Letters to the Editor

Migraine Headache Intensity and Intracranial Pressure

In their article “Migraine Aggravation Caused by Cephalic Venous Congestion” (Doepp F, Schreiber SJ, Dreier JP, Einhäupl KM, Valdueza JM. Headache. 2003;43:96-98), Doepp et al reported that 68% of patients with migraine experienced increased headache intensity when a Queckenstedt maneuver was performed with the patients supine. They concluded that the increased headache intensity was caused by intracranial venous distension. This conclusion may be incorrect.

Schumacher and Wolff reported that by increasing intracranial pressure and thereby reducing intracranial vascular distension, they were able to reduce the intensity of the pain in histamine-induced headache. Increasing intracranial pressure did not, however, reduce pain intensity in patients with migraine. They concluded that the pain of migraine was not due to distension of intracranial vessels.

Doepp et al make the assumption that in the upright position cerebral blood drainage occurs “via the intraspinal and extraspinal venous system.” Principal outflow through the internal jugular veins can indeed be substituted completely by the large vertebral plexus through communications at the cranial base. One would have to ask why this happens when the patient is upright, but not when the patient is supine. There should be adequate drainage even when the jugular veins are occluded.

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References

Response From Doepp

Dr. Shevel raised 2 objections to our article, “Migraine Aggravation Caused by Cephalic Venous Congestion.” First, Dr. Shevel cites an experimental study from Schumacher and Wolff published in 1941, wherein an increase of intracranial pressure (ICP) provoked by connecting the subarachnoid space with a high column of fluid was produced in 7 patients during a migraine attack. The authors assumed that the increase in ICP reduced vascular distension of the basal cerebral arteries. No decrease of headache intensity was observed. Dr. Shevel proposes that these findings argue against the hypothesis that cerebral venous distension is involved in the increase of migraine headache. Even so, several animal studies have shown that an increase in cerebrospinal fluid (CSF) pressure does not cause compression of intracranial arterial or venous vessels; elevation of CSF pressure leads to a marked dilatation of pial arteries and a minor dilatation of pial veins. In another study, the caliber of pial veins remained unchanged during ICP changes due to mannitol infusion in cats with normal and elevated ICP. Bridging veins become only slightly smaller without compression or collapse.

Second, we did not argue that extrajugular venous drainage in a supine position is impossible. Instead, we stated that in physiological circumstances the cerebral venous blood in a horizontal position drains mainly through the internal jugular veins (IJVs). Indeed, bilateral IJV compression in the supine position leads to a sufficient cerebral venous drainage via extrajugular pathways above the intraspinal and extraspinal venous...
system. This is also easily confirmed by the clinical observation that even bilateral radical neck dissection with removal of both IJs is normally well-tolerated despite a horizontal position. Taken together, however, these results do not contradict the assumption that bilateral IJV compression causes a brief increase in cerebral venous pressure and consecutive vessel distension before normalization due to drainage via alternative venous pathways occurs. The observation that the increase in migraine headache in our patients started immediately with the Queckenstedt maneuver supports this hypothesis. Furthermore, it is well-known that a complete venous drainage adaptation following bilateral IJV removal may require up to several months. Finally, 32% of our patients did not experience headache worsening during a Queckenstedt maneuver. This correlates well with our recent findings that in approximately 25% of all humans extrajugular drainage pathways are also relevant in the supine position. Prolonged compression maneuver with the potential for eventual return to baseline headache intensity was not performed in our study.

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REFERENCES


