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doi: 10.1016/j.bja.2020.12.025 Advance Access Publication Date: 22 January 2021 © 2020 British Journal of Anaesthesia. Published by Elsevier Ltd. All rights reserved.

Climate impacts of anaesthesia

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Keywords: anaesthesia; carbon dioxide; climate change; environment; global warming potential

Editor—We congratulate McGain and colleagues¹ on a thorough review of environmental sustainability in anaesthesia and critical care. We wish to place the discussion of volatile anaesthetic agents within the science of climate change, and argue that a move away from the use of these agents cannot be justified based solely on their global warming potential (GWP).

The authors state that the atmospheric concentration of desflurane is increasing and that, because of its high GWP, this is a cause for concern.² Although levels are indeed rising, the concentrations of volatile anaesthetic agents, in comparison with the major greenhouse gases, are exceptionally small. Furthermore, their lifetimes are short and their impacts on Earth's energy budget (i.e. radiative forcing) are minute (see

Table 1 based on Vollmer and colleagues, 2 Hodnebrog and colleagues, 3 and IPCC. 4

Much has been made of the high GWP of volatile anaesthetic gases,² but this is deeply misleading. Global warming potential was designed for multi-gas climate policies (such as the Kyoto Protocol), where emissions of different compounds need to be placed on a common scale to aid international agreements. It has subsequently been taken up very widely as a simple proxy for the climate impact of a greenhouse gas and for converting emissions of that gas to equivalent carbon dioxide (CO₂) emissions.

This is problematic in several ways. Global warming potential represents the time-integrated radiative forcing (usually over 100 yr) attributable to a single burst of a gas, a pulse

Table 1 Atmospheric concentrations, lifetimes, and radiative forcing values for the three main greenhouse gases and the three main volatile anaesthetic agents.^{2–4} Radiative forcing (a change in Earth's energy budget)=radiative efficiency (W m⁻² ppb⁻¹) multiplied by atmospheric concentration, based on a radiative efficiency of 0.4 W m⁻² ppb⁻¹ for volatile anaesthetic agents.^{2–4} Thus, the percentage contribution of volatile anaesthetic agents, compared with the radiative effect that results from anthropogenic CO₂ emissions=(0.00021/1.68)*100, that is, 0.01%. Radiative forcing is the fundamental driver of climate change, not GWP. It avoids the issue of varying lifetimes, which confounds GWP (and its derivative, CO₂ equivalence), and depends only on the present-day accumulation Global Monitoring Laboratory. ¹Sum of atmospheric concentrations for sevoflurane/desflurane/isoflurane). CO₂, carbon dioxide; GWP, global warming potential.

Gas	Atmospheric concentration (parts per trillion)	Atmospheric lifetime	Radiative forcing (W m ⁻²)	
CO ₂ Methane Nitrous oxide Sevoflurane Desflurane Isoflurane Total volatile anaesthetics [†]	411 000 000* 1 870 000* 323 000* 0.13 ² 0.30 ² 0.097 ² 0.53	Centuries-millennia ⁴ 12.4 yr ⁴ 121 yr ⁴ 1.1 yr ² 14 yr ² 3.2 yr ² —	$1.68^4 \\ 0.97^4 \\ 0.17^4 \\ 0.00005 \\ 0.00014 \\ 0.00004 \\ 0.00021$	The three main volatile agents contribute only 0.01% of the climate effect that results from the increases in CO ₂ attributable to human activity.

emission, scaled by the GWP from a pulse emission of the same mass of CO₂. However, GWP does not properly discriminate between the climate effects of long-lived climate pollutants, such as CO₂, and short-lived climate pollutants, such as volatile anaesthetic gases.⁵ Although a strong greenhouse gas with a short lifetime could have the same GWP as a weaker greenhouse gas with a longer lifetime, identical pulse emissions (in mass terms) of the two gases would cause very different impacts on global surface temperature change. This is because global warming at a given time is largely determined by the cumulative total emissions of long-lived climate pollutants because of the thermal inertia of the climate system.4,5 This is why radiative forcing based on current concentrations (Table 1) is a fairer comparator of the potential impacts of different gases on the climate system, and why it is used by the IPCC.⁴ It avoids the issue of varying lifetimes, which confounds GWP (and its derivative, CO₂ equivalence), and depends only on the present-day accumulation of anthropogenic greenhouse gases as measured by atmospheric concentrations.

Second, the use of GWP sidesteps all the complexity of translating greenhouse gas radiative forcing into global and regional climate change that is fundamental to making decisions on what to do. Understanding the climate impacts of greenhouse gases is immensely complex, even for a simple gas like CO_2 that is abundant, long-lived, well mixed, and chemically inactive; for volatile anaesthetic gases, this is further compounded by their short lifetime and non-uniform spatial distribution.³

The sensitivity of the climate system to perturbations in radiative forcing involves multiple feedback mechanisms. These include changes in clouds, increases in water vapour concentrations (itself a greenhouse gas), increases in the surface reflection of the sun's energy back to space from loss of snow and sea ice, and heat uptake by the oceans.⁶ In fact, the oceans absorb around 90% of the excess energy trapped by greenhouse gases. However, their large thermal capacity means that their surface temperatures respond very slowly, and this fundamentally dictates the pace of climate change. Consequently, greenhouse gas radiative forcing needs to be large enough, and persistent enough, to be manifested in higher surface temperatures, which can then lead to further changes in Earth's climate. Quite simply, although per molecule the volatile agents are far more potent greenhouse gases than CO₂, there are simply not enough molecules present, nor do they reside long enough, to cause an appreciable radiative forcing, and hence climate effect (Table 1).

