

Decompressive Hemicraniectomy with Duraplasty: A Treatment for Large-Volume Ischemic Stroke

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Abstract: Increased intracranial pressure (ICP) is a cause of death and disability in neurological patients. Patients experiencing malignant stroke of the middle cerebral artery (MCA) have a high mortality related to cerebral edema, increased ICP, and subsequent cerebral herniation. Decompressive hemicraniectomy with duraplasty is a surgical option for those experiencing large volume MCA stroke. When decompressive hemicraniectomy with duraplasty is performed, functional outcomes improve if the MCA stroke candidate is younger, the onset of increased ICP occurred less than 24 hours before surgery, and surgery is performed before clinical signs of herniation syndrome occur. The level of care required for these patients makes nursing care challenging.

Cerebrovascular disease is the third most prevalent cause of death in the United States, behind cardiovascular disease and cancer (CDC, 2002). Ischemic stroke occurs every 45 seconds, killing someone every 3 minutes (AHA, 2004). While prevention is the best answer to this problem, providing care for those who experience ischemic stroke is a monumental task. In the past decade, there have been advances in stroke care, including the use of thrombolytic therapy. Nonetheless, patients with large ischemic strokes continue to have high mortality and morbidity rates. Decompressive hemicraniectomy with duraplasty (DHWD) is a treatment modality for patients who experience large ischemic strokes with a consequent rise in intracranial pressure (ICP). This article presents a summary of diagnostic and clinical indicators for decompressive hemicraniectomy and highlights nursing care for increased ICP and the DHWD patient.

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Background

Increased ICP is sustained ICP greater than 20 mmHg. It may have many causes, among them traumatic brain injury, subarachnoid hemorrhage, intracerebral hemorrhage, intraventricular hemorrhage, and ischemic stroke. The Monro-Kellie hypothesis explains the concept of ICP. Within the nondistensible cranial vault there is room for the brain, blood, and cerebrospinal fluid (CSF). An increase in the volume of any one of these components can cause increased ICP. Minor changes in volume do not increase ICP because of compensatory mechanisms. When these mechanisms fail, even small increases in volume increase ICP. Failure to treat increased ICP could cause cerebral herniation and death. Cerebral herniation occurs as brain tissue shifts from high pressure to lower pressure downward toward the medulla and the opening of the foramen magnum (Arbour, 2004). Death occurs with medullary compression and subsequent loss of control over vital functions, such as respirations, blood pressure, cardiac function, and vasomotor tone (Arbour, 2004; Hickey, 2003).

Clinical Signs of Increased ICP

Clinical indicators of increased ICP range from subtle to overt. When nurses care for any patient who has—or may develop—increased ICP, they must be diligent in assessing the patient and reporting indicators of increased ICP. Indicators include decreased level of consciousness, confusion, headache, projectile vomiting, unequal pupils (anisocoria), and the presence of a pronator drift or motor weakness (Hickey, 2003). Patients with malignant infarction typically present with severe stroke syndrome including hemiplegia, forced eye and head deviation, and progressive decrease in consciousness (Gupta, Connolly, Mayer, & Elkind, 2004). Because of severe neurological impairment, the patient is usually intubated. Therefore clinicians must rely on pupillary response, signs of early herniation and ICP monitoring to recognize and treat increased ICP.

Vital signs may change as ICP increases. Hypertension is an early response to increased ICP. As ICP rises, the body reflexively becomes hypertensive to increase the cerebral perfusion pressure (CPP). This compensatory mechanism attempts to maintain brain perfusion despite increasing ICP. Indwelling monitoring systems

measure ICP directly. Clinicians can calculate CCP by taking the mean arterial pressure (MAP) and subtracting the ICP (i.e., $MAP - ICP = CPP$). Generally, the CPP is above 60 mmHg in a normal adult, which ensures adequate perfusion to the brain (LeJune & Howard-Fain, 2002). Below 50 mmHg, CPP is associated with cerebral ischemia and can lead to infarction. If increased ICP is not treated, further vital sign changes may occur. Cushing's triad, which comprises systolic hypertension, bradycardia, and bradypnea, is a late response attributed to pressure on the medullary centers of the brain (Hickey, 2003). Brain death occurs as the increase in pressure reduces blood flow to the brain.

