Regional Anesthesia in Children with Epidermolysis Bullosa Dysfrophia

To the Editor.—In the recent case report 1 concerning management of epidermolysis bullosa dystrophica (EBD) with regional anesthesia, the authors state, “Although regional anesthesia has been suggested, its use has not been described for EBD, either as a supplement to or as the main anesthetic.” In fact, regional anesthesia in the EBD patient has been previously described in a case report of axillary block anesthesia, 2 done in much the same way as the authors suggest. Epidural and spinal techniques have also been described in previous publications. 2,3 At our institution, recently designated as a EBD center, we have reported a series of eight pediatric patients in which regional technique was used without any complications. 4 We recommend that a regional technique be viewed as a safe alternative to general anesthesia in this patient population.

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REFERENCES

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In Reply: We wish to thank Dr. Kelly for pointing out the article by Rowlingson and Rosenblum published in the journal of Regional Anesthesia. The case management reported in that article is indeed very similar to the management utilized in our patient. Unfortunately, the journal of Regional Anesthesia is not cited in the Index Medicus, and was, therefore, inadvertently overlooked in our literature search. The article by Broster et al. 1 was published after our article was accepted by ANESTHESIOLOGY, and we were not aware of this report.

Dr. Kelly's own article obviously not available to us, as it is listed as "in press." We are aware of Kelly's group and its efforts on behalf of EBD patients. At the request of one of his authors (KOR), we were happy to share our anesthetic management routine for EBD patients with them several years ago.

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Lidocaine and Cerebral Metabolism

To the Editor.—Allow me to comment on the article entitled “The Detrimental Effect of Lidocaine on Cerebral Metabolism Measured in Dogs Anesthetized with Isoflurane.” 1 It seems to me that the term “detrimental effect” may be somewhat unfair to the good drug lidocaine, and the results may well be questioned. First, the absolute values for CBF and CMRO 2 in the EEG burst-sup-
pression of isoelectric brain seem high (as do previous
data from that laboratory), raising the possibility of extracerebral contamination of blood to the venous out-
flow 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, per-
haps (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 im-
licit difficulties in technique become important when looking for, perhaps, only minor or no differences be-
tween 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

1. Mils LN, Milde JH: The detrimental effect of lidocaine on cere-
bral metabolism measured in dogs anesthetized with isoflu-
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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 communici-
ating with the sinus, and determination of the brain regions drained by the sinus 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 contamina-
tion by post-mortem injection of the posterior sagittal sinus with blue vinyl acetate.3,4 No contamination by extracerebral structures was found.

The apparent pressure-dependent changes in cere-
bral blood flow reported in the article3 were, indeed, due to the loss of autoregulation during deep isoflurane anesthesia. This loss of autoregulation was readily ap-
parent at concentrations greater than 2% end-expired isoflurane,4,4 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 extra-
cranial vessels.

Normal values for cerebral metabolites for our lab-
oratory were obtained from normal awake dogs in whom the stress response to immobilization, as manifested by increased catecholamines, cerebral blood flow, and cere-
bral metabolism, was blocked by sympathetic block-
ade produced by total spinal anesthesia.8–10 The cere-
bral cortical biopsies were taken by a technique that deposits a sample of brain (200–400 mg) into liquid nitrogen within 1.11 Samples were taken for each hemi-
sphere simultaneously in both anterior and posterior regions, so that a minimum of four samples were taken from each animal. These were then immediately pre-
ared for analysis in a refrigerated chamber at −25 deg C.12 Thereafter, the tissue extracts were analyzed by enzymatic fluorometric techniques.13 The concentra-
tion of each metabolite for each animal was the resul-
tant mean of the four samples taken from that animal. The precision of the technique is manifested by the very small standard error (normal ATP = 2.01 ± 0.01 
μmol·g⁻¹). Using the technique exactly as described, normal concentrations of cerebral ATP have been re-
ported from dogs anesthetized with isoflurane,1,5,6 eto-
midate,14 sufentanil,15 and triazolam.9 Abnormal con-
centrations of ATP have been reported only for dogs exposed to incomplete and complete cerebral ischemia,16,18–20 situations in which a lower concentration of ATP would be expected. If there were some error in our technique which allows ATP hydrolysis and, therefore, results in falsely low ATP concentrations, it is not supported by any of our results.

Therefore, we feel that our finding of abnormal con-
centrations of cerebral ATP (1.77 ± 0.05 μmol·g⁻¹) following the administration of lidocaine is real, not the result of systematic error in our methodology. Our findings are further supported by in vitro studies.21 We