Analysis of the curative effect of a new decompressive Craniectomy on the treatment of severe Craniocerebral injury

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Introduction: At present, severe and extra severe craniocerebral injury are still the difficulties in clinical neurosurgery, with a high mortality rate of 30%-50% and a high disability rate [1]. Complications such as cerebral contusion and laceration, hydrocephalus, and diffuse brain swelling are often developed and then induce rises of malignant intracranial pressure, which is the main cause of mortality [2-4]. For severe and extra severe craniocerebral injury, conservative treatments are often ineffective, while decompressive craniectomy is the key to cure patients. Neurosurgeons hold different views on the removing scope of the bone flap and its merits and demerits [5]. We have designed a new decompressive craniectomy based on years of treatment of severe and extra severe craniocerebral injuries.

Subjects and Methods: The objects of this study are patients with severe and extra severe craniocerebral injury who received decompressive craniectomy operations from December, 2012 to March, 2016. Among them 36 are males and 20 are females, aged from 18 to 70; 30 were injured by road accidents, 10 by falling from high places, 9 by blows and 7 by tumbles. The 56 patients were grouped randomly by flipping coins into a test group of 26 cases to receive the new decompressive craniectomy and a control group of 30 cases to receive the standard decompressive craniectomy. The differences of GCS scores, gender and age between the two groups when they were admitted into the hospital have no statistical significance (P>0.05).

The inclusion criteria of patients were:
1) explicit head injury, no evident trauma history of the chest, abdomen, limbs, etc. that threatened the life of the patients; 2) patients with severe and extra severe craniocerebral injury with a GCS scores of 3 to 8; if a patient is in a deep coma, with bilateral or unilateral mydriasis, he/she is suffering from cerebral hernia; 3) patients who, according to the head CT scan, suffered from multiple intracranial cerebral contusion, intracranial hematoma, epidural hematoma, subdural hematoma, diffuse brain swelling after injuries, evident compression or disappearance of the cerebral cistern such as the ambient cistern and the lateral fissure cistern, evident middle line shift, or evident compression of the lateral ventricles.

The exclusion criteria of patients are:
1) patients with severe and extra severe craniocerebral injury whose bilateral pupils were in continuing dilation; 2) patients with severe and extra severe craniocerebral injury who had respiratory dysfunction and continuing decreasing oxygen saturation; 3) patients with Cushing's reaction.

Surgical procedures

The test group received the new decompressive craniectomy and the control group received the standard decompressive craniectomy.

New decompressive craniectomy

Surgical flaps: Starting from 1 cm ahead of the zygomatic arch upper tragus, extending upward from the back of the auricle to the parietal tuber, passing the middle line of the parietal bone, extending forward to the contralateral forehead within the hairline, then cutting 1-2 cm besides the middle line (Figure 1); the flap incision of bilateral decompressive craniectomy was 2 cm behind the convergence of unilateral flap incisions in the coronal suture (approximately a "W" shaped incision) (Figure 2).

Figure 1. Unilateral decompressive craniectomy. A-B: surgical flap of the scalp; C-D: bone flap; E: CT scan preoperation; F: CT scan after decompressive craniectomy (3 days) G: CT scan after decompressive craniectomy (14 days). Arrows to removed unilaterall bone flap.

Bone flap: A free bone flap was applied; the front of the removing unilateral bone flap was flush with the anterior skull base; the inside needed to reach the middle line as far as possible, the back reaching the parietal tuber and the outer or lower side reaching the middle skull base; the interior sphenoid ridge till the superior orbital fissure was completely removed; the greater wing of the sphenoid bone was partially resected and the squama temporalis completely resected, so that the anterior cranial fossa was flush with the middle cranial fossa, and the anterior and middle skull base would be completely
decompressed (Figure 1); the range of the removing bilateral bone flap is the same with the range of the removing unilateral bone flap on both sides; the size of the intermediate beam bone was about 2-3 cm (Figure 2).

**Figure 2.** Bilateral decompressive craniectomy. A-B: surgical flap of the scalp; C-D: bone flap; E: CT scan preoperation; F: CT scan after decompressive craniectomy (2 days); G: CT scan after decompressive craniectomy (5 days); H: the patient after surgery in 1 month. Arrows to Drainage tube.

Cutting the dura mater: The dura mater was cut from the front temporal lobe in the shape of a claw. The frontal lobe, the temporal lobe, the parietal lobe, the anterior cranial fossa, the middle cranial fossa and the intracranial hematoma could be fully exposed.

