more diverse scientific impact and greater heterogeneity, such as in medicine. Compared with researchers in the physical sciences, researchers in medicine may have smaller numbers of researchers in each field, differing areas of research interest, differing citation practices, and varying levels of academic commitment. Bibliometrics can be measured with increasing ease due to electronic scientific citation databases, and benchmarking of research performance of individuals is valuable due to the increasing use of bibliometrics to assess those applying for funding or academic promotion. However, it may be more appropriate to apply bibliometrics to high achieving researchers in medicine rather than ‘young’ researchers with few publications, which is becoming more common. I would suggest that because of the ‘citation window’ of scientific publications and the known limitations of citation indices, a standardized timeframe should be introduced before bibliometrics are used to assess research performance of researchers in medicine, or at a minimum, for assessing researchers of a young academic age.

Hirsch advocated that ‘a single number can never give more than a rough approximation to an individual’s multifaceted profile’ and similarly bibliometrics should not be used exclusively to evaluate performance of researchers in medicine.

Declaration of interest
None declared.

J. D. O’Leary*
Cork, Ireland
*E-mail: j.d.oleary@umail.ucc.ie

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Reply from the authors
Editor—We thank Dr O’Leary for his interest in our paper. We would agree that neither a single number (such as the h-index) nor even a panel of bibliometrics as we presented in our paper can provide a complete description of research quality or potential. However, they do seem to co-associate with other markers of academic standing such as professorships and membership of journal editorial boards. Other markers of quality, such as grant income, are also problematic, partly due to the self-propagating nature of grant awards. Whatever the shortcomings, bibliometrics are used and it is important that the anaesthesia research community has some understanding of its current metrics. As Dr O’Leary correctly implies, the h-index and most other indices are time-dependent and therefore favour longer established researchers. We believe that research in this field should be explicit about the time frame studied. We therefore selected a recent publication window (2004–8) in an attempt to define contemporary rather than historical research output. Of course, this is still disadvantageous to very new researchers, but it does allow future studies in this field to compare like with like. Although there have been other attempts to correct for academic ‘age’ or output, none of them works particularly well at the very early stages of an academic career.

Increase in cerebral metabolites during induction of propofol anaesthesia

Editor—We performed microdialysis of cerebral interstitial metabolites during induction of propofol anaesthesia and tracheal intubation in a case series of patients undergoing asleep–awake–asleep brain tumour surgery. While it is generally assumed that propofol is associated with suppression of cerebral metabolism, we found an unexpected transient increase in cerebral metabolites in parallel with an increase in heart rate after tracheal intubation.

Three patients underwent awake brain surgery for brain tumour resection. Anaesthesia included propofol (4–8 mg kg⁻¹ h⁻¹) supplemented by remifentanil (0.1–1 μg kg⁻¹ h⁻¹) with mivacurium as a neuromuscular blocking agent. After craniotomy and opening of the dura, a microdialysis catheter (CMA, Stockholm, Sweden) was placed in white matter of normal appearance within the predefined tumour resection area. Microdialysis samples (0.5 μl min⁻¹ flow rate, 10–60 min intervals) were analysed for glucose, lactate, pyruvate, glycerol, and glutamate (CMA 600

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Blood samples were drawn for the determination of plasma metabolite concentrations. Craniotomy was subsequently followed by an awake phase for determination of the resection margins and reintubation during propofol anaesthesia for craniotomy closure.

Changes in glutamate, glucose, glycerol, lactate, and pyruvate concentrations during the awake and asleep period are represented in Figure 1. Induction of propofol anaesthesia is marked as an anaesthesia-induced reduction in heart rate (dotted line). In all patients, anaesthesia induction was followed by an immediate (Fig. 1a and b) or slow (Fig. 1c), but transient increase in glutamate, but this was not observed in the arterial blood samples (grey boxes in Fig. 1a-c). In the first patient, the transient increase in glutamate levels (Fig. 1a), whereas the second patient showed a decrease in glucose levels during the increase in glutamate (Fig. 1e). Glucose remained stable in the third patient (Fig. 1f; grey boxes represent plasma glucose levels). The elevation of glutamate was associated with an increase in glycerol (Fig. 1g-i) in all three patients. The increase in the lactate/pyruvate ratio that paralleled the increased glutamate levels in the first and second patients (Fig. 1j and k, respectively) was absent in the third patient (Fig. 1l; grey boxes represent plasma lactate concentrations).

