

Decompressive craniectomy for space occupying hemispheric and cerebellar ischemic strokes: Swiss recommendations

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Introduction

Goal of the recommendations

The general goal of these recommendations is to unify the acute management of patients with space occupying ischemic

stroke in Switzerland by applying currently available evidence, in order to decrease case fatality and morbidity of this disease.

The specific objectives of these recommendations are (a) to avoid performing craniectomy in patients with poor prognosis despite this intervention, (b) to avoid performing craniectomy if the risk of worsening and herniation is small, (c) to decide on craniectomy at an early stage, (d) to improve communication between doctors, patients and relatives, and (e) to use adequate neurosurgical techniques and perioperative management.

State of knowledge regarding space-occupying strokes in the middle cerebral artery (MCA) territory

Space-occupying ischemic strokes in the MCA territory, also referred to as 'malignant MCA infarction,' have a high case fatality (1). The principal cause of death in these patients is focal brain oedema, occurring most frequently within 2–5 days after stroke onset, and leading to cerebral herniation and brainstem compression and brainstem herniation. Results of several observational studies show a beneficial effect of craniectomy in these circumstances, especially in younger patients and in case of intervention at an early stage (2–5). One retrospective Swiss study showed less favorable results than expected (6). Recent controlled-randomized trials (7–9) and their combined analysis have shown a highly significant reduction of case fatality with a number needed to treat (NNT) of two. A trend to a less important handicap in patients undergoing craniectomy was also reported (7). The results of these studies can be applied in clinical practice (10, 11) as part of the specialized stroke care in stroke centers (12).

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These recommendations were approved by the Swiss Neurological Society, the Swiss Society of Neurosurgery and the Swiss Society of Intensive Care Medicine.

State of knowledge regarding space-occupying cerebellar strokes

Space-occupying ischemic strokes in the posterior fossa may lead to worsening of handicap and death from transtentorial (downward) herniation. Neurological worsening may also arise from acute occlusive hydrocephalus following compression of the fourth ventricle or from brainstem compression with transtentorial (upward) herniation. Similar to MCA territory stroke, case series suggest a benefit of decompressive craniectomy in these patients (13). To date, there are no randomized-controlled trials investigating decompressive craniectomy in space-occupying cerebellar infarction. Current recommendations emerge therefore from a consensus, which is based on pathophysiological considerations, analogies to MCA infarction, and clinical experience.

Methodology

The workgroup 'Acute Stroke Management' of the 'Cerebrovascular Working Group of Switzerland' (ZAS) consisting of neurologists of Swiss stroke units and neurosurgeons, has drafted the present recommendations. The latter were presented to the aforementioned societies for comment and approval and adapted by the 'Guidelines Commission' of the Swiss Society of Intensive Care Medicine. For the present recommendations, published studies (randomized-controlled trials, meta-analyses, and case series) as well as recommendations emerging from current literature (10, 12, 14) were taken into account and rated according to established criteria (Table 1) (15). For recommendations reached on a consensus basis despite insufficient scientific evidence, the rating 'good clinical practice' (GCP) was given.

Neurological manifestations and risk factors

Neurological manifestations of space-occupying MCA infarction

Most patients with MCA infarction present severe neurological deficits from the beginning. They include contralateral hemiplegia, contralateral sensory hemisindrome, conjugate eye deviation, nausea, vomiting, homonymous hemianopia as well as hemineglect or aphasia (16). Within 12–72 h after symptom onset, progressive headaches, impairment of consciousness, ipsilateral pyramidal signs, unilateral or bilateral abducens nerve palsy, and pupillary dilation may occur. The risk factors for developing a space-occupying brain oedema after MCA infarction are summarized in Table 2.

Neurological manifestations of space-occupying cerebellar infarction

In patients with space-occupying cerebellar stroke, the following signs and symptoms are frequent: progressive impairment of consciousness, diplopia, other cranial nerve palsies, pro-

Table 1 European Federation of Neurological Societies Guidelines for therapeutic interventions (15)

Evidence classification scheme for a therapeutic intervention

Class I: An adequately powered prospective, randomized, controlled clinical trial with masked outcome assessment in a representative population or an adequately powered systematic review of prospective randomized controlled clinical trials with masked outcome assessment in representative populations. The following are required:

(a) Randomization concealment

(b) Primary outcome(s) is/are clearly defined

(c) Exclusion/inclusion criteria are clearly defined

(d) Adequate accounting for dropouts and crossovers with numbers sufficiently low to have minimal potential for bias

(e) Relevant baseline characteristics are presented and substantially equivalent among treatment groups or there is appropriate statistical adjustment for differences

Class II: Prospective matched-group cohort study in a representative population with masked outcome assessment that meets a–e above or a randomized, controlled trial in a representative population that lacks one criteria a–e

