Monitoring intracranial pressure in patients with malignant middle cerebral artery infarction: is it useful?

Clinical article

MARIA ANTONIA POCA, M.D., PH.D.,1,2 BESSY BENEJAM, M.S.,2
JUAN SAHUQUILLO, M.D., PH.D.,1,2 MARILYN RIVEIRO, M.D.,3 LAURA FRASCHERI, M.D.,4
MARIA ANGELES MERINO, M.D., PILAR DELGADO, M.D., PH.D.,5
AND JOSÉ ALVAREZ-SABIN, M.D., PH.D.5

1Department of Neurosurgery, 2Neurotraumatology-Neurosurgery Research Unit, 3Neurotraumatology Intensive Care Unit, 4Institute of Diagnostic Imaging, and 5Neurovascular Unit and Neurology Department, Vall d’Hebron University Hospital, Institut Recerca Vall d’Hebron, Autonomous University of Barcelona, Spain

Object. Intracranial pressure (ICP) monitoring is increasingly used in the treatment of patients with malignant middle cerebral artery (MCA) infarction. However, neurological deterioration may exist independent from intracranial hypertension. This study aimed to present the findings of continuous ICP monitoring in a cohort of patients with malignant MCA infarction and to correlate these findings with clinical and radiological features.

Methods. The authors studied a prospective cohort of 25 patients with malignant MCA infarction consecutively admitted to the neurotrauma intensive care unit of the Vall d’Hebron University Hospital between March 2002 and September 2006. The patients were treated using a combined protocol of initial moderate hypothermia and hemi-craniectomy. The latter was performed when patients showed a midline shift (MLS) ≥ 5 mm or ICP > 20 mm Hg. Six patients had an MLS ≥ 5 mm on the first CT scan and underwent surgery without prior ICP monitoring. This study focuses on the subgroup of 19 patients who underwent intraparenchymatous ICP monitoring before surgery.

Results. Intracranial pressure readings were evaluated and correlated with pupillary abnormalities, MLS, and ischemic tissue volume. In 12 of the 19 patients, ICP values were always ≤ 20 mm Hg, despite a mean (± SD) MLS of 6.7 ± 2 mm and a mean ischemic tissue volume of 241.3 ± 83 cm³. In 2 patients with anisocoria, ICP values were also normal.

Conclusions. In patients with a malignant MCA infarction, pupillary abnormalities and severe brainstem compression may be present despite normal ICP values. Therefore, continuous ICP monitoring cannot substitute for close clinical and radiological follow-up in the management of these patients. (DOI: 10.3171/2009.7.JNS081677)

Key words • intracranial pressure monitoring • middle cerebral artery • infarction • midline shift • pupillary abnormality • intracranial hypertension

Malignant MCA infarction is a devastating disease associated with a high mortality rate (70–80%) and severe disability in survivors when standard medical management is used. In addition to the extensive amount of necrotic brain tissue involved in malignant MCA infarction, poor neurological outcome occurs because of severe postischemic edema, leading to cerebral herniations, progressive brainstem dysfunction, and intracranial hypertension. Recently, the use of aggressive treatments such as decompressive hemicraniectomy alone or combined with moderate hypothermia has been shown to reduce mortality rates and improve clinical outcomes in survivors. These findings suggest that hemicraniectomy or moderate hypothermia should be considered before neurological deterioration occurs. Therefore, when early predictors of malignant MCA infarction are detected clinically using CT scans or MR imaging, treatment should be started.

Continuous ICP monitoring is increasingly used in the care of patients with large cerebral ischemic infarctions admitted to the ICU. However, some stud-
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...ies have suggested that these patients can show neurological worsening or even die despite normal ICP values, casting doubt on whether ICP monitoring is an adequate tool for monitoring and decision-making in the care of patients with malignant MCA infarction. However, these studies used a variety of probes in different locations, which could have influenced the results. The aim of this study was to analyze the findings of continuous ICP monitoring in patients with malignant MCA infarction, using parenchymal probes always implanted in the ischemic hemisphere, and to correlate ICP readings and their temporal evolution with clinical (pupillary abnormalities) and neuroradiological findings (MLS and ischemic tissue volume) in sequential CT scans.