The translation of GWPs into CO₂-equivalent values is also frequently used to make comparisons with, for example, vehicle and aviation emissions, but this is again deeply misleading for the reasons given previously. Although mathematically correct, a pulse emission of 1 kg of desflurane does not equal the climate impacts of a pulse emission of 2540 kg of CO₂. In other words, the emission of a given anaesthetic cannot be 'equivalent to' a certain number/mileage of car journeys or equated with a certain emission of CO_2 .⁷

Calls have been made for the unilateral 'abandonment of volatile anaesthesia'⁸ and that 'anaesthetics with a lower carbon footprint are quick wins that should be implemented now'.⁹ This rhetoric is misleading on several levels. First, anaesthetic gases do not have a directly equivalent carbon footprint based on GWP, for all the reasons we have stated previously. Second, the impact on the climate system of these

gases is markedly different from CO_2 because of their short lifetimes and exceptionally low accumulations.

Although life-cycle analysis of anaesthetic agents may appear to favour i.v. anaesthesia, these calculations have again been made using misplaced CO_2 equivalence.¹⁰ Thus, moving away from inhalational anaesthesia to TIVA may actually increase the addition of long-lived carbon to the atmosphere because of the vast quantity of plastic required.¹¹

The story for nitrous oxide (N₂O) is slightly different, but still needs to be placed in context. Nitrous oxide is the third most important well-mixed greenhouse gas contributing to radiative forcing (Table 1).⁴ It has both natural sources (60%) and anthropogenic emissions (40%; e.g. fertiliser use). The contribution to the latter from healthcare is not easy to quantify, and it is likely that the majority of emissions arise from outside the operating theatre (maternity and emergency departments; prehospital settings). Thus, it has been estimated that the use of N₂O in anaesthesia represents only 0.1% of the climate effect attributable to anthropogenic increases in CO_2 .¹² Quantification and reduction of healthcare-related N₂O emissions would be advantageous, but should not detract from the 'big picture'.

As stated by Shine¹² in 2010, and reinforced by our commentary, volatile anaesthetics as a whole make a minute contribution to greenhouse gas radiative forcing; only 0.01-0.02% of the radiative effect that results from the increases in CO₂ as a result of human activity (Table 1). Thus, we would argue that climate change should not be invoked as the driving factor for a change in anaesthetic technique.

In conclusion, as we strive towards net zero, we must direct our efforts towards the 'big wins', not the apparent 'quick wins'. We should remain vigilant in our use of anaesthetic drugs and equipment, but we must keep this in perspective with the aggressive reduction of true CO_2 emissions in both our personal and professional lives. Finally, the impacts of climate change on human health and health services are likely to be profound. It behoves us all to engage in a much richer conversation between scientists and practitioners across the medical and climate science domains.

Declarations of interest

The authors declare that they have no conflict of interest.

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doi: 10.1016/j.bja.2021.03.004

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Sphenopalatine ganglion block for the treatment of post-dural puncture headache in paediatric patients

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Keywords: epidural blood patch; headache; paediatric; pain management; post-dural puncture headache; sphenopalatine ganglion block

Editor—Lumbar puncture is a common clinical procedure, widely used in the diagnosis of CNS infections in children. post-dural puncture headache after lumbar puncture is a relatively frequent complication with a reported incidence rate of 5–6% in paediatric populations.¹ Sphenopalatine ganglion block for the treatment of post-dural puncture headache is minimally invasive, easy to perform and does not appear to be associated with significant adverse events, therefore we suggest it could be considered as a first-line treatment for post-dural puncture headache in children.

Autologous epidural blood patch is a well described treatment of post-dural puncture headache, typically after a trial of conservative therapy has failed. While efficacious in children,² epidural blood patch is resource demanding, invasive, and painful. Therefore, children are often anaesthetised for the procedure. Rare, but severe complications, such as meningitis and nerve damage, are associated with epidural blood patch in adults,^{3,4} but such complications are not well described in children.

Dural puncture can decrease CSF volume, resulting in a compensatory intracranial vasodilation. Presumably, postdural puncture headache develops when uncontrolled vasodilation remains after the decrease in CSF volume has been adjusted. Regulation is mediated by parasympathetic activity in the sphenopalatine ganglion.⁵ Sphenopalatine ganglion block by a transnasal approach has been reported to be effective in adults with post-dural puncture headache,^{6,7} and a recent trial showed a 50% reduction in epidural blood patch rates when using a sphenopalatine ganglion block.⁸

To our knowledge, there is only one report of sphenopalatine ganglion block for post-dural puncture headache in children. The report describes a 12-yr-old boy where a successful sphenopalatine ganglion block was performed with sedation in the operating theatre.⁹ Here we describe an 8-yr-old boy with severe symptoms of post-dural puncture headache treated at the bedside with a sphenopalatine ganglion block (reported with the consent of the mother). The patient presented with facial paresis and was admitted to the hospital with suspected Lyme disease, which was confirmed by lumbar puncture. Antibiotic treatment was initiated, and the patient was discharged the same day. Four days later the boy was readmitted to the hospital because of dehydration and severe malaise. He had suffered from orthostatic headache, nausea, and vomiting since discharge. He was unable to stand or sit upright without severe headache and nausea. When lying down symptoms were manageable, but he could not eat and drink sufficiently. The symptoms were compatible with post-dural puncture headache, which he was at increased risk of since the lumbar puncture had been difficult to perform, requiring several attempts with a 22 G sharp tip needle.¹⁰ Specialists recommended conservative treatment with bed rest, fluid therapy and clonidine while expecting spontaneous recovery, with an epidural blood patch considered if no remission occurred.