Management of Increased ICP

For individuals with increased ICP, interventions aim to reduce intracranial volume and pressure. Measures to lower increased ICP include draining CSF with external ventricular devices, evacuating blood if a hemorrhage is present, craniotomy with selective brain amputation, and hyperosmolar therapy, such as mannitol, to decrease brain edema (Coplin et al., 2001). The goal is to raise the CPP by lowering ICP. Hypertension may also be induced to improve CPP, but doing so is exquisitely difficult in the ischemic stroke population (Hickey, 2003). Other measures that decrease ICP include maintaining normothermia or inducing mild hypothermia (32–34° Celsius) and preventing hypercapnia (Georgiadis, Schwartz, Aschoff, & Schwab, 2002; Hickey, 2003; McDonald & Carter, 2002).

Some medical measures to control ICP could put the patient at risk for complications. Hyperventilation should be avoided in patients with increased ICP because it constricts the cerebral vessels, which may lead to cerebral ischemia (AANN, 2004). While sedatives may be indicated to calm an agitated patient, sedation may mask neurological findings. Hypervolemia used to induce hypertension could put a patient at risk for pulmonary congestion. Medications to increase blood pressure also increase cardiac workload and could induce myocardial damage.

The nurse caring for a patient with a brain insult must know interventions that positively and negatively influence ICP. To promote venous outflow and subsequently decrease ICP, patients should be positioned with elevation of the head of bed approximately 30° as long as the blood pressure is stable (Beitel, 1998; Hickey,

Table 1. Diagnostic Studies for Patients with Suspected Acute Ischemic Stroke

Diagnostic Study	Reason Performed
Brain computed tomography (CT) scan	Identify hemorrhagic or ischemic stroke
Brain magnetic resonance imaging (MRI) scan	Locate and quantify area of infarction and penumbra
Electrocardiogram	Check for acute myocardial infarction, cardiac ischemia, or presence of dysrhythmias
Blood glucose	Check for hypoglycemia or hyperglycemia
Serum electrolytes	Identify electrolyte imbalance
Renal function tests	Identify baseline renal function identify if dysfunction exists
Complete blood count	Identify baseline, identify if anemia is present
Platelet count, PFA-100	Identify if thrombocytopenia is present and determine if platelet dysfunction exists
Prothrombin time/international normalized ratio, and activated partial thromboplastin time	Baseline coagulation studies, necessary before administering thrombolytic agents or surgical intervention

2003; McLean, 2001). Because head elevation can lower blood pressure, it should be avoided in patients with hypotension or in those with precipitous blood pressure drops on position change. Neck flexion, hyperextension and rotation should be avoided, and the head should be left in a neutral position to promote venous outflow to minimize rises in ICP (Hickey, 2003). Nurses should take measures to prevent patients from coughing, vomiting, shivering, tremoring, posturing, and straining for bowel movements, all of which increase ICP (Hickey, 2003). Nursing activities known to increase ICP that should be avoided or limited include tracheal suctioning, noxious stimulation, painful procedures, and clustering of activities (Hickey, 2003).

Ischemic Stroke

Despite medical management, mortality remains high among patients experiencing high-volume infarcts. Mortality is the highest when the distal internal carotid artery or the proximal middle cerebral artery is occluded; this condition is referred to as a *malignant stroke* (Lanzino & Lanzino, 2000). Hacke et al. (1996) reported that despite medical management, increased ICP, herniation, and death occur within 5 days in up to 78% of patients with complete middle cerebral artery territory infarction.

