Neurosurgical forum

TO THE EDITOR: I read with interest the recent article of Nakaguchi and colleagues, which discusses the relationship between drainage catheter location and postoperative recurrence of CSDH (Nakaguchi H, Tanishima T, Yoshimatsu N. Relationship between drainage catheter location and postoperative recurrence of chronic subdural hematoma after burr-hole irrigation and closed-system drainage. J Neurosurg 93:791–795, November, 2000; see Abstract, pp 1023–1024 of this issue).

When the authors describe the random positioning of the drainage catheter tip, it seems to me that they neglect to mention the criteria of burr-hole placement (for example, volume of the hematoma, its radiological features, the presence of multiloculation or clots).1,2 Moreover, information is lacking regarding the method adopted for brain volume reexpansion.

In my opinion, the age range of the presented series of patients may be too great (25–92 years). In fact, if drainage is greatly advisable in the elderly patient in whom immediate brain reexpansion is often absent, it seems questionable to use a draining catheter in younger patients in whom good restoration of brain volume is often achieved at surgery.

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RESPONSE: We performed surgical intervention with burr-hole placement and evacuation of CSDH in the following circumstances: 1) when the thickness of the hematoma was 1.5 cm or more; 2) when brain sulci were effaced by the subdural collection on CT scans; or 3) when there were neurological symptoms (such as headache, disorientation, gait disturbance, incontinence, nausea, and so forth) that were considered to be caused by increased intracranial pressure due to the subdural fluid collection.

We instructed our patients to keep their head at the same level as the heart all day long, from immediately after surgery until the drainage catheter was removed to promote reexpansion of the brain. We also performed moderate hypervolemic therapy in all our patients with CSDH from admission until the day after surgery (intravenous saline injection at the rate of 60 ml/hr).1

Because CSDH rarely occurs in younger patients, we do not know whether younger patients have a lower recurrence rate than older patients. Although we had the impression that younger patients tend not to suffer from recurrence of the hematoma, we compared the postoperative recurrence rate in patients younger and older than 60 years of age and found no statistically significant difference between them. Nonetheless, we do not have any data about the postoperative recurrence of CSDH in much younger patients (for example, < 40 years old), so further investigation is necessary.

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References

Potassium and Intracranial Pressure


Abstract
Object. Disturbed ionic and neurotransmitter homeostasis are now recognized as probably the most important mechanisms contributing to the development of secondary brain swelling after traumatic brain injury (TBI). Evidence obtained in animal models indicates that posttraumatic neuronal excitation by excitatory amino acids leads to an increase in extracellular potassium, probably due to ion channel activation. The purpose of this study was therefore to measure dialysate potassium in severely head injured patients and to correlate these results with measurements of intracranial pressure (ICP), patient outcome, and levels of dialysate glutamate and lactate, and cerebral blood flow (CBF) to determine the role of ischemia in this posttraumatic ion dysfunction.

Methods. Eighty-five patients with severe TBI (Glasgow Coma Scale Score < 8) were treated according to an intensive ICP management-focused protocol. All patients underwent intracerebral microdialysis. Dialysate potassium levels were analyzed using flame photometry, and dialysate glutamate and dialysate lactate levels were measured using high-performance liquid chromatography and an enzyme-linked amperometric method in 72 and 84 patients, respectively. Cerebral blood flow studies (stable xenon computerized tomography scanning) were performed in 59 patients.

In approximately 20% of the patients, dialysate potassium values were increased (dialysate potassium > 1.8 mM) for 3 hours or more. A mean amount of dialysate potassium greater than 2 mM throughout the entire monitoring period was associated with ICP above 30 mm Hg and fatal outcome, as were progressively rising levels of dialysate potassium. The presence of dialysate potassium correlated positively with dialysate glutamate (p < 0.0001) and lactate (p < 0.0001) levels. Dialysate potassium was significantly inversely correlated with reduced CBF (p = 0.019).

