Decompressive craniectomy for acute stroke: early is better

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Decompressive craniectomy for large, life-threatening supratentorial infarcts has been considered a controversial procedure for a long time. In the past 2 decades, authors of numerous single-center series have documented improved survival after decompressive craniectomy compared with maximal medical therapy. However, the procedure is not universally accepted. Detractors have been most vocal about the lack of Level A evidence and the danger that surgery could lead to a high proportion of survivors with severe disability. This controversy has sparked a few randomized clinical trials, and a landmark report of a pooled analysis of 3 such trials has answered some of the questions raised by critics. Among patients younger than 60 years old with a large middle cerebral artery infarct treated within 48 hours of stroke onset, the mortality rate decreased from 78% in the medically treated group to 29% in the decompressive craniectomy–treated group. This difference was statistically significant. The authors of this report also concluded that 2 patients must be treated to prevent 1 death regardless of functional outcome. Four patients must be treated for each patient with a modified Rankin Scale score of 3 or less (a score most would agree corresponds to a decent quality of life). Despite these “high-quality” scientific data and Level A evidence, several questions remain unanswered. In particular, is there a “neuroprotective” role for therapies (including decompressive craniectomy) aimed at decreasing the secondary insult from vasogenic edema? In this issue of the Journal of Neurosurgery, Walberer and coworkers report their findings from an experimental model of reversible middle cerebral artery occlusion. These authors aimed to study the effects of vasogenic edema on infarct volume and functional outcome. To eliminate the space-occupying effects of postinfarction edema, a bilateral decompressive craniectomy was prophylactically performed before reversible cerebral ischemia; the control group consisted of sham-operated animals. Infarct size was measured on MR images obtained 5 and 24 hours after ischemia onset. Ischemic lesions were consistently smaller in the craniectomy group at both the 5- and 24-hour time points. Clinical scores were also significantly better and the midline shift was less in rats that had undergone prophylactic craniectomy. These observations lend further support to the notion that vasogenic edema aggravates the extent of infarction. Thus, therapies targeting vasogenic edema and its deleterious effects can prove neuroprotective. Hypothetically, vasogenic edema around the ischemic tissue causes regional compression of leptomeningeal vessels providing critical collateral blood flow to the penumbra. Indeed, experimental studies have suggested that decompressive craniectomy improves cortical perfusion by increasing local cerebral blood flow through leptomeningeal collateral vessels. In the study by Walberer et al., animals were killed after 24 hours. Note, however, that postinfarction edema progresses well beyond that time point, and we don’t know for sure if the protective effect demonstrated in their experimental setting is maintained beyond 24 hours. An extension of this study might address this issue in the future. Can we use these observations to propose an ultraearly decompressive craniectomy and very aggressive intracranial pressure–targeted therapies in patients with acute cerebral infarction to improve functional outcome? The easy answer is that randomized clinical trials are required. Experience shows that such trials are very difficult, expensive, and impractical to perform and often do not provide definitive answers. The decision to proceed with a decompressive craniectomy in a patient with a life-threatening infarct is always difficult because of the significance and impact (on the quality of life of the patient as well as the caregiver[s]) of the residual neurological deficits. Nonetheless, when craniectomy is considered, data from the following and other studies suggest that early is better.

References
Response

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We thank Dr. Lanzino for his thoughtful comments. Our study reveals the impact of the space-occupying effect of vasogenic edema formation for the propagation of a lesion in acute ischemic stroke. This phenomenon has been largely underestimated in the past, although it may be important with respect to surgical interventions as well as for pharmacological approaches targeting blood–brain barrier breakdown in acute stroke. Reducing the space-occupying effect of vasogenic edema formation—surgically or pharmacologically—might provide “secondary neuroprotection” and improve functional outcome and the quality of life in patients suffering from a large territorial stroke.

With respect to decompressive craniectomy, “early is better” exactly sums up the conclusion that should be drawn from our data, although we agree with Dr. Lanzino that additional preclinical work should be completed before the first clinical trials. Such trials in fact would be expensive and difficult, although not impossible. Our experimental data indicate that more than 50% of the ischemic lesion volume may be a result of “collateral damage” due to edema formation. We hope that this finding encourages neurologists and neurosurgeons to launch randomized controlled trials in this field. (DOI: 10.3171/JNS/2008/109/8/0285)