Dichloroacetate After Incomplete Ischemia

To the Editor:

We would like to comment on the article by Colohan and coworkers in the May-June 1986 issue of Stroke regarding the effect of dichloroacetate (DCA) after incomplete ischemia in rats. Using the same model, our evidence that pretreatment with DCA does not decrease the amount of lactic acid build-up in the brain during ischemia is in agreement with the findings of these authors. However, additional data of ours shows that treatment with DCA does result in lower lactate levels 30 minutes after the termination of ischemia.

We agree with these authors that even though pyruvate dehydrogenase enzyme complex (PDHC) in the brain might be activated by DCA during ischemia, the lack of oxygen is probably what limits the entry of pyruvate into the citric acid cycle. Having made this assumption, we measured cerebral cortical lactic acid levels 30 minutes after reperfusion in rats that had been treated with DCA 15 minutes prior to, immediately after, or 15 minutes after ischemia. Our results showed that cerebral lactate is near control levels 30 minutes after reperfusion in DCA-treated rats.

References
1. Colohan ART, Welsh FA, Miller ED, Kassel NF: The effect of dichloroacetate on brain lactate levels following incomplete ischemia in the hyperglycemic rat. Stroke 1986;17:525-528

The following letter is sent in response.

To the Editor:

Dichloroacetate is theoretically an attractive agent for treatment of cerebral ischemia. We were quite disappointed when the results of our studies failed to suggest a beneficial action. However, the additional studies cited by Drs. Dimlich and Barsan indicate that dichloroacetate may indeed be useful in the management of ischemic stroke. Our enthusiasm has been rekindled, and we agree completely that further studies should be conducted.

Very truly yours,
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Dichloroacetate after incomplete ischemia.
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