Conclusions. Dialysate potassium was increased after TBI in 20% of measurements. High levels of dialysate potassium were associated with increased ICP and poor outcome. The simultaneous increase in dialysate potassium, together with dialysate glutamate and lactate, supports the concept that glutamate induces ionic flux and consequently increases ICP, which the authors speculate may be due to astrocytic swelling. Reduced CBF was also significantly correlated with increased levels of dialysate potassium. This may be due to either cell swelling or altered vasoreactivity in cerebral blood vessels caused by higher levels of potassium after trauma. Additional studies in which potassium-sensitive microelectrodes are used are needed to validate these ionic events more clearly.
This is clearly a major contribution to our understanding of the pathophysiology of head trauma and provides clinical evidence that strongly supports the results of previous laboratory work in animals. Based on their results, the authors postulate that it is the glutamate/K+ ion-induced cytotoxic cellular swelling that is responsible for the uncontrollable rise of intracranial pressure (ICP), which in turn is responsible for the poor outcome in patients with severe traumatic brain injury (TBI). The alternative interpretation of the presented data, however, is that the uncontrollable rise in ICP is the final stage of irreversible brain trauma and is by itself only a reflection of its irreversibility rather than its cause. Our previously published work supports this interpretation.

Extensive laboratory work measuring the K+ and Ca++ ionic fluxes following experimental traumatic subarachnoid hemorrhage and intracerebral hematoma in cats by using ion-sensitive microelectrodes previously indicated the important role K+ and Ca++ plays in the dysfunction of the brain cellular elements. Similar to the authors’ observation, we have reported different patterns of K+ elevation coupled with Ca++ reduction in extracellular space following onset of experimental trauma. The observation of patterns that ranged from rapid K+ clearance to persistent K+ elevations led us to suggest the existence of traumatic penumbra. According to this concept, which is similar to the concept of ischemic penumbra proposed earlier, different cell populations sustain different degrees of injury at the time of initial impact. Whereas the most severely injured cells are damaged irreversibly due to their membrane rupture, others referred to as the cells of the marginal zone sustain only a potentially reversible injury. Their loss of function is due to the failure of the electrochemical pumps located at the membranes, but their membranes remain physically intact and they have the potential to fully recover. It is in fact the extent of the marginal zone injury and the degree of its recovery that determines the outcome in brain injuries. Thus, the persistent K+ elevations seen in the most severely injured patients with the highest levels of ICP most likely represented large components of cells with membrane destruction and signified the irreversibility of the injury. This type of injury was represented in our model by a cortical rupture, in which the elevated K+ level never returned to normal and remained substantially elevated for the duration of the experiment. The design of our experiments, which were performed with the skull removed, eliminated the increased ICP as the potential cause of the persistent K+ elevations. Furthermore, in other experiments performed with the skull closed (unpublished data), elevations of ICP up to 40 mm Hg did not cause K+ elevation or Ca++ reduction.

The article by Reinert, et al., provides strong evidence that the processes we have observed in cats take place in humans and that several of the concepts we have proposed may in fact be applicable to humans. It appears that brain injury induces a complex process in which K+ release into the extracellular space and Ca++ influx into the cells leads to a cascade of multiple processes that, in the end, result in cellular death and compromise local microcirculation, thus perpetuating the cycle of cellular injury. The K+ and Ca++ movement is inextricably linked with the release of neurotransmitters, and the Ca++ influx into the cells specifically may be the most critical process in trauma as it is in ischemia and other injury events as proposed by Schanne, et al. Thus, it appears that severe trauma is on a cellular level no different than ischemia or toxic cell damage. Future treatment, therefore, must target the marginal zone injury of traumatic penumbra in ways conceptually similar to those currently emerging in treatments of ischemia. This would mean that in the most severe cases of trauma our attempts to lower the ICP by using our current agents will remain unsuccessful and that a different pharmacological approach is necessary. The search for new drugs should target the electrochemical pumps at the cell membranes that are responsible for maintaining osmotic equilibrium rather than acting only to remove the excess water from the edematous cells by using osmotic agents. Unfortunately, this also means that the concept of uncontrollable ICP elevation is synonymous with irreversible brain injury.

Clarification of these issues requires further extensive work but, in the meantime, the authors are to be congratulated for a major contribution to our understanding of head trauma, and we hope that they continue their exceedingly demanding investigations.

**References**


**RESPONSE:** We thank Dr. Hubschmann for his laudatory comments regarding our manuscript. We are, of course, well aware of the seminal work from Dr. Hubschmann’s group, which together with that of Dr. Kimmelberg and others provided the theoretical foundation for our understanding of ionic homeostasis in ischemia and intracerebral hematomas. We agree entirely that our findings reveal strikingly similar changes in TBI in humans compared with those seen in animal models. The central issue is that persistent elevations of K+ in the extracellular space can occur only in the absence of homeostatic mechanisms, such as the Na+-K+ pump, and astrocytic buffering, either by the glutamate-linked transporter or the voltage-dependent K+ IR channel. Our neuroimaging data in over 200 severely head injured humans strongly supports the concept that persistent ionic leakage of this type can occur only in the presence of...