

Microstate quantifiers under total intravenous (TIVA) versus inhalational anaesthesia: an observational study in 48 patients undergoing major surgery

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Introduction

EEG microstates describe the brain's resting activity as a sequence of quasi-stable scalp topographies (canonical maps A–D)¹, characterised by their duration, occurrence, coverage and global field power (GFP). General anaesthesia profoundly reorganises these dynamics, and at deep anaesthetic levels the EEG fragments into an alternating suppression-burst pattern². Prior work in this surgical cohort showed that microstate quantifiers follow distinct trajectories over anaesthesia time in patients who do or do not develop postoperative delirium (POD), most markedly during suppression EEG³. Whether total intravenous (propofol-based) anaesthesia (TIVA) and inhalational anaesthesia shape these microstate dynamics differently over prolonged anaesthesia remains poorly characterised. The aim of this study was to examine how microstate quantifiers evolve with anaesthesia duration, separately within suppression and burst phases, and to test whether these time-courses differ between anaesthesia types and delirium status.

Methods

This observational study is a subsample of an original study with 73 patients³. Of these, 48 patients exhibited a suppression–burst pattern on EEG and were included in the present analysis. Among the 48 included patients undergoing major surgery, 27 (56%) received inhalational anaesthesia and 21 (44%) received TIVA. Sixteen (33%) patients developed POD. EEG was segmented into short artefact-free suppression or burst epochs and consecutive suppression–burst epochs formed 6,570 analysable pairs (those with both phases present and suppression preceding burst; aggregated from 16,121 epochs). For each epoch, the mean time since induction of anaesthesia (anaesthesia duration) was available. The four microstate maps were quantified for duration, occurrence, coverage and GFP. Linear mixed-effects models (REML) were fitted separately per microstate, with the microstate quantifier as outcome and a full *anaesthesia duration* × *epoch (burst/suppression)* × *anaesthesia type* × *delirium* fixed-effects structure. The random-effects structure was selected per model from a patient-level random intercept and, where supported, random slopes for anaesthesia duration and epoch; all models retained the richest structure (random intercept plus both slopes). The *anaesthesia duration* × *anaesthesia type* term tested the primary hypothesis that the two anaesthetic regimes drive divergent temporal trajectories. Within-pair terms carry denominator df ≈ 12,970, whereas between-patient effects (anaesthesia type, its interaction with delirium) are tested at the patient level (df = 44).

Results

As shown in Table 1, the two anaesthesia groups did not differ significantly in age, sex, physical status (ASA class), surgical site, total anaesthesia duration and POD development (all *p* > .2).

Table 1. Cohort characteristics by anaesthesia type.

Characteristic	TIVA (n=21)	Inhalation (n=27)	p
Age, years (mean ± SD)	69.7 ± 5.5	72.0 ± 7.0	0.24
Male sex, n (%)	10 (47.6)	17 (63.0)	0.38
ASA-PS III–IV, n (%)	11 (52.4)	16 (59.3)	0.77
Intra-abdom. / intra-thoracic surgery, n (%)	9 (42.9)	7 (25.9)	0.24
Total anaesthesia duration, min (median [IQR])	205 [105]	210 [79]	0.51
Postoperative delirium, n (%)	6 (28.6)	10 (37.0)	0.57

Age: independent-samples *t*-test. Total anaesthesia duration: Mann–Whitney *U* test. Categorical variables (ASA-PS III–IV vs I–II; surgical site intra-abdominal/intrathoracic vs other; postoperative delirium yes vs no): Fisher's exact test. TIVA= total intravenous anaesthesia; ASA-PS= American Society of Anesthesiologists Physical Status; SD= standard deviation; IQR= interquartile range; n= number.

GFP showed a consistent divergence between anaesthesia types over prolonged anaesthesia duration in all four microstates. The anaesthesia duration × anaesthesia type interaction was significant throughout ($F(1, \sim 12,970) = 10.0\text{--}13.5$, all *p* ≤ .002), and the divergence was concentrated in the burst phase, as indicated by a strong anaesthesia duration × epoch × anaesthesia type interaction ($F(1, \sim 12,970) = 79\text{--}139$, all *p* < .0001); the between-patient main effect of anaesthesia type was not significant ($F(1, 44) = 1.3\text{--}3.4$, all *p* > .07). The divergence was largely confined to the burst phase in non-delirious patients, where inhalational GFP remained high while TIVA GFP fell markedly with anaesthesia duration; suppression-phase GFP stayed uniformly low (Figure 1). The four-way *anaesthesia duration* × *phase* × *anaesthesia type* × *delirium* interaction was significant in every microstate ($F(1, \sim 12,970) = 25\text{--}45$, all *p* < .0001), indicating the temporal divergence itself depended on both phase and delirium. **Microstate duration** displayed a more selective, microstate-specific pattern. In contrast to GFP, the anaesthesia duration × anaesthesia type interaction was not significant in any microstate ($F(1, \sim 12,970) = 0.1\text{--}1.3$, all *p* > .26); the anaesthesia-type difference in duration instead manifested as a phase-dependent contrast (epoch × anaesthesia type significant for B, C and D: $F(1, \sim 12,970) = 5.5\text{--}6.2$, *p* = .013–.019).

and, for microstates C and D, a between-patient main effect of anaesthesia type ($F(1, 44) = 7.9$ and 11.0 , $p = .007$ and $.002$). During bursts, microstate durations tended to be longer under inhalational than TIVA anaesthesia, most pronounced for microstate C and D, while microstate B showed no anaesthesia-type divergence (Figure 2). A delirium-dependent modulation was evident only for microstate C, where the four-way *anaesthesia duration* $\times$ *phase* $\times$ *anaesthesia type* $\times$ *delirium* interaction was significant ($F(1, \sim 12,970) = 6.0$, $p = .014$).

