slight reduction in ventricular size, and CSF pressure measured 170 mm H2O.

Facial myokymia in this patient was associated with clinical and radiological signs of obstructive communicating hydrocephalus. Ataxia of gait, urinary and fecal incontinence, and the facial myokymia disappeared following insertion of the VA shunt, suggesting that the symptoms were interrelated. Since neither CT nor auditory evoked potential studies revealed evidence of a structural brain-stem lesion or extra-axial facial nerve pathology, it is believed that the facial myokymia in this case was secondary to obstructive communicating hydrocephalus. Thus, the presence of facial myokymia does not necessarily indicate a localized primary structural abnormality confined to the pontine region.  

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References

Blood-Brain Barrier in SAH

TO THE EDITOR: I would like to congratulate Drs. Peterson and Cardoso for their excellent observation on blood-brain barrier permeability following experimental subarachnoid hemorrhage (SAH) (Peterson EW, Cardoso ER: The blood-brain barrier following experimental subarachnoid hemorrhage. Part 1: Response to insult caused by arterial hypertension; Part 2: Response to mercuric chloride infusion. J Neurosurg 58: 338-351, March, 1983). I would like to make some comments.

Trojanowsky has noted an early profound increase in capillary permeability (also measured with Evans blue dye) in cats suffering from SAH produced by puncturing the internal carotid artery. The greatest leakage was seen in animals with arterial hypertension. In addition, Shigeno, et al., have observed extravasation of Evans blue dye only in cats with both SAH and arterial hypertension. Peterson and Cardoso failed to find demonstrable blood-brain barrier lesions in cats after intracisternal administration of whole blood. Moreover, they found that in cats with SAH that were rendered hypertonic with an intravenous injection of metaraminol, there was no leakage of Evans blue dye, while the same intravenous injection of the drug caused a marked extravasation of dye in the control animals. We have found increased barrier permeability after SAH in rats with no arterial hypertension and only slightly elevated intracranial pressure. There is also controversy concerning the mechanism of interaction of blood substances with endothelial cells after SAH.

Peterson and Cardoso point out that pinocytosis and maintenance of tight junctions are active cellular mechanisms, requiring consumption of energy. Pinocytosis is a morphological sign of increased barrier permeability. The authors hypothesize that experimental and clinical SAH give rise to generalized inhibitory mechanisms, including cerebral metabolism. Inhibition of the endothelial cell metabolism suggested as a manifestation of the inhibitory action of subarachnoid blood should prevent pinocytosis. The authors proposed that the absence of blood-brain barrier leakage after injection of mercuric chloride in their animals with SAH resulted from a lack of pinocytosis due to the decreased endothelial cell metabolism. They did not comment on the fact that a lowered endothelial cell metabolism should fail to maintain tight junctions, resulting in increased barrier permeability.

In our investigations (T Dóczi, et al., unpublished data), blood-brain barrier permeability in the early stage of SAH was found to have clinical significance in predicting the outcome, morbidity, and mortality of the disease.

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References

RESPONSE: Drs. Dóczi and Huszka cite Shigeno, et al.,7 and Trojanowsky,8 who observed early breakdown of the blood-brain barrier (BBB) demonstrated by extravasation of Evans blue dye following subarachnoid hemorrhage (SAH). We believe that the differences reported by these observers are related to the mechanism of production of SAH. They punctured the internal carotid artery to produce SAH, which led to a significant elevation of the intracranial pressure (ICP) with a consequent severe decrease of the cerebral blood flow (CBF). Such changes are enough to cause BBB breakdown.1,6 In our experiments, we isolated the effect of subarachnoid blood by a slow cisternal injection of blood with controlled pressure monitoring. This avoided a sudden rise in the ICP and any effect that it might have on the BBB response. The ICP never exceeded a level of 40 mm Hg below the diastolic blood pressure.
We observed no BBB breakdown to Evans blue dye in our animals when mercuric chloride infusion and arterial hypertension followed SAH. Despite this lack of evidence of breakdown, we realize that some degree of BBB dysfunction had occurred that, with a longer period of observation, might have resulted in leakage of dye. This is analogous to the response to ischemia that first produces dysfunction and, later, BBB breakdown. We believe that the results of Shigeno, et al., and Trojanowsky complement rather than contradict our own. We assume that the presence of blood in the subarachnoid space leads to early BBB dysfunction, with later leakage of Evans blue dye. However, if the SAH is accompanied by significant ICP elevation and compromise of the CBF, then the BBB breakdown occurs sooner and is more intense.

Drs. Döczy and Huszka also claim that the outcome after SAH correlates with the degree of initial BBB breakdown. This may merely reflect the severity of hemorrhage and its attendant effect on the ICP and CBF.

The mechanisms responsible for BBB breakdown are incompletely understood. Increased endothelial cell pinocytosis seems to be the main factor; however, the role of the tight junctions and the transendothelial channels remains unclear. The tight junctions have been observed to widen after acute arterial hypertension. However, metabolic inhibition of the endothelial cells or traumatic BBB damage cause a decrease of pinocytosis, without affecting the tight junctions.

References

Primary Intranasal Encephalocele
To The Editor: In the article by Drs. Choudhury and Taylor (Choudhury AR, Taylor JC: Primary intranasal encephalocele. Report of four cases. J Neurosurg 57:552–555. October, 1982), it is clearly demonstrated how difficult it may be to correctly diagnose an intranasal encephalocele without endangering the patient by causing cerebrospinal fluid rhinorrhea and a subsequent meningitis. Our experience in a series of nine patients was similar to theirs. In eight of these nine patients, ear, nose, and throat specialists performed punctures, biopsy, or at least partial removal of the lesions, and the procedures were followed by serious complications in nearly all cases. It is our strong conviction that every rhinologist should know that these simple intranasal procedures are contraindicated if a meningoencephalocele is suspected. In order to clarify the features that should raise such a suspicion, we emphasize the following points:

1. Age of the Patient. An intranasal mass in a newborn or young child is usually a meningoencephalocele, whereas in older children, and particularly in adults, it is probably a polyp.

2. Position of the Intranasal Mass. With the exception of polyps originating in the posterior ethmoid cells, all ordinary nasal polyps are located lateral to the middle concha bone. Almost every intranasal meningoencephalocele is situated medial to the middle concha bone.

3. Pedunculation of the Mass. Unlike the nasal polyp, the meningoencephalocele is rarely pedunculated and, if it is, then only partially. Over most or all of its medial side the mass is connected directly to the nasal septum. This anatomical relationship can usually be detected by simple inspection, but palpation with a probe may be even more instructive. The probe can be introduced high up in the nose, passing along the lateral side of the meningoencephalocele, but on the medial side its passage will be stopped by the mostly broad connection of the nasal septum and the meningoencephalocele. In the case of a polyp, the probe can be forcefully introduced high up in the nose along the medial side of the mass.

4. Pulsations and Fürstenberg Sign. A meningoencephalocele may pulsate synchronous with the heartbeat and respiration. Compression of the jugular veins may produce swelling of the mass (Fürstenberg sign). Polyps are not associated with pulsations or the Fürstenberg sign.