

vide a critical resource for addressing these controversial questions and for evaluating existing and new single-drug and combination-drug therapies. The complex factors contributing to responsiveness to opioids demand sophisticated methods of analgesic drug design in order to advance the chemo-

therapy of pain. Opioids should not be withheld on the basis that the pain mechanism or any other factor precludes a favorable response.

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Diffuse Cerebral Infarction after Cardiac Arrest

TO THE EDITOR: In the Images in Clinical Medicine entitled “Fatal Cerebral Hypoxemia after Cardiac Arrest” (Feb. 27 issue),¹ the term “hypoxemia” is misused.² How can hypoxemia be cerebral, when the term refers to oxygen levels in the blood? More important, hypoxemia is a distinct insult from ischemia,^{3,4} and controlled experiments in animal models have shown that pure hypoxemia without heart stoppage does not cause brain necrosis.^{5,6} A better title for the image would have been “Widespread Brain Necrosis after Cardiac Arrest.” This title would not have perpetuated the myth that hypoxemia by itself causes necrotizing brain damage.

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DR. KELSEN REPLIES: I thank Dr. Auer for his interest in the images and his comment on the inappropriate use of the term “hypoxemia” in the title. On the basis of research by Dr. Auer and others, a clear distinction has been established between the terms “hypoxia” and “ischemia” and between their respective roles in cerebral infarction.^{1,2} I understand that the title could lead to the misinterpretation that hypoxemia caused the reported brain damage. This patient obviously had primary cerebral ischemia — as opposed to primary cerebral hypoxia — due to cardiac and circulatory arrest. The proposed title by Dr. Auer refers to a morphologic description of the cerebral injury. However, the dramatic changes seen on three consecutive computed tomographic (CT) scans rep-

resented developing edema and infarction but did not reflect widespread brain necrosis. Panel A of our image shows a CT scan obtained one hour after presentation, and Panel B shows a scan obtained four hours later — not eight hours later, as stated in the legend. The scan obtained at eight hours is shown in Figure 1. An alternative title that summarizes the underlying pathophysiological features of the case could be “Evolving Diffuse Cerebral Infarction after Cardiac Arrest.”

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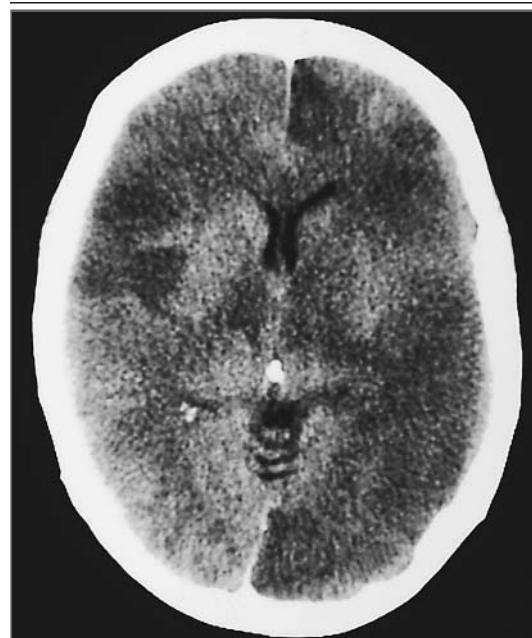


Figure 1. Computed Tomographic Scan of the Brain, Obtained Eight Hours after Cardiac Arrest, Showing Severe Edema and Infarction in the Anterior and Posterior Circulation of Both Hemispheres.