Conclusions

Intravenous and inhalational anaesthesia shape EEG microstate dynamics differently, an effect that emerges predominantly during burst phases. For GFP this took the form of distinct temporal trajectories that diverged with anaesthesia duration across all microstates, whereas for microstate duration the anaesthesia-type difference was state-specific and expressed mainly as a phase-dependent level shift rather than a divergence over time. These differences interacted with delirium status, suggesting microstate measures may carry information about anaesthetic mechanism and vulnerability to postoperative delirium. Key limitations are the observational design, unequal and partly small subgroups (TIVA/POD $n = 6$), and a large sample of pairs, which inflates the significance of within-pair (time- and phase-related) effects.

Figures

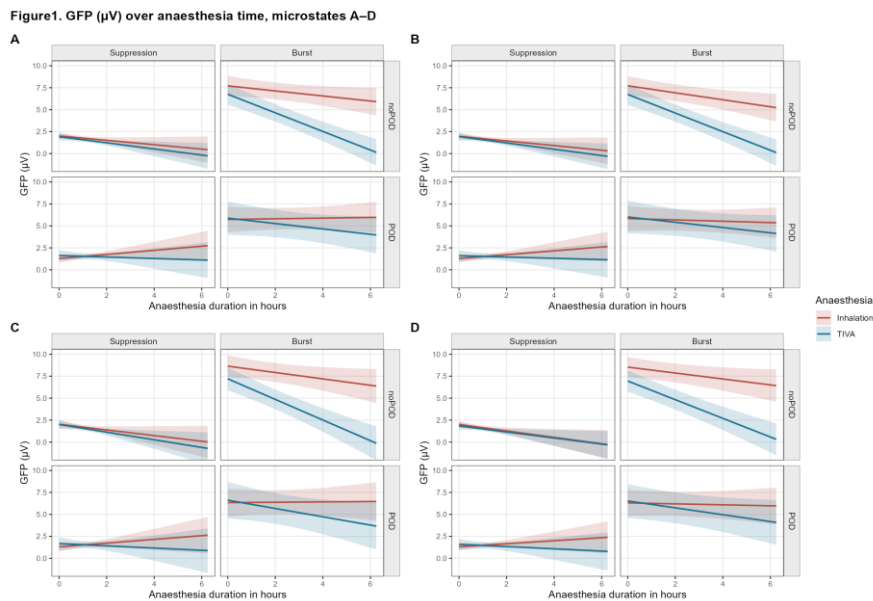


Figure 1. GFP trajectories over anaesthesia time, microstates A–D. Each block shows one microstate, faceted by phase (Suppression left, Burst right) and delirium (noPOD top, POD bottom). Lines are model-predicted GFP for TIVA (blue) and inhalational (red) anaesthesia with 95% CI ribbons. The TIVA–inhalation divergence during bursts in non-delirious patients is the dominant feature.

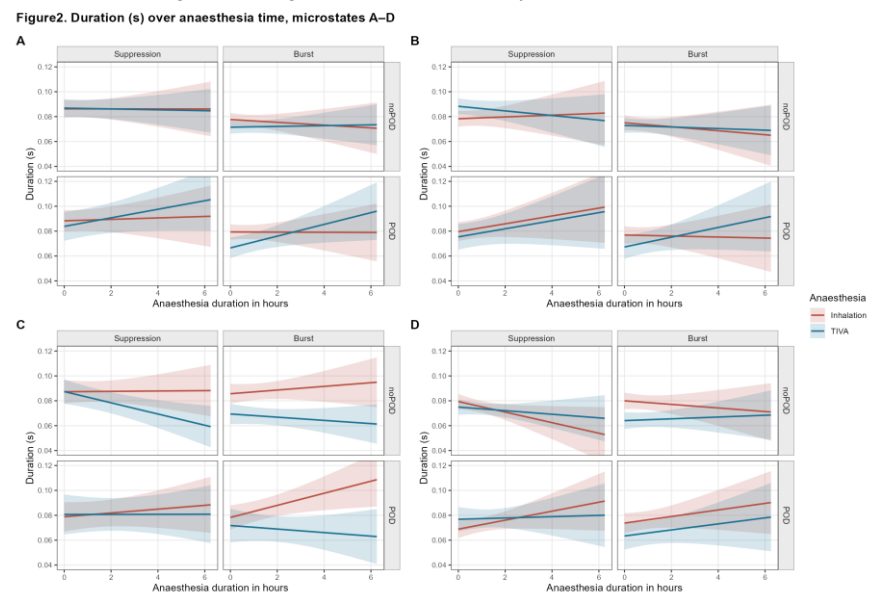


Figure 2. Microstate duration trajectories over anaesthesia time, microstates A–D. Layout as in Figure 1. Inhalational anaesthesia is associated with lengthening burst-phase microstate durations over time (notably C and D), while TIVA remains comparatively stable; microstate B shows no anaesthesia-type divergence.

References

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