Adams et al. (2003) state that all patients who might have suffered an ischemic stroke should receive the following diagnostics: a brain computed tomography (CT) scan, brain magnetic resonance imaging (MRI) scan, electrocardiogram, blood glucose, serum electrolytes, renal function tests, complete blood count (CBC) including platelet count, prothrombin time/international normalized ratio, and activated partial thromboplastin time (Table 1). Subtle hypodense changes are present on brain CT in ischemic stroke patients within 24 hours of injury. MRI further reveals stroke severity by showing quantity of infarcted brain tissue and the ischemic penumbra. The larger the area of infarction, the greater

the risk for increased ICP and edema. Using an MRI may also help to pinpoint the vessel involved. Both a CT and an MRI scan can be used to detect presence of midline shift or herniation, which are signs of substantial cerebral edema (Adams et al., 2003). Schwab and Hacke (2003) favor immediate diffusion- and perfusion-weighted MRI studies to allow early detection of infarct size and vessel involved.

Decompressive Hemicraniectomy with Duraplasty

Decompressive hemicraniectomy with duraplasty (DHWD) is a surgical procedure designed to decrease ICP. More room becomes available after removal of part of the skull and release of the dura, thereby reducing ICP. Smith, Carter, and Ogilvy (2002) report a 15% decrease in ICP with craniectomy and a 70% decrease in ICP after release of the dura. Thus, opening both the skull and dura affords the greatest benefit.

The concept of removing part of the skull to alleviate increased ICP is not new. It dates back to 1901, when Kocher reported the first surgical decompression for post-traumatic brain swelling (Smith et al., 2002). Decompressive hemicraniectomy has not been widely used because of the lack of evidence of benefit in functional outcomes (Gupta et al., 2004; Schwab & Hack, 2003). Decompressive hemicraniectomy for severe ischemic stroke currently is being investigated in major clinical trials, such as the Hemicraniectomy after MCA Infarction with Life-threatening Edema Trial (HAMLET), Hemicraniectomy and Durotomy for Deterioration from Infarction Related Swelling Trial (HeaDDFIRST), Hemicraniectomy and Moderate Hypothermia in Patients with Severe Ischemic Stroke and Hemicraniectomy for Malignant Cerebral Artery Infarcts (HeMMI) (Internet Stroke Center, 2004). Inclusion and exclusion criteria differ by study and outcome measures generally include mortality and functional status. Decompressive hemicraniectomy is not used more widely in the head trauma population because the ischemic process is more global. In ischemic stroke, however, the process is more focal and theoretically would be of more benefit in this population.

Hemicraniectomy involves removal of a substantial portion of the skull on one side of the head over the area of cerebral infarction. The landmarks for the portion of bone removed are anterior—frontal to the midpupillary line; posterior—posterior to the external auditory meatus; superior—to the lateral edge of the superior sagittal sinus; and inferior—to the floor of the middle cranial fossa at the origin of the zygomatic arch (Fig 1). Surgeons avoid the sigmoid and superior sagittal sinuses to prevent the possibility of sinus thrombosis or hemorrhage (Coplin, 2001).

A DHWD involves performing a cruciate opening of the dura and attaching the pericranium or a dural substitute (Ziai et al., 2003). Examples of dural substitutes

