
Hydrocephalus is one of the most frequently encountered neurosurgical problems necessitating surgical intervention. To date, shunting the cerebrospinal fluid to the extracranial (peritoneal, pleural, ureter, thoracic duct, gall bladder, transverse sinus, and ayzygous vein) cavities are the most readily used and accepted therapeutic approaches in the treatment of hydrocephalus. Even though hydrocephalus is an old neurosurgical problem, controversies about the etiology and therapeutic approaches still remain.1 Shunting the cerebrospinal fluid from the ventricular cavity to one of the cavities mentioned earlier is considered to be a simple procedure, although ventriculoperitoneal shunt revision interventions constitute a huge part of the neurosurgical practice.

The main problems that occur in ventriculoperitoneal shunting are divided in three groups: 1) proximal end; 2) distal end; and 3) valve problems. Early pre- or postoperative proximal end problems include the improper placement of the ventricular end of the shunt, intracerebral hemorrhage, and obstruction of the catheter.1–3 Savitz, et al., dealt with the intracerebral hemorrhage caused by ventricular catheter placement.

We have some questions for the authors.

We know that all of the cases are cannulated through a posterior parietal hole, without frontal shunting. Do the authors have any experience with the differences of hemorrhage rate between frontal shunting and occipital shunting? Some authors reported differences between frontal and parietal shunt patency. There also are some reports discussing the difference of frontal or parietal shunting in epilepsy risks.1

Do the authors have any experience with the rate of hemorrhage when the catheter has been inserted with the help of ultrasonography or navigation? The improper placement of a ventricular catheter and multiple attempts to have a proper placement causes an increased rate of hemorrhage, and using ultrasonography or navigation during insertion of the catheter assists with proper placement.

Does the age of patient or opening pressure of cerebrospinal fluid have any affect on the hemorrhage rate? The authors reported that their patients were adult, but they did not mention the opening pressure of the ventricles.

Finally, I wonder about the causes of hydrocephalus in those patients. Were they congenital or secondary to central nervous system pathology? This is important, because with vascular pathology or tumor, bleeding may occur while the catheter passes through.

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References

J. Neurosurg. / Volume 92 / June, 2000 Neurosurgical forum

RESPONSE: I appreciate Dr. Cirak taking the time to review my article. Wound infection is a major sequela of ventriculoperitoneal shunting. In addition to the three groups of proximal end, distal end, and valve problems.2 Disposable instrumentation, simplicity of insertion, and adult patients contributed to the overall low incidence of postoperative complications in my own series of 125 one-piece catheters without reservoirs.3–6

My preference for parietal insertion of the proximal end was primarily due to training as a resident at the Mount Sinai Medical School of Medicine. My experience with right frontal cannulation has been reported with the use of ventriculoperitoneal shunting in 22 cases of acute head trauma manifesting increased intracranial pressure.3 Even with collapsed ventricles and the presence of cerebral concussion, no instance of intraparenchymal hematoma around the shunt tubing was documented on follow-up computerized tomography scans. Since all of these patients had grossly hemorrhagic cerebrospinal fluid, no conclusion can be drawn about possible intraventricular bleeding caused by catheter insertion.

I have no experience with ultrasonographic guidance as reported by Mahoney, et al., but the availability of real-time demonstration should be a significant improvement in intraoperative monitoring of shunt placement and any resultant bleeding. Opening pressures of the ventricles were recorded only in the 22 cases of right ventriculoperitoneal shunting performed by a subarachnoid intracranial pressure monitor bolted into the left frontal skull.3 The Discussion section of my article compares the incidence of delayed intracerebral hemorrhage caused by ventriculoperitoneal shunt insertion in the pediatric population with the few previously reported cases. Table 1 lists 58 instances of idiopathic normal-pressure hydrocephalus and 67 patients who developed hydrocephalus secondary to known central nervous system pathology.

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Hypothermia and Head Injury

To THE EDITOR: I read with interest the paper by Dr. Shiozaki and associates (Shiozaki T, Kato A, Taneda M, et al: Little benefit from mild hypothermia therapy for severely head injured patients with low intracranial pres-
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sure. J Neurosurg 91:185–191, August, 1999), in which the authors reported that they failed to find improvements in the neurological outcome of a limited number of brain-injured patients with low intracranial pressure.

