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[2]Glasgow Royal Infirmary, Glasgow, UK

The Acute Physiology and Chronic Health Evaluation II (APACHE II) model is widely used for in-hospital mortality prediction in ICUs. There is a suspicion that it poorly predicts mortality in patients with COVID pneumonia.

Prospectively collected data from January 2019 to June 2021 from two Scottish tertiary ICUs (Queen Elizabeth University Hospital [QEUH] and Glasgow Royal Infirmary [GRI]) were used in a retrospective study. Data from 3054 patients, including 214 with a diagnosis of COVID pneumonia, were suitable for calculation of APACHE II scores. Performance of APACHE II in patients with COVID pneumonia was assessed in terms of discrimination and calibration. The data set from the QEUH was divided into a training set ($n$=843) and a test set ($n$=561). The APACHE II model was recalibrated on the training set using logistic regression modelling to generate ICU mortality predictions, creating new diagnostic coefficients for COVID pneumonia and eight other diagnoses from the original APACHE II diagnostic categories. Patients admitted to ICU for a reason outside these categories were combined into an 'other' category. Discrimination and calibration of the original APACHE model and the recalibrated model in the test set were assessed using the concordance (C) statistic, mean absolute error between actual and predicted mortality, and Brier's score. Discrimination and calibration of the original APACHE model and the recalibrated model were then assessed in the GRI data set for external validation.

The APACHE II model showed poor discrimination and calibration for patients with COVID pneumonia with C statistic of 0.67 (0.56–0.77) and absolute error of 20.2%. Compared with the original APACHE II model, the recalibrated model had comparable discrimination (C statistic=0.85 [0.81–0.88] *vs* 0.86 [0.82–0.89], respectively) and improved calibration in the test set (mean absolute error=18.5% *vs* 5.8% and Brier's score=0.128 *vs* 0.119). In the external data set, compared with the original model, the recalibrated model had comparable discrimination (C statistic=0.91 [0.89–0.93] *vs* 0.90 [0.88–0.91]) and improved calibration (mean absolute error=10.6% *vs* 5.3% and Brier's score=0.081 *vs* 0.088).

Recalibrating the APACHE II model to create a new diagnostic category for COVID pneumonia improved the predictive accuracy for ICU mortality. A larger cohort is required to recalibrate the APACHE II model to include all original diagnostic categories and a new COVID pneumonia category.

## Brainwave viscosity in propofol anaesthesia

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Human EEG during propofol anaesthesia shows large-scale changes, including travelling slow waves.[1] Slow-wave saturation is a potentially individualised marker of loss of perception.[2] However, much remains unclear about the dynamics of slow waves. Iterated empirical mode decomposition (itEMD[3]) is a novel data-driven method for segregating data into physiologically relevant oscillatory modes. We used itEMD to identify spectral modes and their sources/sinks in propofol EEG.

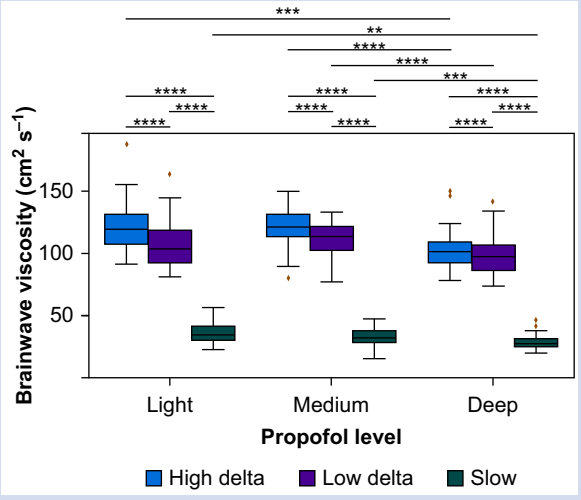


Fig 1. Brainwave viscosity in propofol anaesthesia. Only significant comparisons are shown with Bonferroni-corrected $P<10^{-N}$ as $N$ stars.

Viscosity is a physical quantity expressing the magnitude of resistance to flow. Considering travelling electric potentials in the brain as a flow, we extended the notion of viscosity to travelling brainwaves. Using this, we explored how brainwave viscosity changes in volunteer propofol anaesthesia.

Data came from an ultra-slow propofol infusion to 4 μg ml$^{-1}$ in 16 healthy volunteers.[2] In addition, 32-channel EEG referenced to FCz was acquired. Data were re-referenced to linked earlobes, down-sampled to 200 Hz, bandpass filtered 0.1–30 Hz, and decomposed using itEMD. Instantaneous amplitude from each channel was found using the Hilbert transform, interpolated onto a 15 × 15 scalp grid, and its velocity field calculated. Normalised velocity was computed for the data and for a surrogate data set with a pre-motor 2 Hz dipole. Time points with significant flow ($P$<0.01 against surrogate) were analysed. Singularities in the flow were identified as extremal contours in the velocity divergence. Multiplying area and divergence of sinks/sources, $V = A\nabla\cdot\vec{v}$ (m$^2$ s$^{-1}$) gave brainwave viscosity. Induction was divided into three equal concentration bins. Median viscosity across singularities for each level was extracted for each subject. Group-level differences were tested using a Bonferroni-corrected paired $t$-test.

Three low-frequency modes with different properties were identified in the data: high delta (~4 Hz), low delta (~2 Hz), and slow (<1 Hz). Waves originated from frontally dominant sources and ended in posteriorly dominant sinks. Brainwave viscosity was lowest for slow waves ($P<10^{-4}$) and decreased at high propofol doses ($P<10^{-3}$; Fig. 1).

