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 = 0.8134–0.9857) 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 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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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 spectrum 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

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