Class III: All other controlled trials (including well-defined natural history controls or patients serving as own controls) in a representative population, where outcome assessment is independent of patient treatment

Class IV: Evidence from uncontrolled studies, case series, case reports, or expert opinion

Rating of recommendations for a therapeutic intervention

Level A rating (established as effective, ineffective, or harmful) requires at least one convincing class I study or at least two consistent, convincing class II studies

Level B rating (probably effective, ineffective, or harmful) requires at least one convincing class II study or overwhelming class III evidence

Level C (possibly effective, ineffective, or harmful) rating requires at least two convincing class III studies

gressive ataxia, pyramidal signs, nausea, vomiting, and headaches (17). Signs of impending brainstem compression include hypertension, bradycardia, progressive reduction of consciousness. These life-threatening signs only appear late before herniation and occasionally lead to a sudden respiratory arrest. The risk factors for developing a space-occupying brain oedema after cerebellar infarction are summarized in Table 3.

Prognostic factors after craniectomy for malignant MCA infarction

The main prognostic factors after craniectomy for malignant MCA infarction are summarized in Table 4. The side of the

Table 2 Predictors of space-occupying brain edema in MCA infarction

Clinical predictors	Radiological predictors
NIHSS > 20 (left MCA) (27)	Hypodensity > 50% of the MCA territory on initial CT (28)
NIHSS > 15 (right MCA) (27)	Involvement of additional vascular territories (28)
Nausea/vomiting (13)	Early and large abnormalities on diffusion-weighted MRI imaging (29, 30)

CT, computer tomography; MCA, middle cerebral artery; MRI, magnetic resonance imaging; NIHSS, National Institute of Health Stroke Scale.

lesion does not represent an independent prognostic factor for the resulting functional handicap (2, 3, 7). Nevertheless, the possibility of persistent speech disorders in infarctions of the dominant hemisphere should be assessed during the preoperative discussion with the patient and his relatives.

A delayed intervention > 24 h after symptom onset was also a possible predictor indicating unfavorable outcome in a study of Schwab et al. (5), which, however, could not be confirmed by meta-analysis (2). The time of intervention might not be an independent predictor if other variables are taken into account such as a Glasgow Coma Scale (GCS) score below 8 or a pupillary dilation. Parallel to these findings, the degree of midline shift is closely related to an impairment of consciousness and thus, it does probably not represent an independent predictor of outcome either.

Regarding cerebellar infarctions, the only known predictor of unfavorable outcome is advanced brainstem dysfunction (such as locked-in syndrome) (18).

General recommendations

Measures on admission

All patients presenting at least one risk factor for developing a space-occupying brain oedema after hemispheric or cerebellar infarction (Tables 2 and 3) should be evaluated immediately in a stroke unit both by a neurologist and a neurosurgeon (19). An intensive care specialist could also be involved in the risk assessment of conservative vs. surgical management. A precise management plan should be defined as soon as possible, i.e. soon after admission, as a delayed intervention may cause additional and irreversible harm for the brain.

The aforementioned NNT of two can be referred to when discussing with the patient and/or his relatives. The patient's will should be documented and taken into account whenever possible as a considerable long-term handicap is frequent despite craniectomy. In most situations, the patient's will has to be determined with the aid of his relatives. If the patient's will cannot be determined because of impaired consciousness, aphasia, lack of relatives or a health care representative, the attending physicians should act in their patient's best interest

Table 3 Predictors of space-occupying brain edema in cerebellar infarction

Clinical predictors	Radiological predictors
Impairment of consciousness (13)	Hypodensity > 2/3 of the cerebellar hemisphere (31)
	Compression/displacement of the 4th ventricle (32)
	Obstructive hydrocephalus (32)
	Displacement of the brainstem (32)
	Compression of the basal cisternae (32)
	Hemorrhagic transformation of the cerebellar infarction (32)

Table 4 Prognostic factors indicating unfavourable outcome after craniectomy for malignant MCA infarction

Established unfavourable prognostic factors	Possible unfavourable prognostic factors
Age > 50 years (2, 3)	Involvement of additional vascular territories (3)
	Unilateral pupillary dilation (5)
	Glasgow Coma Scale < 8 (18, 33)

MCA, middle cerebral artery.

according to Swiss law. Resuscitation orders (DNR = 'Do not resuscitate') should in general be adapted to the decision whether the patient is a candidate for craniectomy or not.

