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Figure. (A) Axial fluid-attenuated inversion recovery (FLAIR) MRI reveals prominent signal hyperintensity within the medial temporal lobes. (B) In the corresponding diffusion-weighted image, the territory of acute ischemia (high signal intensity) is found to outline the hippocampus bilaterally. Thin rims of hyperintense signal are also present in the temporal poles and occipital cortices.

Carbon monoxide-mediated hippocampal injury

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A 48-year-old man was found unconscious after deliberate carbon monoxide poisoning and flurazepam overdose. His blood pressure was 119/75 mm Hg; his pulse was 102 beats/min, and his respiratory rate was 18 breaths/min. He was intubated, hospitalized, and given 100% normobaric oxygen for elevated blood carboxyhemoglobin (9.1%). He was arousable to voice and had preserved brainstem re-

flexes, but no spontaneous movements. Short-term memory loss became apparent when he used eye-blinks to communicate. Infarctions in bilateral hippocampi, temporal poles, occipital cortex (figure), globus pallidi, cerebellum, and brainstem were most consistent with carbon monoxide toxicity.^{1,2} Six months later he is ambulatory, with normal communication but residual anterograde amnesia.

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Neurology 2001;57;17

DOI 10.1212/WNL.57.1.17

This information is current as of July 10, 2001

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