

critically ill patients. Theoretically, our data also suggest that when therapy for low  $[Ca^{++}]$  is initiated during low-output states, intravenous  $CaCl_2$  may have to be administered at rates as high as 1.5 mg/kg/min. This recommendation cannot be justified without close monitoring of  $[Ca^{++}]$  since absence of a constant relationship between changes in  $[Ca^{++}]$  and  $[Ca]$  is clearly demonstrated by the present report.

Our observations suggest, however, that improvement of hemodynamic function by isoproterenol infusion was instrumental in the restoration of normal  $[Ca^{++}]$ , since  $[Ca^{++}]$  rose to normal before the effectiveness of  $CaCl_2$  therapy was apparent.

Finally, increased neuromuscular irritability was not apparent in our patients despite the remarkably low  $[Ca^{++}]$ 's. The reason for its absence is unclear.

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### Erratum

An error appeared in the title of a recent article, "Humoral immunity to a metabolite of halothane, fluroxene, and enflurane" (*ANESTHESIOLOGY* 42:612-616, 1975). "Enflurane" should read "isoflurane," since the former does not metabolize to form trifluoroacetate. The error also appears in lines 2 and 10 of the abstract.

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