Hemicraniectomy for Ischemic and Hemorrhagic Stroke
Facts and Controversies

Aman Gupta, MD[a,b,c], Mithun G. Sattur, MD[a,b,c],
Rami James N. Aoun, MD, MPH[a,b,c], Chandan Krishna, MD[a],
Patrick B. Bolton, MD[d], Brian W. Chong, MD, FRCP(C)[a,e],
Bart M. Demaerschalk, MD, MSc, FRCP(C)[f],
Mark K. Lyons, MD[a], Jamal McClendon Jr, MD[a],
Naresh Patel, MD[a], Ayan Sen, MD[g], Kristin Swanson, PhD[a,b],
Richard S. Zimmerman, MD[a],
Bernard R. Bendok, MD, MSCI[a,b,c,e,h,*]

KEYWORDS

• Decompressive craniectomy • Malignant infarct • Cerebellar stroke • Modified Rankin score
• Cerebral edema • Surgery for stroke

KEY POINTS

- Acute occlusion of a proximal large intracranial artery has high risk of progression to malignant infarct.
- The results of best medical management including aggressive ICU measures are dismal, with mortality of up to 80%.
- Decompressive craniectomy (DC) is demonstrated to conclusively improve mortality to around 30%.
- DC also improves the chances of good functional outcome when performed within 48 hours in patients younger than 60 years of age but should be considered beyond these circumstances on a case-by-case basis.
- Better indicators of edema progression, patient eligibility, and clinical outcome measures are urgently required for use in larger randomized controlled trials.
- DC also serves an important role in managing select patients with supratentorial spontaneous hemorrhage, and posterior fossa hemorrhage and ischemic stroke.

[a] Department of Neurological Surgery, Mayo Clinic Hospital, Mayo Clinic, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA; [b] Precision Neuro-theraputics Innovation Lab, Mayo Clinic Hospital, Mayo Clinic, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA; [c] Neurosurgery Simulation and Innovation Lab, Mayo Clinic Hospital, Mayo Clinic, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA; [d] Department of Anesthesia & Periop Med, Mayo Clinic Hospital, Mayo Clinic, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA; [e] Department of Radiology, Mayo Clinic Hospital, Mayo Clinic, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA; [f] Department of Neurology, Mayo Clinic Hospital, Mayo Clinic, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA; [g] Department of Critical Care Medicine, Mayo Clinic Hospital, Mayo Clinic, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA; [h] Department of Otolaryngology, Mayo Clinic Hospital, Mayo Clinic, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA
* Corresponding author.
E-mail address: bendok.bernard@mayo.edu

Neurosurg Clin N Am 28 (2017) 349–360
http://dx.doi.org/10.1016/j.nec.2017.02.010

INTRODUCTION

Progressive global or local intracranial mass effect is frequently encountered with large hemispheric infarcts ("malignant infarcts"[1]). The resulting increased intracranial pressure (ICP) results in the potential for worsened outcomes and dramatically increased mortality. Decompressive craniectomy (with capacious duraplasty; DC) is a highly effective procedure that is often warranted in such situations. DC for ischemic stroke has been conclusively proven to reduce mortality in large hemispheric infarcts[2] and is a powerful tool in a comprehensive neurovascular team's armamentarium. Despite the utility of DC, there are schools of thought that discourage surgery because of the biased perception that patients survive but are left with severe burdensome disability. This article discusses the available literature and demonstrates the effectiveness of DC in malignant infarction and functional outcome. Also discussed is the role of DC in posterior fossa (cerebellar) stroke and spontaneous intracerebral hematomas (ICH).

ISCHEMIC HEMISPHERIC STROKE (SUPRATENTORIAL)

Epidemiology

The prevalence of malignant ischemic stroke is reported to be between 2% and 8%[3–5] of all patients with ischemic stroke. The mortality rate of patients with malignant stroke who undergo aggressive nonoperative management is in the range of 40% to 80%.[6] Performing a DC can reduce this mortality rate to 30% (**Table 1**).

Pathophysiology of Malignant Stroke

Microscopic and cellular changes

Neuronal death in ischemia occurs through apoptosis, necrosis, and autophagy.[7] Briefly, cellular events begin with deprivation of glucose and oxygen, energy failure, ATP depletion and loss of ion exchange function of membrane pumps, terminal depolarization and glutamate-mediated calcium excitotoxicity, calcium-dependent enzyme activation, generation of free radicals, and ultimately degradation of cellular molecules. Both cytotoxic edema (from above) and vasogenic edema (later on from blood-brain barrier disruption) occur in malignant stroke. Identifying individual genetically determined factors of cell death and inflammation could serve as molecular markers of malignant stroke risk in a given patient.

Macroscopic pathology

Within the macroscopic region of infarct, there are well-described zones of decreasing cerebral blood flow and increasing impairment of function[8] (**Fig. 1**). This concept of ischemic penumbra and potentially salvageable brain tissue is central to strategic implementation of therapies including DC. Animal studies have convincingly demonstrated improvement in cortical perfusion, reduction in infarct size, and clinical function following experimental middle cerebral artery (MCA) occlusion.[9] Progressive swelling of infarct tissue leads to transtentorial herniation in a craniocaudal direction and midline shift with subfalcine herniation.[10] Continued swelling can recruit additional arterial territories (posterior and anterior cerebral arteries, respectively) resulting in multiterritory infarction and worsened outcome.

Predictors of Progression

Time since stroke

Cerebral edema progresses during the first 24 to 48 hours after the onset of ischemic stroke and can result in herniation after Day 2[3] (although herniation can occur earlier than this in some cases).

Table 1
Existing evidence on decompressive craniectomy

Trial	Year	Age (y)	No. of Patients (n)	Time to Surgery from Onset of Stroke (h)	Medical Arm		Surgical Arm	
					Mortality (%)	mRS >4 (%)	Mortality (%)	mRS >4 (%)
Destiny-I	2007	18–60	32	12–36	53	73	17.6	53
Decimal	2007	18–55	38	<24	77.8	23	25	50
Hamlet	2009	18–60	64	<96	59	15	22	53
Pooled Analysis	2007	18–60	32	<48	71	7	22	35
Destiny-II	2011	>61	49	<48	70	28	33	60
HeADDFIRST	2014	18–75	26	<96	40	60	21	72