Methods

Study Population

Between March 2002 and September 2006, 25 patients with a malignant MCA infarction were treated at the NICU of the Vall d’Hebron University Hospital. Patients at risk for developing a malignant MCA infarction were selected on the basis of clinical and neuroimaging criteria. In all patients the stroke involved territories of the internal carotid artery (total anterior circulation syndrome) with persistent proximal MCA or intracranial internal carotid artery occlusion, while neuroimaging (brain CT or multiparametric MR imaging) showed an infarction affecting > 50% of the MCA territory. Study exclusion criteria were: 1) patient or family refusal to be included in the protocol; 2) the presence of previous functional disability assessed by the modified Rankin Scale score; and 3) the presence of terminal disease and/or 4) severe heart failure (New York Heart Association Class III or IV).

After admission, a hemicraniectomy (frontotemporal craniectomy with duraplasty) was immediately performed in 6 of the 25 patients with an MLS ≥ 5 mm demonstrated on the first neuroimaging scans. In 1 patient, although the initial MLS was 12 mm, a hemicraniectomy was delayed for 10 hours because the duty neurosurgeon considered the patient unsuitable for surgery. This patient was admitted to the NICU and was initially treated with medical treatment guided by continuous ICP monitoring. The remaining 18 patients, who showed hemodynamic and respiratory stability, no pupillary abnormalities, and MLS < 5 mm, were admitted to the NICU, intubated, and sedated using midazolam and fentanyl. In these 18 patients, continuous ICP monitoring was immediately begun. Moderate hypothermia (32–33°C) was induced in 17 patients through an endovascular cooling method (Coolgard 3000 system, Alsius Corporation). Once admitted to the NICU, clinical and radiological follow-up were performed and the decision to perform hemicraniectomy was made when, despite medical treatment, the patients showed ICP > 20 mm Hg and/or MLS increases ≥ 5 mm. This management protocol is summarized in Fig. 1 and was approved by the Ethics Committee of the Vall d’Hebron University Hospital (CoolStroke protocol).

![Diagram of Coolstroke Trial](image-url)

**Fig. 1.** Summary of the inclusion criteria for induction of moderate hypothermia or hemicraniectomy (Coolstroke Trial). According to this protocol, when a hemicraniectomy is performed and ICP is < 20 mm Hg, very slow and controlled rewarming is begun at a rate of 1°C/day, depending on the ICP response to small temperature increments. NeuroICU = NICU.
no. PR-HG-128/2004). Written informed consent was obtained from each patient, or next of kin when the patient was unable to provide informed consent.

The present study focuses on ICP values and their temporal profile in the subgroup of 19 patients who did not undergo immediate surgery, were admitted to the NICU, and underwent early ICP monitoring after stroke. All ICP values analyzed in this study were limited to the presurgical period.

Intracranial Pressure Monitoring Protocol

Continuous ICP monitoring was performed in all 19 patients using an intraparenchymatous ICP sensor (Camino Model 110-4B, Integra Neurosciences). To avoid interhemispheric gradients, the ICP sensor was implanted in the ischemic hemisphere in all patients (precoronal region at 11 cm from the nasion and 3 cm from the midline; Fig. 2). Intracranial pressure readings taken at the end of each hour were recorded manually by nurses every hour. This information was complemented by analysis of ICP patterns that we designated for this study.

Quantifying Treatment Intensity Using the E-TILS

The general care of patients in this study included continuous sedation and analgesia, induced and maintained by continuous infusion of midazolam and morphine. Further treatment to control high ICP included inducing muscular paralysis using vecuronium (also routinely used in patients undergoing hypothermia), using moderate hyperventilation, and administering intermittent boluses of mannitol and/or 7.2% hypertonic saline. Inducing hypothermia (32–33°C) was used as a neuroprotective maneuver. Decompressive craniectomy was performed when MLS was ≥ 5 mm or ICP > 20 mm Hg. Barbiturates were used only if all previous therapeutic maneuvers failed to control high ICP.

To determine the different levels of treatment needed in patients with similar ICP profiles, we used a modification of the 15-point TILS proposed by Maset et al.22 The modified version (E-TILS) includes the following changes: 1) a score of 0 points is given when PaCO₂ is ≥ 45 mm Hg, or when muscular paralysis, CSF drainage, and hyperosmotic solutions are not used; 2) both hypertonic saline or mannitol are considered within hyperosmotic therapy; 3) the possibility of draining CSF from a lumbar or a ventricular drain is included; and 4) 15 points are added for moderate hypothermia (32–33°C) or high-dose barbiturates used to treat intracranial hypertension, and 20 points are added for decompressive craniectomy.

