Focus Forum

Letters to the editor

Skull Base Tumors

To The Editor: I would like to express my deep gratitude to all involved for their contributions to the March 2001 issue of Neurosurgical Focus on "Skull Base Chordomas", and to the publishers as well. I am a chordoma patient who 8 years ago was unable to find chordoma references at several large medical teaching facility libraries, except for two articles that presented a dire and morose outlook at best. I eventually concluded that the lack of histological and series data was largely due to the rarity of chordomas, wherein money and research were better spent on those illnesses that beset many more people. In the past 8 years I have undergone three resections and two proton radiation treatments for chordoma, including radiotherapy for an inoperable (4-cm) chordoma intruding upon and encasing an internal carotid artery. There is no question that without the technology, diligence, and tremendous care I received from my doctor(s), I would not be on this planet to share the love, fulfillment, and joys of this life. I often think about how fortunate I am to have been born at a time when soft-tissue imaging, surgical access, and fractionated radiotherapy are available, due largely to the efforts of yourselves and your predecessors. I now see that presently, through your efforts, the bar is being raised and new research is being undertaken in chordoma treatment approaches, which provides new hope for those of us living with chordoma. At first onset in 1993 I was told that the intention was to control the tumor growth; the hope was that someday new treatments would be available. That is exactly what your work has generated—hope! On behalf of all of us, and our families, who will continue to benefit from your research into chordoma control and abatement; Vielen Dank, shokran Gazillan, Mo’tech’shaker’am, tack sa mycket, Bolshoe spasibo, Arikato gozaimasu, Muchas gracias, aapakaa bahut, and Thank you! I would also like to extend my utmost personal gratitude to contributor Dr. Eugen Hug!

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Pulsatility Index

To The Editor: I have read the paper by Cosan and colleagues (Cosan TE, Gucuyener D, Dundar E, et al: Cerebral blood flow alterations in progressive communicating hydrocephalus: transcranial Doppler ultrasonography assessment in an experimental model. Neurosurg Focus 9 (5): Clinical Pearl, November, 2000) with great interest.

Abstract

Object. In many cases communicating hydrocephalus is the result of impairments in cerebrospinal fluid absorption in the arachnoid villi at the cranial convexity. Reported methods of creating experimental hydrocephalus have not sought to produce an arachnoidal adhesion in the cranial convexity. In this study the authors investigate alterations in cerebral blood flow (CBF) in experimental communicating hydrocephalus induced by the injection of kaolin into the subarachnoid space at the convexity in neonatal rats.

Methods. In neonatal rats, kaolin was injected into the subarachnoid space at the cranial convexity. Assessment of CBF alterations was performed using transcranial Doppler ultrasonography pre- and postinjection and at 10 days, 4 weeks, and 8 weeks postinjection. Light microscopy examination was also performed at 4 weeks and 8 weeks postinjection.

Conspicuous lateral ventricle enlargements of different dimensions were observed in kaolin-injected rats at 4 to 8 weeks postinjection. The third and fourth ventricles were dilated to a lesser extent. Resistance to CBF and increased mean CBF velocity were apparent 8 weeks after kaolin injection. Further, destruction and even loss of ependymal layers were more prominent at the chronic stage.

Conclusions. The present model may be considered a progressive communicating hydrocephalus because of marked changes in blood flow dynamics and destruction of the ependymal layer at the chronic stage.