General measures in management of patients at high risk for a space-occupying infarction (12, 20)

- Close neurological and cardiovascular monitoring in an intermediate or intensive care stroke unit (Class I, Level A), in patients with territorial cerebellar infarctions for up to 5 days, even if the patient seems to be stable (18)
- Ensuring sufficient cerebral oxygenation (Class IV, GCP) for cerebral infarction in general
- Prophylaxis and treatment of hyperthermia (Class III, Level C)
- Correction of hypovolemia with isotonic fluids (Class IV, GCP)
- Avoid oral intake of food and fluids (Class IV, GCP)
- Elevation of the upper part of the body between 0 and 30° during periods of increased intracranial pressure (ICP) (Class IV, GCP)
- Treatment of hyperglycemia > 8 mmol/l (Class IV, GCP)
- No antiplatelet agents if a craniectomy is likely to be carried out (Class IV, GCP). If there is a low probability of performing a craniectomy or if such an intervention has been ruled out, the administration of aspirin (100–300 mg orally or intravenously) is recommended. In case of known aspirin intolerance, Clopidogrel 75–300 mg can be admi-

nistered orally. The intake of antiplatelet agents does not constitute an absolute contraindication for craniectomy.

- Treatment of headaches (paracetamol, optionally additional metamizol; nonsteroidal anti-inflammatory drugs and opioids are to be avoided) (Class IV, GCP)
- Treatment of nausea and vomiting (Class IV, GCP)
- Thromboembolic prophylaxis with subcutaneous low-dose heparin, low molecular weight heparin or heparinoids (Class I, Level A); in addition, consider intermittent pneumatic compression and elastic stocking of the lower limbs (Class IV, GCP)
- Therapeutic anticoagulation with full-dose unfractionated heparin, low molecular weight heparin or heparinoids is not recommended during the acute phase of cerebral infarction (Class I, Level A)
- Consider intraarterial blood pressure (BP) monitoring (Class IV, GCP) in case of:
 - BP exceeding the upper limits and not controllable by medication
 - Imminent cerebral hypoperfusion [cerebral perfusion pressure (CPP) < 60 mmHg].
 - BP control (Class IV, GCP):
 - Nonthrombolysed patients: upper limits: systolic BP 220 mmHg, diastolic BP 120 mmHg [mean arterial pressure (MAP) 150 mmHg]
 - Thrombolysed patients: upper limits: systolic BP 185 mmHg, diastolic BP 110 mmHg (MAP 130 mmHg) (21)
 - In case of complications due to hypertension such as myocardial infarction, heart failure, aortic aneurysm, etc., the BP should be reduced slowly and cautiously to the following target values:
 - Systolic BP 140–160 mmHg
 - Diastolic BP 80–90 mmHg
 - MAP 90–120 mmHg.
 - In case of cardiac complications due to hypertension, BP reduction should be achieved by means of a short-acting, intravenously administered β -blocker (e.g. esmolol)
 - An arterial hypotension or a cerebral hypoperfusion (CPP < 60 mmHg) should be avoided in any case.

Nonindicated measures in management of patients at risk for a space-occupying infarction (12, 20)

- Administration of corticosteroids (Class III, Level C)
- Administration of hypotonic fluids (Class IV, GCP)
- Administration of sedatives (except benzodiazepines for alcohol withdrawal) (Class IV, GCP)
- Controlled hyperventilation and/or administration of mannitol or hypertonic saline are of little value if a decision against a craniectomy has already been made, as these measures show only a transitory effect in the majority of cases and are possibly associated with a rebound phenomenon after discontinuation and thus an increase in ICP (Class III, Level C)

- Preoperative invasive ICP monitoring is not routinely recommended (Class IV, GCP). However, increasing ICP values may serve as an additional indicator for surgical intervention. Furthermore, ICP monitoring can be helpful in optimising postoperative management in the intensive care unit (ICU). It is important to note that the interpretation of ICP values alone in massive MCA infarction has to be done with caution, due to the phenomenon of intracranial compartmentalization (22).

Specific recommendations

Indications for decompressive craniectomy

MCA infarction (Class I, Level A)

- Usually, age < 60 years, and
- Impairment of consciousness or progressive reduction of consciousness, and
- Mass effect on brain imaging (oedema exceeding 50% of the MCA territory and midline shift), and
- Exclusion of other causes of impaired consciousness (e.g. hypoperfusion, hypotension, cerebral reinfarction, epileptic seizures).

Cerebellar infarction (Class III, Level C)

- Neurological signs of brain stem compression (e.g. hypertension, bradycardia, progressive reduction of consciousness, etc.), and
 - Mass effect on brain imaging, and
 - Exclusion of other causes of impaired consciousness.
- In these patients, no age limit is suggested, but patients should not have a significant prestroke handicap.

