pression of isoelectric brain seem high (as do previous data from that laboratory), raising the possibility of extracerebral contamination of blood to the venous outflow in the experimental model. This possibility seems supported by the apparent pressure-dependent increase in flow when lidocaine was given, indicating either abolished autoregulation in the brain, or, more likely, perhaps (because why should autoregulation be disturbed?), extracerebral contamination. Second, the ATP values measured with—as well as without—lidocaine are a bit low, addressing the technique of tissue sampling and freezing being, perhaps, not fast enough to prevent some degree of ATP hydrolysis. Such implicit difficulties in technique become important when looking for, perhaps, only minor or no differences between one regimen and another, unless experiments are conducted with blinding and placebo. I suggest that the use of the terms “protective effect” and “detrimental effect” of lidocaine in the brain await further studies.

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REFERENCE


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In Reply.—Direct measurement of cerebral blood flow by diversion of the sagittal sinus blood flow of the dog, obliteration of the extracerebral veins communicating with the sinuses, and determination of the brain regions drained by the sinuses used in this study was first described in 1968,1 and has been the methodology used in the measurement of CBF in at least 45 published articles since that time. This measurement of CBF has been validated by two independent techniques.2,3 In several studies involving more than 60 dogs, the brains were examined for evidence of extracerebral contamination by post-mortem injection of the posterior sagittal sinus with blue vinyl acetate.4,5 No contamination by extracerebral structures was found.

The apparent pressure-dependent changes in cerebral blood flow reported in the article4 were, indeed, due to the loss of autoregulation during deep isoflurane anesthesia. This loss of autoregulation was readily apparent at concentrations greater than 2% end-expired isoflurane,4–6 especially when phenylephrine was used to support mean arterial pressure. The loss of autoregulation has also been reported for lower concentrations of isoflurane.7 Therefore, the observed cerebral blood flow changes are not due to contamination by extracranial vessels.

Normal values for cerebral metabolites for our laboratory were obtained from normal awake dogs in whom the stress response to immobilization, as manifested by increased catecholamines, cerebral blood flow, and cerebral metabolism, was blocked by sympathetic block-
agree with Dr. Astrup that lidocaine is a "good drug," but we want to emphasize that it may have detrimental effects in some situations.

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REFERENCES


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Differences in the Response of Humans and Dogs to Succinylcholine

To the Editor:—We were interested to read Professor Merin's comments on the duration of action of succinylcholine (SDC) in dogs compared with humans.1 Our findings indicate strongly that the prolonged relaxation experienced by dogs when treated with SDC, in a dose which would be appropriate for a human, can be attributed to differences between canine and human cholinesterase.

We measured plasma cholinesterase activity in 14 dogs of different breeds and both sexes. For each animal, we performed four measurements using as substrates propionylcholine,2 from which thiocholine is released and coupled with 5′-dithiobis (2-nitrobenzoic acid) (DTNB); butyrylthiocholine,3 again utilizing DTNB; benzoyl choline,4 relying on its absorption at 240 nm; and SDC,5 measuring choline by use of choline