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Delayed environmental enrichment reverses sevoflurane-induced memory impairment in rats

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Anesthesia given to immature rodents causes cognitive decline, raising the possibility that the same might be true for millions of children undergoing surgical procedures under general anesthesia each year. We tested the hypothesis that anesthesia-induced cognitive decline in rats is treatable. We also tested if anesthesia-induced cognitive de- cline is aggravated by tissue injury. Seven-day old rats underwent sevoflurane anesthesia (1 minimum alveolar concentration, 4 hr) with or with- out tail clamping. At 4 weeks, rats were randomized to environmental enrichment or normal housing. At 8 weeks rats underwent neurocognitive testing, which consisted of fear conditioning, spatial reference memory, and water maze- based memory consolidation tests, and interrogated working memory, short-term memory, and early long-term memory. Sevoflurane-treated rats had a greater escape latency when the delay between memory acquisition and memory retrieval was increased from 1 min to 1 hr, indicating that short-term memory was impaired. Delayed environmental enrichment reversed the effects of sevoflurane on short-term memory and generally improved many tested aspects of cognitive function, both in sevoflurane-treated and control animals. The performance of tail-clamped rats did not differ from those rats receiving anesthesia alone. Sevoflurane-induced cognitive decline in rats is treatable. Delayed environmental enrichment rescued the sevoflurane-induced impairment in short-term memory. Tissue injury did not worsen the anesthesia-induced memory impairment. These findings may have relevance to neonatal and pediatric anesthesia.

Biography

Laura May spent two years as lab manager of the Stratmann Lab at University of San Francisco, investigating the effects of anesthesia on neurogenesis in the rat brain. She is currently completing her fourth year of medical school at the University of Southern California.

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