coherence and larger power/amplitude than dexmedetomidine induced slow oscillations. Sleep-spindles observed during sleep and dexmedetomidine induced unconsciousness have morphology that is intermittent in nature in contrast to propofol-induced frontal alpha oscillations which are continuous in nature. They further hypothesized that alpha-oscillations induced during general anaesthesia with propofol are different and significantly more coherent than the dex-spindle induced during sedation with dexmedetomidine.

The authors measured 64-channel electroencephalogram under dexmedetomidine (n = 9) and propofol (n = 8) in healthy volunteers, 18–36 years of age. In addition to standard preanesthesia assessment, a urine toxicology screen and urine pregnancy test for each female was performed. After adequate fasting of 8 h, the subjects were administered dexmedetomidine loading bolus 1 mcg/kg over 10 min followed by 0.7 mcg/kg/h (50 min) in dexmed-group. For propofol, the authors used a computer controlled infusion to target the effect site concentration of 0–5 mcg/ml and each concentration level was maintained for 14 min. The subjects were administered oxygen and respiration was assisted with bag-mask ventilation if apnea occurred. The monitoring included heart rate, electrocardiogram, oxygen saturation, respiration and expired carbon dioxide with capnography and blood pressure cuff (dexmedetomidine) or arterial line (propofol). Electroencephalography (EEG) was recorded using 64-channel Brain Vision Magnetic Resonance Imaging Plus System (Brain Products Munich, Germany) with a sampling rate of 1000 Hz (dexmedetomidine) and 5000 Hz (propofol), resolution 0.5 μV least significant bit and bandwidth 0.016–1000 Hz. Volunteers were instructed to close their eyes; and asked to respond by button presses when auditory stimuli were given to assess the level of consciousness.

An antialiasing filter was applied and EEG data was down-sampled to 250 Hz before analysis. First, 2 min EEG segments were selected during awake, eyes-closed baseline and then on the basis of behavioural response. For dexmedetomidine, the onset of unconsciousness was defined as first failed behavioral response that was followed by a series of at least five successive failures (10 min). For propofol, two states were identified; one where subjects had a nonzero probability of response to auditory stimuli and another where subjects were unconscious with a zero probability of response, propofol induced unconsciousness trough-max (TM) and propofol induced unconsciousness peak-max (PM) respectively. TM pattern marks the earliest part of propofol induced alterations in consciousness that were identified neurophysiologically to border the state of consciousness and unconsciousness. These neurophysiological pattern were maintained over changing propofol-effect site concentration 1–2 mcg/ml for TM and 3–5 mcg/ml for PM. Spectra and spectrograms were computed using the multitaper method, implemented in the Chronux toolbox.[1] Similarly coherence and coherogram between two frontal EEG electrodes F7 and F8 was estimated.

The spectrogram during dexmedetomidine-induced unconsciousness exhibited increased power across a frequency range of 2–15 Hz. Propofol induced unconsciousness was characterized by broadband (1–25 Hz) increased power during TM and increased power confined to slow, delta, and alpha frequency band during PM. The amplitude of slow oscillations during PM was approximately six-fold larger than during TM. EEG power was larger during dexmedetomidine-induced unconsciousness in a frequency range spanning slow delta, theta and alpha frequencies, while during propofol induced unconsciousness (TM) EEG power was larger in a frequency spanning beta and gamma frequencies ($P < 0.0005$). Spectrum during dexmedetomidine induced unconsciousness showed a clear dex-spindle peak at approximately 13 Hz. EEG power was larger across all frequencies between 0.1 and 40 Hz during propofol induced unconsciousness (TM) and the amplitude of slow oscillations and frontal alpha oscillations during PM were 3.9-fold larger than dex-spindles.

Dexmedetomidine induced unconsciousness was characterized by an increase in coherence across frequency range 1–15 Hz. Propofol induced unconsciousness was characterized by broad increase in coherence (1–25 Hz) and narrow band of alpha oscillations centered at 10 Hz during TM and PM respectively. During dexmedetomidine-induced unconsciousness coherence was larger in the delta, theta and spindle frequency bands with a coherent dex-spindle peak. Coherence was larger within beta/gamma frequency bands during propofol induced unconsciousness (TM); whereas during PM, coherence was significantly larger at frequencies surrounding the alpha oscillation peak and at a narrow gamma band.

The present analysis identifies differences in the power spectrum and coherence that likely relate to the specific underlying mechanisms and clinical properties of these drugs. At the neuronal levels, slow oscillations are associated with an alteration between ON states where neurons are able to fire and OFF states where neurons are silent. The authors speculated that propofol-induced slow oscillation and the duration of the associated OFF states could come from propofol's action at interneurons which would support larger slow waves and deeper levels of hyperpolarization required to sustain OFF states. Propofol's beta oscillations and its highly coherent frontal alpha oscillations appear to be generated by enhanced gamma-amino-butyric acid inhibition at cortical and thalamic interneurons.[2] Dexmedetomidine probably acts through endogenous nonrapid eye movement sleep circuits which may explain why dex-spindles appear similar to sleep-spindle.[3] The data suggest that propofol and dexmedetomidine have specific EEG signatures that can be computed, displayed in real time which would allow them to be readily interpreted by anaesthesiologists.

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Stroke is a leading cause of morbidity and mortality and the most significant source of disability in the United States. Recent trials have demonstrated that although outcomes for stroke patients are improving with aggressive medical therapy, the overall long term prognosis is poor. In the initial stenting and aggressive medical management for preventing recurrent stroke in intracranial stenosis (SAMMPRIS) trial, patients with a recent transient ischemic attack or stroke attributed to stenosis of 70–99% of the diameter of a major intracranial artery were randomized to aggressive