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Brain circulation is autoregulated to provide a constant supply of nutrients despite changes in blood pressure, cellular metabolism, or oxygenation. Cerebral blood flow is regulated less well during anesthesia; however, the impact of anesthesia on the autoregulation of brain tissue oxygen tension (PO₂) has not been measured. To investigate the impact of isoflurane on cerebral autoregulation, we recorded PO₂ with oxygen sensitive electrodes in the somatosensory (whisker barrel) cortex of rabbits before and during anesthesia with isoflurane. Animals were implanted with chambers over the cortex in a sterile surgery. At least ten days after recovery from the surgical procedure, rabbits were restrained for the measurements but showed no signs of distress. After a period of electrode stabilization, PO₂ was recorded while the awake animal breathed air (baseline) followed by a 90 second episode of 100% oxygen (hyperoxia), during which time the brain PO₂ stabilized. For each episode, Δ PO₂, the hyperoxic PO₂ minus the baseline PO₂, was computed. Following several episodes of hyperoxia, the animal was anesthetized with 0.5% or 1.5% isoflurane, and the hyperoxic episodes were repeated. The anesthetic was then terminated and responses were again recorded in the awake animal. Brain PO₂ increased during hyperoxia in both awake and anesthetized rabbits, but under 1.5% isoflurane, the hyperoxic Δ PO₂ was significantly larger than during air breathing in all four rabbits studied. The responses during 1.5% isoflurane were 2.15 ± 0.6 (mean and SD) fold greater than when the rabbits were awake. The effect of 0.5% isoflurane was variable. These results indicate that the ability of the somatosensory cortex to regulate PO₂ in response to hyperoxia is worse during anesthesia, which may have implications for respiratory management during anesthesia.