In reply—The objective of our study was to examine the effect of PEEP on cerebral blood flow (CBF) and cerebrospinal fluid pressure (CSFP) with and without the maintenance of mean arterial pressure (BP) at pre-PEEP levels. With the application of PEEP, decreases in BP were prevented by gradual infusion of intravenous fluid titrated against BP changes at each of the three levels of PEEP applied. This required approximate infusion volumes of 11 per cent of the estimated blood volume in the saline group and 4 to 7 per cent in the mannitol group.² We did not, as stated in the letter above, "return cardiac output (CO) to pre-PEEP levels" with volume infusion: BP, and not CO, was the controlled variable and only small decreases in BP were permitted before volume infusion was increased. Concerning the mechanism of the increase in CSFP in the saline group, we agree that the most likely explanation is an onotic pressure gradient given the available data, but we cannot exclude shifts in intracranial contents resulting in regional differences in perfusion pressure which are known to occur.² We regret that onotic pressure data are not available and we also agree that additional studies should be done using colloid for BP control as it may offer advantages over hyperosmotic and crystalloid solutions.

With reference to the mannitol data, the paper by Burke et al. (reference 2 of the above letter), which was not available to us as it was published a month after ours, examines several mechanisms for blood flow increases with mannitol but does not permit any conclusions as to which of the effects of mannitol (decreased viscosity and red cell volume, increased red cell deformability, direct vasodilating effects, and brain dehydration) predominates in the CBF response to mannitol.

In the early period of the infusion of mannitol we did not observe significant increases in CSFP probably because of the slow rate of injection and the low total volume infused. We did not cite the paper by Cottrell et al. (reference 3 of the above letter) because their use of a bolus injection of mannitol and the lack of supporting hemodynamic data did not permit comparison of our results. It should not be surprising that the difference in technique of administration of mannitol might result in a difference in the CSFP responses as similar results have been reported for nitroprusside by one of the authors of the above letter.³

We take issue with the "apparent inconsistencies" in the central venous pressure (CVP), pulmonary arterial pressure (PAP), and cardiac output data suggested above. It appears that the authors above, without supporting data, expected to see additive effects of PEEP and volume infusion on central venous pressure. We did not observe this, rather we found that proportional increases in CVP and PAP with PEEP occurred in all three groups. Our data are consistent with those of Sykes et al.,⁴ who demonstrated that the small increases in CVP with the addition of PEEP (10 cm H₂O) in euclidean dogs contrasted with decreases in CVP in hypervolemic dogs placed on PEEP. The literature is replete with conflicting reports of the effects of PEEP on hemodynamics and ventricular function.⁵⁻⁷ Both increases⁵ and decreases⁶ in transmural right ventricular pressure have been documented with PEEP therapy suggesting that the prevailing conditions of the study exert significant influence on the resulting data. In this regard, we are not aware of data from a study which closely matched our protocol which would allow comparison of the data.

We see no reason to reconcile the small or absent gradients between CSFP and CVP reported for the control group (Group 1) and the mannitol group (Group 2) animals as they pertain to the existence of steady state conditions during our data collection. As stated in our manuscript, the animals were studied in a position where the head was at the same level as the heart (horizontal). In this position we found that the progressive application of PEEP did reduce the gradient between CSFP and CVP from 4.1 to 0 cm H₂O in Group 1 and from 5.6 to −0.7 cm H₂O in Group 3. As we stated in the text, during the 30-min equilibration period no significant changes in the measured variables were observed. It is likely that small changes in CSFP were occurring due...
to CSF formation but with time constants long enough to minimize the effects on our data. Perhaps the authors above are referring to a study by Aidinis et al.9 which was co-authored by one of them, where PEEP application resulted in CSFP changes with a shorter time course than we report. In that study9 where BP was not controlled, they did speculate that “the absolute increases in ICP (intracranial pressure) might have been higher and longer lasting if BP had not simultaneously decreased.” In our study where BP changes were prevented, we found longer time constants as Aidinis et al.9 predicted.

The last issue to be addressed is the question of the influence of the level of stress on the resulting data. Our protocol, which was approved by the Animal Studies Committee, was designed to closely mimic the intensive care setting where head-injured patients are ventilated without sedation or anesthesia in order to evaluate mental status and neurological function. Our animals were given general anesthesia at least three days prior to the study for the implantation of the flow probes and catheters. On the day of the study the introduction of percutaneous catheters using local anesthetics was performed. The use of general anesthesia during the studies would not mimic the clinical setting and would further complicate the interpretation of the data because of the effects on CBF autoregulation. We have no evidence of stress based on heart rate, CO1 CO2 production (116.2 to 142.5 ml/min), O2 consumption (123.4 to 151.5 ml/min), or CBF (71 ml/min this study as compared to 75 ml/min10 and 47-68 ml/min in another study11). Thus, we do feel that our data are of value in understanding another aspect of the potential adverse effects of ventilation with PEEP.

We thank Drs. Drummond, Todd, and Shapiro for their interest and comments.

An Unusual Cause for Increased Resistance to Injection during Administration of Spinal Anesthesia

To the Editor—An 83-year-old man with hypertension and an ischemic ulcer on his ankle was scheduled for split thickness skin graft under spinal anesthesia. He was hospitalized 18 years previously following a neck injury. At that time a myelogram using Pantopaque* and an anterior cervical fusion were performed.

The patient was placed in the lateral position and the subarachnoid space entered without difficulty using a 22-gauge, 8.9-cm Travenol† spinal needle. Cerebral spinal fluid flow was slow but continuous and contained an oily substance. One-tenth of a milliliter of CSF was withdrawn into a 6-ml Travenol disposable plastic syringe

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