Anesthetic Neurotoxicity and Neuroplasticity

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Introduction

The BJA Salzburg Seminar on Anesthetic Neurotoxicity and Neuroplasticity was held on June 14–15, 2022, at Schloss Arenberg in Salzburg, Austria. It was attended by a group of professionals in anaesthetic neuropharmacology and neurotoxicity. The British Journal of Anesthesia organized this targeted workshop to review and evaluate the present body of research findings from both human and animal studies as well as to think about the future course of study. Hugh Hemmings of New York and Vesna Jevtovic-Todorovic of Charlottesville organized and co-directed the seminar. Last summer, at Schloss Arenberg, they gathered 20 additional neuroscientists and anesthesiologists from around the globe for two days of rigorous lectures, talks, and debates. A Special Article (summary statement) was as a result of this that was simultaneously published in the British Journal of Anesthesia. Last summer, at Schloss Arenberg, they gathered 20 additional neuroscientists and anesthesiologists from around the globe for two days of intensive lectures, meetings, and discussions.

A Special Article as a result was published concurrently in the British Journal of Anesthesia. This Special Issue of the Journal also includes a collection of original submissions from meeting participants and other papers submitted in response to a call for manuscripts. This Special Issue marks a turning point in the Journal's history because it is exclusively published online and all entries are made freely available to all readers right away.

The BJA's dedication to promoting anesthetic research and teaching is demonstrated by the publication of a thematic issue of the BJA on these quickly developing advancements in anesthetic research in an open access format on the BJA website (http://bja.oxfordjournals.org). Two review articles and eleven original entries make up the Special Issue. The three main topics covered here are neuroprotection, Postoperative Cognitive Dysfunction (POCD), and developmental neurotoxicity. The section on developmental neurotoxicity contains six publications and a review article. Glycogen synthase kinase-3ß, a vital survival enzyme, is investigated by Liu and colleagues in the context of ketamine-induced embryonic neuroapoptosis. General anesthetics do not affect the developmental expression of the cation-chloride cotransporter KCC2, which has been linked to anaesthetic neurotoxicity, according to research by Lacoh and colleagues [1].

Pramipexole, a mitochondrial protectant, protects long-term cognitive damage in rats exposed to early Anesthesia, according to Boscolo and colleagues research [2]. Sevoflurane and isoflurane Anesthesia have different effects on long-term neurocognitive results in rats when exposed early, according to Ramage and colleagues [3]. The fact that isoflurane affects the cytoskeleton but not the survival or proliferation of astrocytes in rats, according to Culley and colleagues, suggests that its neurotoxic effects are direct and not indirect [4]. According to Creeley and colleagues, propofol causes neurones and oligodendrocytes to die in the foetal and newborn macaque monkey brain, proving that it is equally hazardous to non-human primates as isoflurane. Finally, a review article by Sanders updated knowledge on how Anesthesia and surgery affect neurodevelopment [5, 6].

The section on POCD and Delirium contains four studies. The first two are medical research investigations. Steinmetz and colleagues investigate the possibility that POCD may be a risk factor for dementia development. According to Radtke and colleagues, monitoring the level of Anesthesia lowers the incidence of postoperative delirium but not POCD [7]. Lecker and colleagues demonstrate that inverse agonists acting on a particular GABAA receptor subunit inhibit the potentiation of type A g-Aminobutyric Acid (GABAA) receptor activity by volatile anesthetics [7,8]. Finally, Zhang and colleagues demonstrate that isoflurane and sevoflurane activation of inflammatory signaling pathways involving nuclear factor-kB increase interleukin-6, potentially causing neuro-inflammation and cognitive dysfunction.

General Anesthetics can be neuroprotective in specific circumstances, in contrast to their developmental neurotoxicity, as are two publications in the third part on neuroprotection. The noble gas argon, as demonstrated by Brucken and colleagues, lessens brain damage and preserves functional recovery in rats that have experienced cardiac arrest. In a review article, Bilotta and colleagues discuss the data from randomized clinical studies supporting pharmacologic perioperative brain neuroprotection [9].

We believe that this carefully chosen collection of articles on neuro-anesthesia and neuroscience will give the global anesthesiology community up-to-date information on crucial Anesthesia research topics that are vital to both researchers and clinicians and their patients. We acknowledge Oxford University Press and Production Editor Hilary Lamb for their contributions to the production of this Special Issue as well as the writers of these outstanding papers. In addition, the Salzburg seminar in Scholes Arnsberg benefited greatly from the assistance of the Salzburg Stiftung of the American Austrian Foundation.

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