![Figure 2](image-url)

**FIG. 2.** Case 11. Computed tomography scans illustrating the position of the ICP sensor before (A–D) and after (E–H) surgery. Before surgery, the ICP sensor was implanted in the ischemic hemisphere, within the ischemic tissue. After hemicraniectomy, the ICP sensor was implanted in the contralateral hemisphere covered by the skull. Before surgery, despite normal ICP values, MLS was 8 mm and there was subfascial and uncal herniation and some cisternal effacement.
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After these modifications, the minimum possible score is 1 (sedation and analgesia only) and the maximum score is 62. Table 1 shows the original TILS and the E-TILS.

**Pupillary Examination**

Pupillary examination was performed hourly by the NICU nurses, who recorded the size and response of both pupils to light. According to the Brain Trauma Foundation recommendations, pupillary asymmetry (anisocoria) was defined as a difference of ≥ 1 mm between both pupils, and pupillary dilation was defined as a pupil size > 4 mm.

**Midline Shift and Ischemic Brain Tissue Volume Quantification**

The extension of ischemic brain lesions was confirmed using the sequential CT scans. Computed tomography scans were performed at admission and at least once daily before decompressive craniectomy. Additional CT scans were performed if patients showed signs of cerebral herniation (anisocoria) or ICP increases not controlled by medical treatment. At our center, the CT scan unit is close to the NICU, facilitating radiological monitoring. All CT scans were conducted using a Phillips MX-8000 machine, with a 3-mm slice thickness and a 512 × 512 matrix. For infarct volume quantification and MLS calculations, images were acquired and stored in Digital Imaging and Communications in Medicine (DICOM) format. Midline shift was determined by halving the distance between the inner tables of the skull, and measuring the absolute distance (in millimeters) of the septum pellucidum from this line. The total presurgical volume (in cubic centimeters) of the hypodense brain (which, if > 1 vascular territory was involved, included the sum of all the territories) on the CT scan was measured using a semiautomated method, based on the “seeded region growing algorithm” (Fig. 3).

**Statistical Analysis**

Normal or nonnormal distribution of quantitative variables was determined by the Kolmogorov-Smirnov test. Normally distributed variables were described using the mean and SD. The median and IQR were used to describe variables that followed a non-Gaussian distribution. The Mann-Whitney rank sum test was used to detect statistically significant differences in age, MLS, and ischemic tissue volume between patients with normal and increased ICP. Differences were considered statistically significant when the p value was ≤ 0.05.

**Results**

Of the initial series of 25 patients, 6 were not included in this study because they had an MLS ≥ 5 mm at admission and underwent surgery before transfer to the NICU and before ICP monitoring was begun. The present study focuses on the remaining 19 patients (4 women and 15 men, mean age 52.9 ± 9.7 years, range 32–68 years). At admission, the median GCS score in these 19 patients was 14 (IQR 13–15). The initial NIH Stroke Scale scores ranged from 16 to 23 points (mean 19.2 ± 2.4 points). Hemicraniectomy was also performed in 18 of the 19 patients. After surgery, the patients were slowly rewarmed at a rate of 1°C/day. The remaining patient died due to a cardiac rupture. Tables 2 and 3 list the clinical details of individual patients.

**Location and Size of Infarction**

The right hemisphere was affected in 12 patients (63.2%) and the left hemisphere in the remaining 7 patients. Radiologically, MCA infarction was incomplete in 3 patients and complete in 16. Concomitant ischemic lesions were observed in the territory of the anterior cerebral artery in 7 patients and 1 patient had ischemic lesions in both hemispheres. The mean ischemic tissue volume was 256.8 ± 107 cm³ (range 98–460 cm³).

**Intracranial Pressure Findings**

The mean time from beginning ICP monitoring to surgery was 43 ± 34.4 hours (range 9–145 hours). In 12
(63%) of the 19 patients, ICP values were always ≤ 20 mm Hg (ICP pattern 1), even though all these patients showed an MLS ≥ 5 mm (mean MLS 6.7 ± 2 mm), uncal herniation, or cistern effacement on the CT scan (mean ischemic tissue volume 241.3 ± 83 cm³; Fig. 2). The remaining 7 patients (37%) had short-lived (ICP pattern 2) or persisting increases in ICP values (ICP patterns 4 and 6) during the first hours after stroke onset (Table 3).