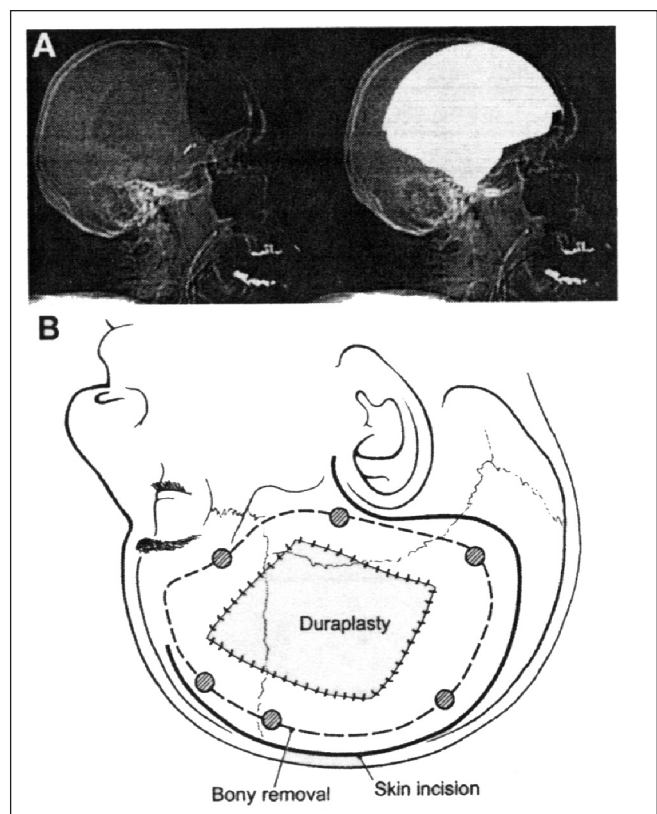


Fig 1. Proposed use of prophylactic decompressive craniectomy in poor-grade aneurismal subarachnoid hemorrhage patients presenting with associated large Sylvian hematomas. (Reprinted with permission from Smith et al., (2002). *Neurosurgery* 51, 117–124.)

include Lyoplast™, produced from bovine pericardium and Neuro-Patch™, created from polyesterurethane. The duraplasty leaves a “bag” for the intracranial volume to fill while ICP is elevated. The skin is closed in the usual manner. The bone flap is preserved in one of two ways. It can be placed in an abdominal wall surgical pouch or frozen in an antibiotic solution. The bone flap is left out of the patient’s head for 6 weeks to 5 months, depending on the patient’s speed of recovery (Iwama et al., 2003; Flannery & McConnell, 2001). It is generally agreed that it is best to save and use the patient’s own bone flap for replacement instead of using synthetic cranioplasty materials because the flaps are safe, efficient, and cost-effective (Iwana et al., 2003; Flannery & McConnell, 2001; Bruce & Bruce, 2003).

Patient Selection

Historically, DHWD was considered a “last-ditch” effort (Coplin, 2001) but more recently has gained some acceptance. Controversy persists over the criteria for acute ischemic stroke patients receiving DHWD. It is understood that no one criterion should be looked at independently when deciding if a patient is a DHWD candidate; instead the entire clinical picture should be considered.

Table 2. Functional Outcome Classifications Reported by Gupta et al., 2004

Functional Outcome Classification	Barthel Index	Modified Rankin Scale	Glasgow Outcome Scale	Outcome Percentage in Classifications
1) Functionally independent	≥ 90	0 to 1	5	42%, good outcomes reported as Group 1 or 2 58%, poor outcomes reported as Group 3 or 4
2) Mild to moderate disability	60-85	2 to 3	4	
3) Severely disabled	< 60	4 to 5	2 to 3	
4) Death				

Timing of DHWD is critical. The European Stroke Initiative (2003) states “early decompressive surgery within the first 24 hours after stroke can reduce mortality markedly” (p. 11). Schwab et al. (1998) reported a 16% mortality rate for patients receiving early hemicraniectomy, less than 24 hours after onset of signs of increased ICP, and a 34% mortality rate for patients with late surgery, more than 24 hours after first reversible signs of herniation.

Clinical indicators for DHWD include further deterioration in neurological status from admission and signs of early herniation syndrome, such as unilateral fixed and dilated pupils. Once signs of late herniation are present in patients, such as fixed dilated pupils, coma, respiratory difficulty, and extensor posturing, those patients no longer qualify as good candidates for DHWD (Lanzino & Lanzino, 2000; Schwab & Hacke, 2003). A sustained ICP greater than 25 mmHg has been used as an indicator for surgical decompression but in itself does not warrant DHWD (Lanzino & Lanzino, 2000). A CT or MRI finding of complete middle cerebral artery (MCA) infarction is an indication for DHWD (Donnan & Davis, 2003; EUSI, 2003).

