Acute hemispheric cerebral infarction in the territory of the middle cerebral artery (MCA), and sometimes including the territories of the anterior cerebral artery and/or the posterior cerebral artery, may cause severe life-threatening brain edema in approximately 10% of patients. It is associated with raised intracranial pressure (ICP) and brain herniation known as malignant MCA infarction. Although mortality rates for unselected groups of patients with MCA infarction range from 30% to 60%, the death rate in patients who develop malignant hemispheric infarction is as high as 80%, even with maximum conservative intensive care. Because of the limitations of conservative management, decompressive surgery has been proposed in patients with neurologic deterioration caused by malignant hemispheric infarction. This therapy is presumed to limit brain tissue shifts, to decrease ICP, and to preserve cerebral blood flow, thus preventing secondary brain damage. The technique of decompressive surgery is relatively simple and consists of a large hemicraniectomy and a duroplasty and/or internal decompression. Animal studies have shown that decompressive surgery reduces mortality and improves functional and histologic outcome. In the guidelines of the American Heart Association published in 1994, there was general agreement to recommend surgical decompression and evacuation of large cerebellar infarctions that compress the brain stem (Levels of Evidence III through V, Grade C). However, they described that surgical decompression and evacuation of a large hemispheric infarction can be a life-saving measure, but survivors may have severe residual neurological deficits (Levels of Evidence III through V, Grade C). However, after this guideline, case reports and retrospective studies suggested that hemicraniectomy lowers mortality without increasing the number of severely disabled survivors. This finding has been confirmed in two large recent prospective series of clinical and computed tomographic evidence of acute severe MCA infarction. In the first series, 32 patients were prospectively selected for surgical decompression and 21 patients were treated conservatively. Mortality was reduced from 79% in controls to 34% in surgically treated patients, and poor functional outcome from 95% to 50%. The mean interval between the onset of symptoms and surgery was 39 hours. In a subsequent study, in which hemicraniectomy was performed in 31 patients within 24 hours after the onset of symptoms, mortality was reduced even further, to 16%, only with Rankin Score of severe handicap in 13% of patients. Moreover, early hemicraniectomy led to a significant reduction in the length of time critical care therapy was needed. Operative complications had no effect on patient outcome in two series. In another small prospective series of 19 patients, hemicraniectomy also reduced mortality and improved short-term clinical outcome (Glasgow Outcome Scale at 3 months) as compared with a nonrandomized 15 control patients. Although the two larger prospective studies suggest a substantial benefit of decompressive surgery as compared with medical therapy alone, they have weak points of nonrandomized group selection, small number of cases, and insufficient information on functional outcome. Conclusively, surgical decompression has been advocated to improve the mortality and morbidity of malignant MCA infarction. However, for level I evidence and the functional outcome, the results from the large randomized prospective study is needed.