I congratulate the authors on their model, although I think that it has been underinvestigated. First of all, it is very risky to study hydrocephalus without a measurement of intracranial pressure. All points included in the Discussion of the article, which relate to intracranial (or rather intraventricular) pressure, remain highly speculative. I strongly resist an interpretation of pulsatility index (PI) as a measure of cerebrovascular resistance. If anyone still has doubts, consider two examples, which can be found in individuals who have preserved vascular reactivity: 1) hypercapnia, when cerebrovascular resistance falls and PI also falls; and 2) decrease in cerebral perfusion pressure, when cerebrovascular resistance falls but PI increases. Actually, the authors’ finding that blood flow velocity increases in progressive hydrocephalus blood flow (it is not the same as CBF; with due respect, I think there is an error in the title of the paper) contrasts with findings in other studies reporting blood flow volume in hydrocephalus. Almost all indicate a decrease in CBF, particularly in periventricular and frontal cerebral areas. Pulsatility index is one of the most misleading variables ever used in cerebral hemodynamic studies. First, it is dependent on arterial pressure pulsatility. A high systolic–diastolic difference of arterial pulse pressure increases PI directly. Second, PI depends on heart rate, cerebral perfusion pressure, arteriovascular carbon dioxide concentration, cerebrovascular resistance, and compliance of the big vessels. To conclude that high cerebrovascular resistance is a reason for PI to rise, one must prove that all other factors were unlikely to contribute. In clinical (and experimental) practice, situations in which both PI and mean blood flow velocity rise together are extremely rare. (I have seen it only a few times, in patients with vasospasm following poor-grade subarachnoid hemorrhage and with intracranial hypertension.) The PI is a result of dividing peak-to-peak pulse amplitude by a mean flow velocity. For this reason we usually see an increase in flow velocity correlated with a decrease in PI (hypercapnia and hyperemia). Increases in mean flow velocity by 100% and in PI by approximately 50% would create a 300% increase in flow velocity pulse amplitude. Perhaps it caused an increase in arterial pulse pressure (data are not provided, although the authors mention that arterial blood pressure had been measured), arterial hypotension, or deep intracranial hypertension (these latter two factors are unlikely because mean flow velocity increased). This leaves the reader in a shadow of uncertainty. The authors’
assertion that high PI was due to increased cerebrovascular resistance (caused by reduction of the capillary network) and that cerebral autoregulation caused an increase in mean flow velocity is difficult to understand and is not based on the results presented. Cerebral autoregulation is meant to stabilize blood flow, not to increase it. Certainly these data cannot be interpreted in a convincing manner without parallel monitoring of arterial and intracranial pressure dynamics.

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References


RESPONSE: We appreciate the enlightened comments of Dr. Czosnyka on our report. Our primary purpose in the article was to describe a new experimental communicating hydrocephalus model. Transcranial Doppler (TCD) ultrasonography measurements were obtained to determine the clinical applicability of this model. The results of the parameters investigated in our model might be considered inconsistent with other models. Because we created adhesions in the subarachnoid space at the cranial convexity in neonatal rats, cerebral cortical vessel blockage may have occurred as a result of these adhesions. The new complexity arising from this possibility, together with the fact of a progressive ventricular enlargement, would have to be taken into consideration in any evaluation of the data. Not to be overlooked, also, is the remodeling of the leptomeningeal vascular network in growing brains.1 Vascular changes in the variable studies on sagittal sinus and cortical vein occlusion may be reconsidered in this neonatal hydrocephalus model.2,4 On the other hand, the PI is accepted as a general measurement of the resistance to flow encountered by moving blood in the vessel. In higher PI there is a resistance to flow in the vascular bed, and in lower PI there is a reduced resistance.5 Dynamic changes in the PI, as calculated from TCD data, allow us to assess the forces acting on the terminal vasculature of the brain. In studies3,7,8 on CBF volume during which PI and resistance index (RI) in healthy persons were compared with those in patients with hydrocephalus before and after shunt placement, it has been proven that whereas CBF volume values were significantly decreased, PI and RI values were significantly increased, thus indicating an increased distal cerebrovascular resistance. Immediately after shunt insertion, intracranial pressure (ICP) returned to normal, and PI and RI values decreased significantly, whereas the mean CBF volume increased. The authors conclude that TCD ultrasonography may provide a tool for assessment of ICP with hydrocephalus. It has also been reported that changes in the RI and PI appear to be useful indicators of elevated ICP.7 A theoretical relationship has been demonstrated between absolute cerebral artery blood velocity and absolute CBF.6 Many factors, such as arterial pressure pulsatility, heart rate, arterial blood gas levels, and so on are known to affect PI. As mentioned in our manuscript, rats that could not be monitored in physiological ranges were excluded. The increased PI and blood flow velocity in our study was an unexpected result for us, although it should be borne in mind that this was a new model, with unknown pathogenesis and progression. We acknowledge that it is, as yet, an underinvestigated model, in which all parameters are open to further investigation, and it will doubtless contribute to our knowledge of the pathogenesis of infantile communicating hydrocephalus.

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References


Real-Time Surgery on The Internet

TO THE EDITOR: About a month ago we performed a brain tumor surgery, which was transmitted (broad-band) live over the internet (www.Second-opinion.com and www.NonStop.co.il). The project aroused interest, and although we had only 10 hours of advance notice and promotion, there were about 5000 “hits” and e-mails from 15 countries.

We are uncertain about the new situation. Was this a