Evidence of MCA infarction includes a large parenchymal hypodensity of greater than 50% of the MCA territory. Donnan and Davis (2003) suggested “in younger patients with large MCA infarcts who are developing obvious edema and with clinical deterioration, decompressive surgery should be considered earlier rather later” (p. 2307). Evidence of local brain swelling, presence of shift, and evidence of pending herniation may influence the decision for decompressive hemicraniectomy. Gupta et al. (2004) reported 52% of patients with signs of herniation before surgery had poor functional outcomes, while 32% of patients without signs of herniation before surgery had poor functional outcomes. Size, location, presence of shift, presence of herniation on CT, early clinical herniation syndrome, time from onset of stroke and ICP are all considerations in identifying patients who could benefit from DHWD. Patients under 50 years of age without comorbid factors experiencing ischemic stroke with increased ICP symptoms and MRI verification of large-volume ischemic stroke are ideal candidates for DHWD. Patients over the age of 50 years with multiple medical conditions who experience late herniation syndrome symptoms and MRI verification of large-volume ischemic stroke with substantial shift and central herniation are poor candidates for DHWD.

Outcomes

Mortality rate and functional outcomes are reported for patients with malignant acute ischemic stroke receiving DHWD. Functional outcomes are often reported using the Barthel Index, modified Rankin Scale and Glasgow Outcome Score. Gupta et al. (2004) systematically reviewed the literature on hemicraniectomy performed to relieve intracranial hypertension in patients with large hemispheric infarct patients. Gupta et al. (2004) classified outcomes in four categories in a systematic review (Table 2). A Barthel Index score less than 60 and a modified Rankin Scale rating of less than 4 indicate a poor outcome. Age was the single variable found to be statistically significant in the multivariate model. Of patients older than 50 years, 80% experienced poor outcomes, while only 32% of patients younger than 50 years had poor outcomes. Gupta et al. (2004) report good outcomes in 42% and poor outcomes in 58% of patients reviewed with DHWD for MCA infarction. Gupta et al. (2004) did not find statistical significance in outcomes of patients receiving hemicraniectomy within 24 hours of increased ICP symptoms, compared with those with decompression after 48 hours of symptom onset. Overall survival rate and functional outcomes in patients with malignant acute ischemic stroke with DHWD were better when compared with those who did not have surgery (Leonhardt et al., 2002; Pranesh et al., 2003; Schwab et al., 1998; Smith et al., 2002). Rieke et al. (1995) reported the mortality for the control group (no surgery) was 76.2%, compared with 34.4% in the hemicraniectomy group. Gupta et al. (2004) reported a 24% mortality overall for those undergoing DHWD for MCA infarction.

Complications

Complications associated with hemicraniectomy include infection, CSF leak, shunt-dependent hydrocephalus, subdural fluid collection, and seizure (Yang, Hong, Su, & Yang, 2003). One study ($N = 68$) that examined complications of patients who underwent DHWD found that shunt-dependent hydrocephalus, subdural fluid collection, and CSF leakage from the scalp occurred more frequently with DHWD than with “standard” craniotomy (Yang et al., 2003). Stroke complications include aspiration pneumonia, urinary tract infection, deep vein thrombosis (DVT) and pulmonary embolus, pressure ulcer formation, and seizures.

Nursing Care of the DHWD Patient

Once a patient returns to the neurological intensive care unit (NICU) after surgery, nursing care is aimed at preventing increased ICP and providing supportive care. In the intensive care unit (ICU), the patient receives care according to the *American Association of Neuroscience Nurses Guide to the Care of the Patient with Ischemic Stroke* (AANN, 2004). Expected outcomes for patients with ischemic stroke include freedom from injury, depression, DVT, bowel and bladder dysfunction, contractures, skin breakdown, malnutrition, seizures, respiratory complications, and increasing neurological deficits (AANN, 2004).

