Therefore, they were not exposed to CO. In the subsequent work, we fixed the FGF:Ve at 0.5 (low-flow anaesthesia, LFA) for the first hour and then set FGF:Ve equal to 1 (high-flow anaesthesia) for the second hour. LFA resulted in an increase in exhaled and inspired CO, while HFA resulted in a plateau in exhaled CO and a decrease in inspired CO. With this study design, all patients were exposed to CO during an hour of LFA and the youngest patients experienced an increase in COHb. Furthermore, in the first study, we utilized a conventional carbon dioxide absorbent while in the follow-up work, we used an absorbent lacking strong metal alkali. Thus, endogenous and exogenous sources may have accounted for CO exposure in the first study, while exhaled endogenous CO could be the only source of the CO detected in the second study. We hope this clarifies the differences observed in these two distinct, but related studies.

In conclusion, we believe that the take-home messages of both of these works are: (i) CO is detectable within the anaesthesia breathing circuit, especially during LFA. (ii) LFA permits re-breathing of exhaled exogenous CO. (iii) CO re-breathing during LFA results in an increase in COHb, especially in the youngest children indicating CO exposure. The major question to consider next is the safety of such subclinical CO exposure.

Declaration of interest
None declared.

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Optimized method for correct left-sided central venous catheter placement under electrocardiographic guidance

Editor—The study by Kremser and colleagues1 to determine the optimized method for correct left-sided central venous catheter placement under electrocardiographic guidance makes interesting reading. Among other things, it highlights a different approach to improving the safety of central venous catheter insertion from that which we have adopted in the UK.

The authors report that they used three established thoracocervical puncture sites for the purpose of central venous catheterization. These are the internal jugular, subclavian, and innominate (brachiocephalic) veins. I wish to clarify the latter as I have not worked anywhere in the UK where the brachiocephalic vein is used as a central venous access site and I had great difficulty picturing where exactly the authors performed the needle puncture.

The anatomists describe the brachiocephalic vein in adults as commencing deep to the sternoclavicular junction on both sides. The bulk of the brachiocephalic vein therefore lies behind the body of the sternum. Performing a needle puncture of the brachiocephalic vein implies that the operator must either approach through bone or that the needle is introduced between the clavicle and the first rib close to the sternal margin aiming to pierce the vein behind the body of the sternum. It does not appear that the authors used either of these methods. There are various reports and studies on a supraclavicular approach to the subclavian vein. Is this perhaps what the authors meant?

A supraclavicular approach to the subclavian vein has been described in which the needle is introduced 1 cm lateral to the lateral head of the sternocleidomastoid muscle and 1 cm posterior to the clavicle at 45° and aiming towards the contralateral nipple.2 The authors make a case for the relative merits of this approach compared with the infraclavicular approach to the subclavian vein. Similar landmarks were used in a prospective study on mechanically ventilated patients.3 Two separate groups of investigators chose to modify this needle entry point. After three-dimensional computed tomography on normal anatomy, a modified angle of needle puncture during a supraclavicular approach was then tested on 60 patients requiring central venous line insertion.4 Another group of investigators tested their approach on cadavers before using it on patients.5 These studies all have the unspoken consensus that the access point was the subclavian vein. If this or a similar entry point is what was used by the authors, then I suggest that a supraclavicular approach to the subclavian vein is a more anatomically correct description.

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Optimizing cerebral oxygenation in anaesthetized patients with carotid artery stenosis: the influence of inspired oxygen fraction

Editor—Carotid artery stenosis is a clear herald of poor neurological outcome and is associated with postoperative stroke, delirium,1 and lower regional cerebral oxygenation (rSO2) as measured by near-infrared spectroscopy (NIRS).2 A decrease in rSO2 of 10% from baseline or a decrease below an absolute value of 50% is associated with cerebral ischaemia.3 Increases in cerebral oxygenation have been demonstrated by increasing inspired oxygen fraction (FIO2) in patients without vascular disease4 and during carotid endarterectomy (CEA).5 In our previous study,5 we analysed cerebral oximetry data obtained after the application of the carotid cross-clamp. Here we analysed previously unpublished data obtained before carotid cross-clamp placement in the same patient group to test the hypothesis that increasing FIO2 results in a significant improvement in cerebral oxygenation for anaesthetized patients with carotid artery stenosis.

After approval by the Institutional Review Board of the University of Michigan, Ann Arbor, written informed consent was obtained from patients presenting for CEA on the day of surgery. Cerebral oxygenation was measured using the INVOS 5100B monitor (formerly Somanetics Corporation, Troy, MI, USA). Patients received a standardized i.v. induction and maintenance of inhaled anaesthesia as previously described.6 Phenylephrine (200 µg ml−1) was titrated by infusion to maintain a stable arterial pressure between 100 and 125% baseline.

After tracheal intubation and patient positioning, FIO2 was adjusted first at 100%, then 30%. End-tidal carbon dioxide (PETCO2) was held in the range 5.3–6.0 kPa. After ≥5 min at each FIO2, rSO2 was recorded as a ‘snap-shot’ at the time of arterial blood gas analysis. A mean value for rSO2 from both sides was calculated. A related sample Wilcoxon signed-rank test was used to evaluate the change in rSO2, with each patient acting as their own control. P-values of <0.05 were considered statistically significant.

To standardize the assessment of the degree of carotid artery stenosis, we excluded two patients recruited for our original study5 who did not have their abnormality demonstrated with carotid Doppler ultrasonography performed at our institution. Analysis was completed for the remaining 18 patients. The average baseline rSO2, measured with patients awake breathing room air, varied between 36% and 71%. There was no significant difference between rSO2 measured on operative and non-operative sides at baseline or at either study point. One patient received N2O. Seventeen patients required phenylephrine by infusion. rSO2 was 8% higher when 100% O2 was delivered compared with 30% (median 69, IQR 58–82 vs median 61, IQR 57–72, P≤0.001) (Fig. 1). Intended increments in the partial pressure of oxygen in arterial blood were statistically significant (52.5

References

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