

Dexmedetomidine cuts γ -aminobutyric acid receptor function

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(HealthDay)—Dexmedetomidine prevents excessive γ -aminobutyric acid type A receptor function after anesthesia, according to a study published online June 7 in *Anesthesiology*.

Dian-Shi Wang, M.D., Ph.D., from the University of Toronto, and colleagues studied injectable and inhaled anesthetic drugs (etomidate and sevoflurane) using cultured murine hippocampal neurons, cultured murine and human cortical astrocytes, and ex vivo murine hippocampal slices. Using electrophysiologic and biochemical methods, γ -

aminobutyric acid type A [receptor function](#) and cell-signaling pathways were studied; in addition, memory and problem-solving behaviors were studied.

The researchers found that [dexmedetomidine](#) reduced the etomidate-induced sustained increase in $\alpha 5$ γ -aminobutyric acid type A receptor cell-surface expression (etomidate: 146.4 ± 51.6 percent; etomidate + dexmedetomidine: 118.4 ± 39.1 percent of control). The persistent increase in tonic inhibitory current in hippocampal neurons was also reduced by dexmedetomidine (etomidate: 1.44 ± 0.33 pA/pF; etomidate + dexmedetomidine: 1.01 ± 0.45 pA/pF). A sevoflurane-induced increase in the tonic current was also prevented by dexmedetomidine. Astrocytes were stimulated by dexmedetomidine to release brain-derived neurotrophic factor, which reduced excessive $\alpha 5$ γ -aminobutyric acid type A receptor function in neurons. Dexmedetomidine also reduced post-anesthesia memory and problem-solving deficits.

"Dexmedetomidine prevented excessive $\alpha 5$ γ -aminobutyric [acid](#) type A receptor function after anesthesia," the authors write. "This novel $\alpha 2$ adrenergic receptor- and brain-derived neurotrophic factor-dependent pathway may be targeted to prevent delirium."

More information: [Abstract/Full Text \(subscription or payment may be required\)](#)

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