In the 7 patients who showed intracranial hypertension, mean ICP ranged from 20 to 30 mm Hg in 5 patients and was > 30 mm Hg in the remaining 2 patients. Fig. 4 summarizes hourly ICP readings in the entire patient series. The median therapy intensity level to maintain these ICP values was 18 points on the E-TILS (IQR 18–20.5), showing that these hypothermic patients only needed sedation-analgesia, muscular paralysis, moderate hyperventilation, and sporadic use of hypertonic solutions.

No statistically significant differences were found between patients with normal and increased ICP for the following variables: total ischemic cerebral volume (t = 81, p = 0.375), MLS (t = 82.5, p = 0.310), or age (t = 53, p = 0.163).

**Pupillary Reaction**

Two of the patients without increased ICP (≤ 20 mm Hg) and 2 of the 7 patients who showed intracranial hypertension before surgery showed anisocoria. Normal pupillary reactivity was maintained in 14 of the 19 patients throughout the study period.

**Discussion**

The results of our study demonstrate that patients with a malignant MCA infarction may show ICP values < 20 mm Hg despite a large volume of infarcted tissue.
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Intracranial Pressure Monitoring in Patients With Malignant MCA Infarction

Because of the poor prognosis of patients with malignant MCA infarction, ICP monitoring has been increasingly used in units that advocate more aggressive therapies such as moderate hypothermia or decompressive hemicraniectomy. Several studies have demonstrated that these new therapeutic measures are only of clear benefit when applied early, requiring ICU admission, elective intubation and continuous monitoring of ICP, and monitoring systemic variables.

The aims of ICP monitoring in patients with malignant MCA infarction are to guide therapeutic decision-making, provide evidence of the effectiveness of the therapeutic maneuvers applied, and detect unexpected complications such as hemorrhagic transformation of the infarcted brain. However, in our study we found that a considerable number of patients (63%) had normal ICP values despite marked MLS and a fairly large volume of ischemic brain (between 124 and 352 cm³). Furthermore, 2 of the 12 patients had anisocoria while ICP was under the accepted threshold of 20 mm Hg.

Elevated ICP has previously been found to be an uncommon cause of neurological deterioration in patients with large cerebral infarctions and edema. Our findings suggest that severe brain herniation and brainstem compression can be found despite normal ICP values. These findings do not imply that ICP monitoring is of no value in these patients because hemorrhagic transformation (Fig. 5), a sudden increase in brain shift, or new lesions may be detected on the basis of an increase in ICP values. Additional monitoring methods might be required in patients with normal ICP values to avoid sudden neurological deterioration or even brain death. In our opinion, sequential CT scans or monitoring of MLS by noninvasive methods such as duplex sonography are the most useful tools in the follow-up evaluation of these patients.

Where and How to Monitor ICP in Malignant MCA Infarction

Previous studies have shown that ICP may be normal in some patients despite marked MLS or large cerebral ischemic tissue volume. However, in these studies the methodology used varied widely: different types of ICP sensor were used (epidural, subarachnoid, intraventricular, and parenchymal); probes were implanted in distinct sites (ischemic or normal hemisphere); and sometimes

### TABLE 2: Clinical data of individual patients

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<th>Age (yrs), Sex</th>
<th>GCS Score</th>
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* NIHSS = NIH Stroke Scale; NP = not performed.
† Time from stroke to the CT scan used to include the patient in the protocol for malignant MCA infarction.
‡ Time from stroke to the time at which the patient reached the target temperature of 32°C.
malignant progression of the cerebral infarction was absent. The present study includes a homogeneous series of patients with malignant MCA infarction in whom the ICP sensor was always implanted in the brain parenchyma.

Another important issue in patients with malignant MCA infarction is the choice of hemisphere for sensor implantation. The presence of a large volume of ischemic tissue in 1 hemisphere with a marked MLS can lead the intracranial compartment to behave as a bicameral space. In this situation, ICP is not uniformly distributed. In 7 patients undergoing bilateral epidural monitoring, Schwab et al. demonstrated that ICP differences between the ischemic and nonischemic hemispheres ranged from 5 to 15 mm Hg. Higher ICP values were always recorded in the ischemic hemisphere, although the gradients were only present for the first 3 days of ICP monitoring. The possibility that these ICP gradients may be transitory could explain the results of Carhuapoma et al., who ruled out the presence of interhemispheric ICP gradients in a patient with large cerebral infarction with bilateral ICP monitoring begun 8 days after neurological deterioration. Altogether, these findings indicate that in patients with malignant MCA infarction, the ICP sensor should always be implanted in the ischemic hemisphere.

