And the same "case series" observations can be spun off, not later or through "data dredging," but at the same time, to create new hypotheses subject to subsequent data collection. And as new hypotheses are generated, the research consortium is in a position to collect the relevant data with few of the usual delays in forming a research team.

By contrast, should the authors' proposals for research be adopted in their extreme form, each study presumably would be pursued using only one hypothesis, and any data collected not directly bearing on the hypothesis would be rigorously set aside. Research of this type, while agreeably pristine, would be expensive indeed and would greatly delay the promulgation of new research hypotheses. Many of the consortia who have labored long and hard to generate the most productivity for the research dollar would be chagrined to learn that subsidiary efforts may be of so little interest.

Raising the issue of expense as a reason to avoid future projects may prove counterproductive. As computers fall in price, rise in power, and get more friendly, many investigators may be tempted into the field. They will find sophisticated programs available, their displays of frequencies and time-based curves beguiling to the unwary along exactly the lines that worry the authors.

If the hostility toward the term "data bank" as reflected in the authors' letter is widespread, existing research groups are well-advised to change their names lest they suffer the "Sambo's effect." That restaurant chain was said to have been forced under by minority groups protesting the name, misconstruing what was the portmanteau of the two owners' first names for the (minority member) hero of the children's story. Thanks to the authors for calling to general attention the ambiguities inherent in the term "data bank." They may have spared many computer-based clinical investigators unwanted future agonies.

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Dichloroacetate After Incomplete Ischemia

To the Editor:

We would like to comment on the article by Colohan and coworkers1 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 ischemia2 is in agreement with the findings of these authors. However, additional data of ours shows that treatment with DCA does result in lower lactates 30 minutes after the termination of ischemia.2,3

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 (lactate) into the cytosolic acid cycle.1 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 fasted rats pretreated with 25 mg/kg DCA.3 In untreated ischemic rats this resolution takes at least 60 minutes after the start of reperfusion.5 When treated immediately or 15 minutes after ischemia, there likewise is a significantly faster amelioration of cerebral hyperlactatemia in DCA-treated ischemic rats when compared with untreated rats.3 Since these effects were achieved with a small dose of DCA (25 mg/kg) that was not effective in resolving systemic acidosis,1 we also agree with these authors5 and Evans,6 that the control of PDHC activity may be different in the brain than in other tissues.

Since most patients with cerebral ischemia will be fed and since high blood glucose correlates with poor physiological and neurological outcome from cerebral ischemia,2,4,5 we examined the effect of postischemic treatment with DCA in fed rats. Untreated rats exhibited mean cerebral lactates of 23 μM/g 30 minutes after ischemia. In contrast, the mean lactate level in DCA-treated rats 30 minutes after ischemia was 13 μM/g,2 significantly less than 18 μM/g that has been shown by other investigators to promote irreversible cell damage.6,12 Since brain ischemia commonly will affect nonfasted patients and since treatment will occur after, not prior to, ischemia, we believe these results have important clinical significance. We therefore urge the continued investigation of the use of DCA for the treatment of cerebral hyperlactatemia in ischemic events.

Sincerely,

Ruth W.V. Dimlich, Ph.D.
William G. Barsan, M.D.
University of Cincinnati
Cincinnati, Ohio

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 a theoretically 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.
R V Dimlich and W G Barsan

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