The DHWD patient is acutely ill and requires specialized nursing care. Such patients require hourly vital sign checks, including ICP and CPP, and hourly neurological exams. The nurse collaborates with the physician and requests parameters for acceptable vital signs, ICP, CPP, and central venous pressure (CVP). Blood pressure is maintained to ensure an adequate CPP. Patients may require fluid and vasopressors to maintain an adequate CPP. Central line placement is common for venous access, vasoactive therapy and determining the patient's fluid volume status. Acute changes in vital signs or neurological status must be reported to the physician immediately. Frequent CT or MRI scanning may be necessary to monitor associated edema and general brain progress and to determine final infarct size.

Patients normally have an ICP drainage device to allow CSF drainage when ICP is elevated. The ICP monitoring device transducer is leveled at the tragus of the patient's ear and relevelled with changes in elevation of the head of bed. Color and amount of CSF drainage must be recorded. The physician may order daily culture and sensitivity on the CSF drainage to check for the presence of bacteria. Patients receive prophylactic antibiotic therapy and antiseizure medication. The nurse should be prepared for seizures. The nurse must also maintain a patent airway and an oxygenation saturation above 90%. Pulmonary toilet is essential to achieve this goal. Nurses perform oral care every 4 hours to prevent ventilator-associated pneumonia (Schleder, Stott, & Lloyd, 2002). Enteral nutrition will be required during ventilation. Maintaining the head of bed elevated at least 30° and checking for gastric residuals is necessary to prevent aspiration.

Once extubated, the clients with dysphagia should maintain nil per os (NPO) status until a speech and language pathologist administers a swallow test. The patient should have the output from a urinary catheter documented hourly. DVT prevention is essential; preventative measures should include the use of compression stockings and subcutaneous anticoagulation, unless contraindicated. Normal thermal to mild hypothermia

regulation helps to prevent increasing brain and metabolic demands. If the client has a cooling device, the temperature of the patient and of the device is recorded. Blood glucose testing is performed and efforts made to keep the patient's blood glucose in an acceptable range using insulin coverage.

A sign should be placed above the bed alerting care providers which side of the skull is missing the bone flap and warning that the patient should not be turned onto that side. Placing the patient with a hemicraniectomy on the side without the bone can push intracranial contents inward, increasing ICP. This position also unduly stresses the duraplasty and incision line and may cause CSF leakage. The patient should be turned every 2 hours, but the head should always rest on the solid side of the cranium. The nurse should assess the hemicraniectomy site for changes in appearance, such as bulging (a sign of increased pressure), inflammation, and for CSF leakage. Any change in appearance of or drainage from the incision should be reported to the physician.

As the patient's activities increase and swelling from the site decreases, special headgear is fitted to protect the surgical site. A cast is made of the patient's head. From it, an orthotic provider custom-makes a protective helmet. Once created, the patients should wear the protective helmet at all times and remove it only for skin assessments and hygiene. If the patient is comatose or remains on bed rest, headgear is not necessary.

Discharge planning and possible long-term care require the assistance of social services. Physical therapy may be ordered to prevent contractures and maintain mobility. Including family in the plan of care is vital. The patient is often in the ICU for an extended period of time; therefore, maintaining an open dialogue with the family facilitates their adjustment to the patient's stroke and keeps them informed of the patient's progress. Cultural and spiritual factors must be identified and respected as the healthcare team plans care for the patient.

The patient will undergo a second surgery to replace the bone flap, usually 6 weeks to 5 months after the initial DHWD, depending on the patient's status. In addition to standard postsurgery care, the bone flap site should be monitored for signs of infection.

Summary

Decompressive hemicraniectomy with duraplasty is another weapon in the battle to aid patients in recovering from acute ischemic stroke and associated increased ICP. Neuroscience nurses must keep abreast of evolving treatment modalities. By understanding the indications, the surgical interventions, and postoperative care of patients undergoing hemicraniectomy, nurses are better able to provide safe, effective care, and ultimately to improve patient outcomes.

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