

PubMed Results

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1. J Neurosurg. 1991 Nov;75(5):774-9.

Restoration of cerebrovascular responsiveness to hyperventilation by the oxygen radical scavenger n-acetylcysteine following experimental traumatic brain injury.[Ellis EF](#), [Dodson LY](#), [Police RJ](#).

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Abstract

Previous experiments have shown that, following experimental fluid-percussion brain injury, cyclo-oxygenase-dependent formation of oxygen radicals prevents arteriolar vasoconstriction in response to hyperventilation. The oxygen radical scavengers superoxide dismutase and catalase restore normal reactivity; however, they are not routinely available for clinical use. The present study tested whether n-acetylcysteine (Mucomyst), an agent currently available for acetaminophen toxicity, could be used as a radical scavenger to restore reactivity after brain injury. N-acetylcysteine (163 mg/kg) was given intraperitoneally prior to or 30 minutes after fluid-percussion brain injury (2.6 atm) in cats, and reactivity to hyperventilation was tested 1 hour after injury. The authors found either that pre- or postinjury administration led to normal reactivity. Additional experiments supported the hypothesis that n-acetylcysteine is an oxygen radical scavenger, since it reduced or prevented the free radical-dependent cerebral arteriolar dilation normally induced by the topical application of arachidonic acid or bradykinin. The mechanism by which n-acetylcysteine is effective in trauma may involve direct scavenging of radicals or stimulation of glutathione peroxidase activity. The results suggest that n-acetylcysteine may be useful for treatment of oxygen free radical-mediated brain injury.

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