Hemicraniectomy for Ischemic Stroke: Temerity or Death Cure?

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When we twist our ankle it swells promptly in minutes. But brain swelling after a stroke is a strange beast for its amazing delay: although some swelling is manifest within the first day, edema does not peak until 3 – 5 days after the stroke. This was already recognized 40 years ago.[1] A sizeable number of patients may show brain mass effect at intervals longer than one week.[2] Ischemic swelling of brain was initially described to "simulate an acutely expanding lesion".[2]

Ischemic edema really is a rapidly expanding tumor from a clinical point of view, and is the most frequent cause of death in acute ischemic stroke.[3] The delay in the onset of ischemic edema was initially thought to be a second infarct.[2] However, in light of the fact that second strokes are relatively less common in the immediate period after a first stroke[4] this seems unlikely *a priori*. Not long after recognition of ischemic cerebral edema as a lethal entity, the difficulty of treatment was recognized.[5]

The question in the pair of articles by Demchuk and Wijdicks in this issue is whether this ischemia-associated edema can be treated by removing a large portion of the hemicranium. Demchuk argues yes, and underscores that the size of the bone flap must be massive for this procedure to allow unhindered brain swelling. Such unfettered expansion of ischemic brain may be less likely to produce brain necrosis compared to edematous brain abutting against the cranium. We just don't know.

Clearly, as Wijdicks emphasizes, this procedure is not for the timid, and to obtain benefit, must be done in an "all or none" fashion because of the geometry of the herniating brain, which acts like a sphere. In plain English: a huge whack of skull must be removed, later to be put back in place after storage in antibiotics or in the patient's own peritoneum. Neurosurgically, it is trivial to do such a large craniectomy, leaving patients, their families (and possibly neurologists!) to be counted among the timorous, with respect to this procedure.

But it is not the psychological reaction to removing half of someone's skull for a few months that counts scientifically: it is fear of converting the dead into the impaired living. Some very useful cautions for hemicraniectomy trials are emphasized by Wijdicks, including control for identical postoperative handling to that in control patients. The reason is that a spurious positive result may come of a trial studying hemicraniectomy if postoperative patients receive more attention. Reducing stroke size could come from closely watching damaging high blood sugars, or high temperatures. Hence, monitoring and control of physiologic parameters must be identical in the untreated and surgically treated groups. Wijdicks, who is clearly not against a hemicraniectomy trial and is participating in the design of one, voices another caution: the natural course of malignant cerebral edema may be more favorable in younger patients and hence aged-matching is at least as important as it is in other clinical trials.

Still, the concern that supercedes all others here is that we not convert the dead into the severely impaired living. Demchuk's enthusiasm is supported by a reference that there can be good long-term outcome in survival of hemicraniectomy.[6] Demchuk also provides evidence that cerebral blood flow and infarct size is reduced by opening the skull to expansion. We are not treating increased intracranial pressure, a pre-terminal event.

Certainly, with the joint experience of these two authors in clinical treatment in ischemic stroke, a trial is worthwhile given the above considerations for trial design, and other concerns voiced in the accompanying article. The old teaching that ischemic cerebral edema is an intrinsic disorder of the parenchyma and is not amenable to treatment by hemicraniectomy must be challenged. Of course, the procedure may be useless, as neurosurgeons who have done hemicraniectomy for brain trauma might attest. Yet we must know the answer *in stroke*, since this treatment may not only be life saving but produce individuals with little neurologic deficit who might have died. To know, we must do the trial. To do the trial, we must do it well. To do the trial well, read the accompanying articles.

Roland N. Auer
Calgary, Alberta

References

1. Shaw C-M, Alvord EC, Berry RG. Swelling of the brain following ischemic infarction with arterial occlusion. Arch Neurol 1959;1:161-177.
2. Ng LKY, Nimmannitya J. Massive cerebral infarction with severe brain swelling. Stroke 1970;1:158-163.
3. Bounds JV, Wiebers DO, Whisnant JP, Okazaki H. Mechanisms and timing of deaths from cerebral infarction. Stroke 1981;12:474-477.
4. Anonymous. Low molecular weight heparinoid, ORG 10172 (danaparoid), and outcome after acute ischemic stroke: a randomized controlled trial. The Publications Committee for the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) Investigators. JAMA1998;279:1265-1272.
5. O'Brien MD. Ischemic cerebral edema. A review. Stroke 1979;10:623-628.
6. Rengachary SS, Batnitzky S, Morantz RA, Arjunan K, Jeffries B. Hemicraniectomy for acute massive cerebral infarction. Neurosurgery 1981;8:321-328.