We used a novel spectral decomposition method to identify important brain modes in propofol anaesthesia. Extending the concept of viscosity, we found that brainwave diffusion is frequency and concentration dependent. This potentially intrinsic property of the brain decreased in deep propofol sedation and was lowest for slow waves. We hypothesised that this may be because of network changes in the anaesthetised brain resulting in easier slow-wave spread.

## References

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## Continuous vital signs monitors: a replacement for traditional vital signs?


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Lightweight, wireless vital signs monitors offer the ability to continuously monitor ward-based patients, which could revolutionise identification of patient deterioration. Continuous vital signs require correlation to traditional vital signs measurements in acutely unwell inpatients if we are to understand how best to integrate continuous data into track-and-trigger systems. We report data collected during Continuous Signs Monitoring in COVID-19 Patients (COSMIC-19) (clinicaltrials.gov: NCT04581031), a feasibility study of continuous vital signs data recorded using wearable sensors in hospitalised patients with COVID-19. The data presented here explore data completeness from the continuous sensors and compare traditional and continuous vital signs.

Patients with COVID-19 were recruited at Manchester University NHS Foundation Trust. Participants wore medical-grade sensors/devices measuring HR, ventilatory frequency (VF), temperature, oxygen saturations ($SpO_2$), and BP. Data were streamed to a nearby Bluetooth monitor (Isansys Patient Status Engine, Oxfordshire, UK). Devices were worn for up to 20 days until hospital discharge or commencement of invasive ventilation. Participants were free to remove the devices. Clinicians were blinded to the continuous vital signs. Traditional vital signs were extracted from each participant's electronic medical record (ward and ICU) and compared with continuous monitoring using Bland–Altman plots.

We recorded 3881 h of vital signs from 43 participants (median=91 [inter-quartile range {IQR}: 27–141] h per participant) of whom seven (16%) required critical care.

The devices recorded 82 [IQR: 64–94]% of expected HR, 96 [IQR: 72–99]% of expected temperature, and 63 [IQR: 44–81]% of expected $SpO_2$ readings. There were 3 [IQR: 0–15] acceptable BP readings per patient.

The 95% limits of agreement (LOA) between continuous and traditional vital signs were −24 to 26 bpm on the ward and −15 to 14 bpm in the ICU for HR. For VF, the LOA were −11 to 11 $min^{-1}$ on the ward and −15 to 26 $min^{-1}$ in the ICU. Traditional VF measurements were narrowly distributed compared with continuous VF measurements (Fig. 2a), with poor agreement between traditional and continuous VF in the ICU at high VF (Fig. 2b).

We observed poor agreement between absolute values from continuous and traditional vital signs. This may reflect human sampling bias in traditional measurements and limitations of continuous measurement technology at high VFs. Some types of wearable device yielded more data than others. The BP measurements were too sparse for statistical significance. Arguably neither traditional nor continuous vital signs are a 'gold standard'. This implies that the two should not be used interchangeably in existing track-and-trigger systems. New early warning scores or novel markers of deterioration may need to be devised to incorporate data from continuous sensors.

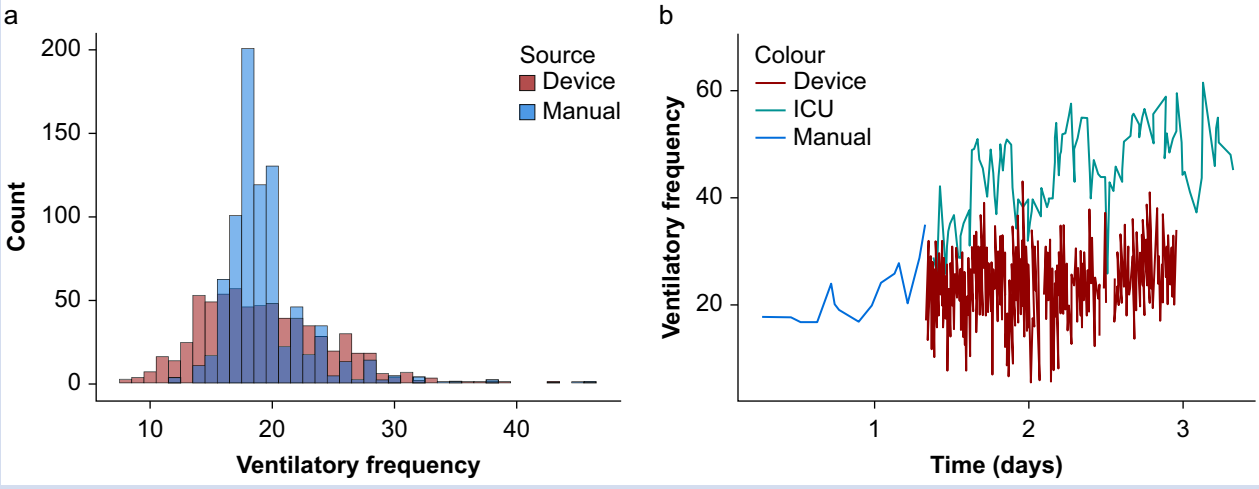


Fig 2. (a) Comparison of ventilatory frequencies measured manually and with the Isansys device across all patients; *n*=43. (b) Example patient in the ICU with high ventilatory frequency.