Contraindications for decompressive craniectomy

MCA infarction

- Bilateral, nonreactive, not drug-induced pupillary dilation, associated with coma
- Simultaneous presence of all four of the following unfavorable prognostic factors:
 - Age > 50 years
 - Involvement of additional vascular territories
 - Unilateral pupillary dilation
 - GCS < 8
- Severe comorbidity (severe heart failure or myocardial infarction, incurable neoplasia, etc.)
- Refusal by the patient of this treatment, as known from current interaction with the patient, from existing written documents or related by the patient's proxies.

Cerebellar infarction

- Clinical or radiological signs of severe, irreversible brain stem ischemia

- Severe comorbidity (see 'MCA infarction')
- (Known or presumed) refusal of this intervention by the patient (see 'MCA infarction')

Procedure in case of clinical deterioration

- Urgent computer tomography (CT) or magnetic resonance imaging
- Exclusion of other causes of impaired consciousness
- Proceed to craniectomy as fast as possible
- If the craniectomy is delayed, the patient should be sedated, intubated, and ventilated and transferred to the ICU, and pharmacological measures should be taken (see the following section)

Sedation, intubation and controlled ventilation, and specific pharmacological measures

In case of decompressive craniectomy

The decision to perform decompressive craniectomy should be made early and surgery should be carried out as soon as possible once clinical criteria and radiological are fulfilled. In the meantime, the following conservative measures might be initiated:

- Placement of a central venous catheter and an arterial catheter (if not already done) (Class IV, GCP)
- Profound sedation, analgesia, intubation, and controlled mechanical ventilation with a target PaCO₂ of 35 mmHg (4.6 kPa) (Class IV, GCP)
- Osmotherapy (Class III, Level C): it is important to note, that the efficacy of osmotherapy is discussed controversially, for in theory, it can aggravate midline shift, if the corresponding pharmacological agents reach exclusively regions with intact blood–brain barrier and not ischemic ones. If an osmotherapy is to be administered, the following options are available:
 - Mannitol 20% (23, 24)
 - Hypertonic NaCl–HAES solution (25)
 - Hypertonic NaCl solution (26)
- Determination of serum osmolality at regular intervals with a target value of 300–310 mOsmol/kg (Class IV, GCP).

In case of decision against decompressive craniectomy

Apply general measures (see 'Nonindicated measures in management of patients at risk for a space-occupying infarction'); in general, abstain from sedation, intubation, and controlled ventilation.

CPP

Regardless of surgical treatment, a CPP > 60 mmHg should be maintained, if necessary by means of volume replacement and/or catecholamines (Class IV, GCP).

Coagulation disorders (Class IV, GCP)

- Coagulation disorders should be corrected before craniectomy according to their aetiology, if necessary in collaboration with a hematologist

- A craniectomy after thrombolysis is possible whether it is for an ischemic or a hemorrhagic mass effect. At the time of craniectomy, the coagulation factors should be in the normal range, particularly the fibrinogen
- If the patient received an antiplatelet drug, a preoperative platelet transfusion should be considered.

Surgical technique

MCA infarction

Fronto-parieto-temporo-occipital craniectomy up to the midline with a diameter of at least 12 cm is performed and a durotomy and an enlargement duroplasty are performed. Removing ischemic brain tissue is not recommended. In case of concomitant intracranial bleeding, the hematoma can be evacuated. Intracranial pressure monitor placement is recommended.

Cerebellar infarction

Craniectomy up to the transverse sinus and opening of the foramen magnum is realized. In addition, durotomy, enlargement duroplasty, and removal of ischemic cerebellar tissue should be performed. In case of concomitant hydrocephalus, an external ventricular drainage with ICP monitor placement or a ventriculostomy should be considered. Shunt placement without realizing a craniectomy is not recommended.

Postoperative management at the ICU (Class IV, GCP)

- Apply general intensive care concepts ('sepsis bundles', lung-protective ventilation, strict blood sugar control, treatment of hyperthermia, early enteral alimentation, etc.) and intensive care treatment of acute ischemic stroke in collaboration with a stroke specialist (12, 20)
- ICP and CPP monitoring, treatment of intracranial hypertension, and maintenance of an adequate CPP if necessary, antiepileptic drugs in case of seizures, etc. (see also 'Nonindicated measures in management of patients at risk for a space-occupying infarction')
- Control CT after 24 h or earlier if signs of intracranial hypertension are present and before any attempt at waking from sedation
- Waking the patient from sedation should be attempted as soon as there are no more signs of significant intracranial hypertension.
- Thromboembolic prophylaxis with subcutaneous low-dose heparin, low molecular weight heparin or heparinoids from the second postoperative day, after consulting the responsible neurosurgeon
- Mobilization after successful attempt at waking, after extubation, and in absence of signs of significant intracranial hypertension
- Early rehabilitation should be initiated already in the ICU.

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