Osmotic Agents and Intracranial Pressure

To The Editor: In the paper by Muizelaar, et al. (Muizelaar JP, Wei EP, Kontos HA, et al: Mannitol causes compensatory cerebral vasconstriction and vasodilation in response to blood viscosity changes. J Neurosurg 59:822-828, November, 1983), the statement is made that there is no proof that osmotic agents such as mannitol lower intracranial pressure (ICP) by decreasing brain water content. The mechanism of action of osmotic agents by dehydration of the brain is one of the most firmly established facts in experimental neuropathology. The authors cite a paper by Reed and Woodbury indicating that the maximum decrease in brain water occurred in rat cortex 60 minutes after the intraperitoneal administration of urea, while the maximum decrease in ICP occurred at 15 minutes. These studies were conducted in nephrectomized animals. In a later study using intact rats, the maximum decrease in brain water was found in the 15-minute sample. In the dog, maximum dehydration of the brain occurs at the same time as the maximum decrease in ICP. Hypertonic urea also dehydrates edematous brain in both ischemic injury and triethyl tin intoxication.

Our own contribution to this problem utilized the model of cryogenic cerebral injury in the rhesus monkey. We showed that the decrease in ICP occurring with the intravenous administration of hypertonic urea was associated with dehydration of the undamaged hemisphere as a whole. A subsequent study in cats, utilizing tissue samples, showed that the dehydration involved normal but not edematous brain. This has also been shown in the dog with intravenous glycerol. It may be noted that, in this study, there was a rise in ICP in 6 hours, at which time the brain water was still decreased, but one cannot reasonably conclude from this that the initial drop in ICP was not due to dehydration of the normal tissue.

Since the edema associated with brain tumors is of the vasogenic type and the cryogenic injury model is the experimental prototype of this form of edema, we inferred that this also occurs in human brain tumors. This has been shown by Takagi, et al., not to be the case. In their study they measured the water content of white matter adjacent to tumors in human biopsy specimens and compared it to the computerized tomography (CT) attenuation number (EMI units) in the preoperative scan. They found a correlation between the decreased CT attenuation coefficient of the edematous tissue and its increased water content. Two patients who received mannitol 1 to 2 hours before sampling showed a decrease in the water content of tissue samples from areas that were shown to be edematous in the preoperative CT scan. This, it seems to me, is unequivocal evidence that intravenous mannitol acts in humans by decreasing the water content of edematous brain.

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RESPONSE: Dr. Clasen cites a number of references showing that osmotic agents can decrease brain water content. Our article, however, did not imply that these agents do not have an effect upon brain water content, but we do deny that there is a close relationship between brain water content and ICP. In the article by Reed and Woodbury cited in our report, such a relationship was not found, and the fact that those authors used nephrectomized rats cannot explain this lack of correlation. In the later paper by Reed and Woodbury, the decrease in water content indeed occurred much earlier, but ICP was not measured, so no conclusion can be reached about the relationship between brain water and ICP. In one of their own papers, Clasen, et al., remarked that intravenous hypertonic glucose promptly reduced intracranial hypertension without demonstrable effect on the increase in water brought about by a cold lesion. The fact that Guisado, et al., noted a rise in ICP at the time that brain water was still decreased may not indicate that the initial drop in ICP was not due to dehydration of the brain, as stated by Dr. Clasen. It is, however, certainly not supportive evidence for the "dehydration explanation."

Other supportive evidence for the autoregulatory nature of the ICP response after mannitol administration comes from our as yet unpublished data. In 42 head-injured patients, we have measured cerebral blood flow (CBF) and ICP and tested autoregulation by increasing blood pressure. In the 28 patients with intact autoreg-

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ulation, a bolus of mannitol resulted in a decrease in ICP in 86% and an increase in CBF in only 18%; when autoregulation was not intact, mannitol caused a decrease in ICP in only 36%, while in 79% an increase in CBF was noted.

Thus, although osmotic agents can reduce brain water content and this must have some influence upon ICP, we do think that the autoregulatory vasoconstriction is quantitatively more important. We agree, however, that many questions still remain unanswered and therefore we plan, in cooperation with Dr. Anthony Marmarou, who developed the very sensitive specific gravity method for brain water content measurements and who now works in our department in Richmond, to quantitate the contribution of each of the three brain compartments to the decrease in ICP seen after administration of osmotic agents.

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References


Anterior Inferior Cerebellar Artery Aneurysms

To the Editor: The article by Nishimoto, et al., concerning anterior inferior cerebellar artery aneurysms surveys the literature concerning 14 previously reported lesions and adds three cases of their own (Nishimoto A, Fujimoto S, Tsuchimoto S, et al: Anterior inferior cerebellar artery aneurysms. Report of three cases. J Neurosurg 59:697–702, October, 1983). Their concluding statement is misleading, for they say that decompression of the seventh and eighth nerves by aneurysmal excision or aspiration of the contents gave no improvement in neurological findings, and in support of this they cited our case.1 This certainly was not the outcome in our patient, who preoperatively had a complete seventh nerve palsy and a partial eighth nerve palsy. He had experienced a complete return of seventh nerve function and partial return of hearing at the time we reported this case, 8 months after aneurysm excision. It is now 6 years since the operation and the patient retains full facial function with only a very mild high-frequency hearing loss and a discrimination score of 95%.

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References


RESPONSE: We apologize to Drs. Johnson and Kline for misrepresenting their paper.4 Their patient recovered fully postoperatively from both seventh and eighth nerve palsies.

The patient in our Case 2, who underwent aspiration of the aneurysm contents, and whose symptomatology remained unchanged during the early postoperative period, has very recently presented with partial regression of facial paresis without any improvement of hearing disturbance.

All six patients who underwent excision or aspiration of the aneurysm (our Case 2 and five others1–5) had both seventh and eighth nerve palsies postoperatively. After surgery, full recovery of both seventh and eighth nerve palsies,4 full recovery of seventh nerve palsy,2 and partial recovery of seventh nerve palsy (our Case 2) were seen in one case each. No improvement was noted in two cases,1,2 and information is lacking in the last case.5 Thus, seventh nerve palsies seem to recover more readily than eighth nerve deficits.

In the patients with carotid artery aneurysms, such as carotid-cavernous, carotid-ophthalmic, and carotid-posterior communicating artery aneurysms, associated with second, third, fourth, fifth, or sixth cranial nerve palsies, recovery of function is frequently observed following volume reduction of the offending aneurysm by neck clipping, trapping, or carotid ligation. Therefore, decompression of the seventh and eighth nerves is reasonable in cases of anterior inferior cerebellar artery aneurysms accompanied by nerve palsies, by methods such as neck clipping, neck ligation, proximal arterial ligation, and trapping with or without aneurysm excision.

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