**Explaining Normal ICP Values in Patients With Malignant MCA Infarctions**

Several factors could explain the presence of normal ICP in patients with large cerebral infarctions and MLS. One of these factors may be the dramatic reduction of cerebral blood flow and therefore cerebral blood volume in the ischemic hemisphere at the beginning of the process and later in the nonreperfused brain. However, in a second stage, the reduction in blood volume in the affected hemisphere.

**Table 3: Clinical data of individual patients**

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<th>Age (yrs), Sex</th>
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<th>MLS (mm)</th>
<th>Mean ICP† (mm Hg)</th>
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* ITV = ischemic tissue volume; PA = pupillary abnormalities.
† Mean ICP in the immediate 8 hours before hemicraniectomy.
‡ Decision of the neurosurgeon on call.

![ICP vs Time](media/fig4.png)

**Fig. 4.** Box-and-whisker plots showing hourly ICP readings recorded in the complete patient series before surgery at 3- and 12-hourly intervals. The first interval includes ICP readings before induction of moderate hypothermia in some patients. Note that most values were < 20 mm Hg. Large black dots represent outliers.
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hemisphere is overcompensated by an increase in intracellular and extracellular water content (cytotoxic and vasogenic edema).

An additional phenomenon that may have contributed to maintaining normal ICP values in these patients is that brain volumetric compensatory mechanisms are at least partially preserved at the beginning of the patient’s clinical course and, therefore, in this period, the patient is still at the initial stage of the pressure-volume curve, when increases in brain volume are easily compensated. The primary involvement of the temporal lobe in malignant MCA infarction could explain the finding in some patients of pupillary abnormalities before ICP becomes increased (third nerve compression and brainstem distortion can occur with slightly increased or even normal ICP).

In our series, a potential confounding factor was the use of moderate hypothermia. This is arguably one of the most important factors contributing to normal ICP after malignant MCA infarction. Moderate hypothermia is a powerful therapeutic maneuver that, in addition to its potential neuroprotective effects, produces clear reductions in ICP. However, there is no evidence that ICP remains under control in patients treated with hypothermia who have an MLS. Furthermore, we have observed the same phenomenon in normothermic patients. To clarify this issue, further studies comparing normo- and hypothermia are required. Before these data are available, the hypothesis of hypothermia masking an abnormal ICP value, although improbable, cannot be completely ruled out. Because hypothermia is one of the therapeutic alternatives used to manage patients with malignant MCA infarction, this issue merits further consideration.

Vahedi et al. have recently published a pooled analysis of the 3 European randomized, controlled clinical trials of early decompressive surgery in malignant infarction of the MCA (DECIMAL, DESTINY, and HAMLET trials), limited to patients < 60 years old and treated within 48 hours of stroke onset. This meta-analysis concluded that decompressive surgery undertaken within 48 hours of stroke onset reduces mortality and increases the number of patients with a favorable functional outcome. In the studies analyzed, ICP was not a factor in the decision to perform surgery. The results of our study reinforce the message of these studies, that is, that clinical and radiological criteria are sufficient to select patients for decompressive surgery. Because hemicraniectomy was required in all patients despite ICP readings, our initial criteria for surgery (MLS > 5 mm or intracranial hypertension) should clearly be reconsidered.

![Fig. 5. Example of hemorrhagic transformation in a patient with right malignant MCA infarction. An increase in ICP values indicated the need for an additional CT scan, which revealed hemorrhagic transformation of the infarction.](image-url)
Conclusions

Patients with a malignant MCA infarction may show ICP values < 20 mm Hg despite marked MLS (> 5 mm), large brain infarctions (especially if patients are treated with hypothermia), and neurological deterioration (pupillary abnormalities) indicating uncal herniation. Consequently, ICP monitoring cannot substitute for strict clinical and neuroradiological follow-up in these patients.

Disclosure

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Address correspondence to: Maria Antonia Poca, M.D., Ph.D., Department of Neurosurgery, Vall d’Hebron University Hospital, Passeig Vall d’Hebron 119-129, 08035 Barcelona, Spain. email: pocama@neurotrauma.net.