BJA
British Journal of Anaesthesia

Volume 110, Number S1, June 2013

British Journal of Anaesthesia **110** (S1): i1–i2 (2013)
doi:10.1093/bja/aet195

EDITORIAL

Special issue on anaesthetic neurotoxicity and neuroplasticity

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A group of experts in anaesthetic neuropharmacology and neurotoxicity convened on June 14–15, 2012 at Schloss Arenberg in Salzburg, Austria (see cover image) for the *BJA* Salzburg Seminar on Anaesthetic Neurotoxicity and Neuroplasticity. This focused workshop, sponsored by the *British Journal of Anaesthesia*, was organized to review and critically assess currently available evidence from animal and human studies, and to consider the direction of future research. The seminar was organized and co-directed by Hugh Hemmings of New York and Vesna Jevtovic-Todorovic of Charlottesville. They convened 20 other neuroscientists and anaesthesiologists from around the world for 2 days of intensive lectures, meetings, and discussions at Schloss Arenberg last summer. This resulted in a Special Article (summary statement) published simultaneously in the *British Journal of Anaesthesia*.[1] In addition, a collection of original submissions from meeting attendees and other papers submitted in response to a call for manuscripts are now collected in this Special Issue of the Journal.

This Special Issue represents a landmark development in the history of the Journal: it is published only electronically and all submissions are freely available to all readers online immediately. Publication of a themed issue of the *BJA* covering these rapidly evolving developments in anaesthesia research in an open access format on the *BJA* website (http://bja.oxfordjournals.org) demonstrates the Journal's commitment to facilitating anaesthesia research and education. The Special Issue includes two review articles and 11 original submissions. These cover three main areas of interest: developmental neurotoxicity, postoperative cognitive dysfunction (POCD) and delirium, and neuroprotection.

There are six papers and a review article in the Developmental Neurotoxicity section. Liu and colleagues[2] examine the role of the critical survival enzyme glycogen synthase kinase-3ß in ketamine-induced developmental neuroapoptosis. Lacoh and colleagues[3] show that general anaesthetics do not impair developmental expression of the cation-chloride cotransporter KCC2, which has been implicated in anaesthetic neurotoxicity. Boscolo and colleagues[4] report that the mitochondrial protectant pramipexole prevents long-term cognitive impairment after early anaesthesia exposure in rats. Ramage and colleagues[5] find differences between sevoflurane and isoflurane anaesthesia in long-term neurocognitive outcomes after early exposure in rats. Culley and colleagues[6] report that isoflurane affects the cytoskeleton but not survival or proliferation of astrocytes in rats, suggesting that its neurotoxic effects are not indirect. Creeley and colleagues[7] report propofol-induced apoptosis of neurones and oligodendrocytes in fetal and neonatal macaque monkey brain, indicating that propofol has similar toxicity to isoflurane in non-human primates. And finally, a review article by Sanders[8] updates current understanding of the impact of anaesthetics and surgery on neurodevelopment.

There are four papers in the section on POCD and Delirium. The first two are clinical studies. Steinmetz and colleagues[9] examine whether POCD is a risk factor for development of dementia. Radtke and colleagues[10] report that monitoring depth of anaesthesia decreases the rate of postoperative delirium but not of POCD. Lecker and colleagues[11] show that

potentiation of type A γ-aminobutyric acid ($GABA_A$) receptor activity by volatile anaesthetics is reduced by inverse agonists acting on a specific $GABA_A$ receptor subunit. And finally, Zhang and colleagues[12] show that activation of inflammatory signalling pathways by isoflurane and sevoflurane involving nuclear factor-κB increase interleukin-6, possibly contributing to neuroinflammation and cognitive dysfunction.

In contrast to their developmental neurotoxicity, general anaesthetics can be neuroprotective under certain conditions, as highlighted in two papers in the third section on Neuroprotection. Brücken and colleagues[13] show that the noble gas argon reduces neurological damage and preserves functional recovery after cardiac arrest in rats. In a review article, Bilotta and colleagues[14] consider the evidence for pharmacologic perioperative brain neuroprotection in randomized clinical trials.

We hope that this targeted collection of articles relevant to neuroanaesthesia and neuroscience provides the international anaesthesiology community with updated knowledge in important areas of anaesthesia research relevant both to researchers and to clinicians and their patients. We thank the authors of these excellent articles, and those involved in the preparation of this Special Issue, including Oxford University Press and Production Editor Hilary Lamb. The support of the Salzburg Stiftung of the American Austrian Foundation was also critical in the organization and direction of the seminar at Schloss Arenberg in Salzburg.

Declaration of interest

H.C.H. is an Editor for the *BJA* and for *Anesthesiology*; V.J.-T.: none.

Funding

H.C.H.: National Institutes of Health GM58055 and NS56315; V.J.-T.: National Institutes of Health/Eunice Kennedy Shriver National Institute of Child Health and Human Development HD 44517.

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