I found several interesting points in the paper that I believe the authors should address.

1) They did not discuss any differentiation of brain temperature between 34˚C and 32 to 33˚C. We have recently reported the depression of the inflammatory cytokine, IL-6, in arterial and internal jugular bulb plasma in traumatically brain-injured patients who received moderate hypothermia of 32 to 33˚C as measured at the internal jugular bulb. Furthermore, we have reported low C-reactive protein (CRP) production, an indicator of inflammatory response induced by IL-6 elevation, in brain-injured patients during hypothermia. In Dr. Shiozaki’s reported protocol of mild hypothermia (34˚C), CRP did increase even during the hypothermia phase, suggesting that hypothermia of 34˚C could not attenuate the systemic inflammatory responses including those of the brain. Therefore, it seems inappropriate to favorably compare their data with the results of another study, in which cytokine reduction in cerebrospinal fluid was demonstrated in brain-injured patients undergoing hypothermia of 32 to 33˚C.

2) The authors did not clearly show the time elapsed from the injury to the initiation of mild hypothermia of 34˚C. Many papers indicate the importance of this window period (within 30 minutes after the injury) for moderate hypothermia to obtain the neuroprotective effects of the therapy in brain injury. Dr. Shiozaki and associates inserted a ventricular catheter to measure intracranial pressure (ICP) and then performed “conventional ICP reduction therapy” (hyperventilation of 25–30 mm Hg, fluid restriction and high dose barbiturate therapy). Unfortunately, I could not find in the paper any mention of how long such a procedure took. After the conventional ICP reduction therapy, the authors randomized the patients whose ICPs were kept to be low, into hypothermia and normothermia groups. I could not understand how this randomization was meaningful.

3) The authors did not report the levels of PaCO2 adjustment during hypothermia or whether or not PaCO2 data were corrected by each body temperature. This is quite an important issue during hypothermia therapy. In our protocol, during the therapy, PaCO2 levels are controlled to 30 to 33 mm Hg; these are not levels that are not corrected by body temperature. This protocol is supported by Dr. Metz’s paper.

In Dr. Shiozaki’s paper, the authors did not report jugular venous O2 saturation. Profound hyperventilation of 25 to 30 mm Hg, as presented in the paper, may cause a decrease of blood flow in the penumbra area, or even brain ischemia. Therefore, profound hyperventilation requires adequate monitoring such as SjvO2 measurement, to prevent potential brain ischemia.

References


RESPONSE: We appreciate the interest of Dr. Aibiki in our recent article. Before going into detail, I would like to reemphasize the bottom line of this study. The aim of our study was to determine whether mild hypothermia is essential for the treatment of severely head-injured patients with low ICP. Many institutes have used mild hypothermia to treat severely head-injured patients regardless of their ICP. It is, therefore, far and away more important to clarify the indication for the use of mild hypothermia than to discuss the trifling differences that might be derived from the small temperature difference between 31˚C and 32 to 33˚C. I understand that Dr. Aibiki made comments based on his experience from a limited number of brain injured patients without ICP monitoring. I’m afraid that Dr. Aibiki has two basic misunderstandings.

First, Dr. Aibiki claimed in his letter that 32 to 33˚C mild hypothermia suppressed plasma interleukin (IL-6) elevation in patients with traumatic brain injury and that the production of CRP was low in brain-injured patients. However, these results were not surprising because they used dexamethasone for the first 4 days in their study and they excluded those patients with complicated conditions such as severe pulmonary infection and associated trauma of the chest or abdomen. As described in our article, we included those patients with additional injuries of Abbreviated Injury Scale scores less than four. Under ordinary circumstances, plasma IL-6 and CRP concentrations are high within 1 week after injury in patients with multiple trauma. The production of CRP in the liver would not be suppressed at 32 to 33˚C. Thus I would like to know why he could conclude that low CRP levels are the evidence of the beneficial effect of hypothermia when steroids are administered. Does low CRP value in serum mean beneficial or favorable response in uncomplicated and isolated head trauma patients to begin with? The most crucial misunderstanding in Dr. Aibiki’s letter is that he simply compared our data with their data without considering the difference between the inflammatory responses in the brain and the systemic inflammatory responses. The