Although it is assumed that general anaesthesia suppresses cerebral energy metabolism,12 we found a transient increase in cerebral metabolites after infusion of propofol and reintubation in the presence of a decrease in heart rate and mean arterial pressure. A similar increase in cerebral glutamate levels during extubation has been shown using cerebral microdialysis.3 There are a few possible explanations for this unexpected observation. Although anaesthetic agents primarily inhibit excitatory neurotransmission, others showed increases in blood plasma and cerebrospinal fluid glutamate levels during anaesthesia.4 Our findings might be explained by a temporary reduction in synaptic uptake of glutamate, but this should be further investigated in a larger group of patients. Secondly, the interstitial increase in cerebral metabolites could be induced by a stress response associated with induction of general anaesthesia and tracheal intubation due to an increase in

![Figure 1](https://academic.oup.com/bja/article/108/1/165/234889/166)

**Fig 1** Representation of individual changes in cerebral glutamate, glucose, glycerol, and lactate/pyruvate during the awake and asleep phase. Induction of propofol anaesthesia was associated with a decrease in heart rate (dotted line), which was followed by tracheal intubation. The figure is further explained in the text.
catecholamines. We indeed observed an increase in heart rate during tracheal intubation, which might be representative for catecholamine release.

This case series shows that propofol anaesthesia and tracheal intubation may be associated with an increase in interstitial cerebral metabolite levels, which is in contrast to the general paradigm of suppression of brain metabolism by general anaesthesia. Our findings warrant further investigation of the relation between anaesthesia induction as a stressful event and brain metabolism in larger patient studies, thereby contributing to further optimization of anaesthetic strategies.

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S. M. Bossers
S. M. Peerdeman
P. Oedayrajsingh Varma
J. C. Baayen
P. C. De Witt Hamer
A. Schauer
S. A. Loer
C. Boer*
Amsterdam, The Netherlands

*E-mail: c.boer@vumc.nl


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**Anaemia tolerance: bridging with intravenous ferric carboxymaltose in a patient with acute post-haemorrhagic anaemia**

Editor—Perioperative anaemia has a high prevalence in surgical patients.1 Allogenic blood transfusion, although life-saving in severely bleeding patients, is associated with specific adverse effects which may ultimately lead to adverse outcome.2 Thus, alternative measures to restore physiological haemoglobin levels are appreciated.3 We report a case of treatment of posttraumatic anaemia i.v. ferric carboxymaltose (Verinject®) without blood transfusion. The 17-yr-old patient was involved in a motor vehicle accident and sustained left femoral shaft, right clavicular, and right metacarpal fractures. On admission, the patient had a haemoglobin concentration of initially 12.3 g dl⁻¹. After volume therapy and surgery, the haemoglobin was 5.0 g dl⁻¹ and on the second postoperative day was 4.2 g dl⁻¹. The patient and his mother refused blood transfusion. We decided to give 500 mg Verinject®. The patient was discharged from the intensive care unit on the third postoperative day (haemoglobin 5.3 g dl⁻¹) and left the hospital on the 19th postoperative day (haemoglobin 11.1 g dl⁻¹) (Fig. 1).

Verinject® has been investigated in patients with iron deficiency anaemia.4 5 Notably, the present case for the first time reports treatment with sole ferric carboxymaltose in a severely anaemic patient refusing allogenic blood transfusion. There were no signs of tissue hypoxia (lactate increase, ECG ST-elevation, etc.). Verinject® proved to be effective, safe, and considerably quick in raising haemoglobin concentration in this young patient. We report this case to generate discussion of the possibilities of ferric carboxymaltose as an alternative, supplement, or both for allogenic blood transfusion in young patients.

**Declaration of interest**

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C. W. Hönemann1*
D. Doll1
T. Kampmeier2
C. Ertmer2
O. Hagemann1
K. Hahnenkamp2
1 Vechta, Germany
2 Münster, Germany
E-mail: christian.hoenemann@kk-am.de