All patients were elevated by the head at an angle of 30 degrees. Their tracheas were cut open and the ventilators were used for breathing assistance. Under mild hypothermia and brain protection, nourishing brain cells were given and oxygen free radicals were removed by drugs. Dehydration drugs were applied according to the intracranial pressure of patients indicated by the intracranial pressure monitor; blood sugar level and electrolyte balance were maintained. The control group received the same treatment with the test group.

**Observational indexes**

- ICP shown indirectly by changes of the ambient cistern on the head CT. The intracranial pressure and severity of the illness were known through the morphological changes (normal, compression, abolition) of the ambient cistern on head CT in preoperative and postoperative 3d, 7d [5].

- The incidence of intraoperative and postoperative complications of emergency, such as intraoperative acute cephalocele and postoperative incision hernia.

- GCS scores in preoperative and postoperative 1d, 3d, 7d.

- ICP changes indicated by the intracranial pressure monitor in postoperative 3d, 5d, 7d.

- GOS scores in postoperative 3 months, 6 months and 12 months.

**Statistical analysis**

In this study, the data were statistically analyzed using SPSS17.0 software package. The data of intraoperative cephalocele, postoperative incision hernia and postoperative GOS scores in 3 months, 6 months and 12 months were tested by chi-square ($\chi^2$); GCS scores in preoperative and postoperative 1d, 3d, 7d were represented as mean ± standard deviation ($\bar{x}$ ± s). $P<0.05$ was considered statistically significant.

**Results:** Statistics of ambient cistern changes on preoperative and postoperative head CT of patients receiving new decompressive craniectomy and standard decompressive craniectomy (Figure 3).

**Figure 3.** Preoperative and postoperative changes of CT ambient cisterns. Note: D0, preoperation; D3, postoperative three days; D7, postoperative seven days. Compressions of ambient cistern of both the test group and the control group were lower after the operations. The abolition and normal rate of ambient cistern of the test group was also significantly higher than the control group in postoperative seven days.

2. Comparison of two groups of patients undergoing cephalocele (Figure 4).

**Figure 4.** Incidence of intraoperative cephalocele.*Compared with the control group, Chi-square ($\chi^2$) test $P<0.05$.
3. The comparative analysis of postoperative incision hernia of the two groups (Figure 5).

Figure 5. Incidence of postoperative incisional hernia.*compared with the control group, Chi-square ($\chi^2$) test P<0.05.

4. Comparative statistics of GCS scores in preoperative and postoperative 1d, 3d, 7d of the two groups of patients (Table 1).

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Preoperative</th>
<th>1 d</th>
<th>3 d</th>
<th>7 d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test</td>
<td>30</td>
<td>4.6 ± 1.4</td>
<td>4.7 ± 1.6</td>
<td>5.6 ± 1.4</td>
<td>6.9 ± 1.5</td>
</tr>
<tr>
<td>Control</td>
<td>26</td>
<td>4.4 ± 1.2</td>
<td>5.6 ± 1.2*</td>
<td>7.0 ± 1.1*</td>
<td>8.9 ± 1.3*</td>
</tr>
</tbody>
</table>

*Compared with the control group, Chi-square ($\chi^2$) test P<0.05.

Table 1. Preoperative and postoperative GCS scores of the patient (X± s).

5. Comparative statistics of intracranial pressure (ICP) in postoperative 1d, 3d, 7d of the two groups of patients (Table 2).

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Preoperative</th>
<th>3 d</th>
<th>7 d</th>
<th>7 d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test</td>
<td>30</td>
<td>247.00 ± 41.95</td>
<td>222.67 ± 35.23</td>
<td>192.67 ± 29.35</td>
<td>6.9 ± 1.5</td>
</tr>
<tr>
<td>Control</td>
<td>26</td>
<td>208.08 ± 25.77*</td>
<td>189.62 ± 23.06*</td>
<td>163.85 ± 22.82*</td>
<td></td>
</tr>
</tbody>
</table>

*Compared with the control group, Chi-square ($\chi^2$) test P<0.05.

6. Comparative statistics of GOS scores in postoperative 3 months of the two groups of patients (Table 3, Figure 6).

Figure 6. GOS scores of patients after 6 months *Compared with the control group, Chi-square ($\chi^2$) test P<0.05.

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mild Disability</th>
<th>Moderate Disability</th>
<th>Severe Disability</th>
<th>Vegetative State</th>
<th>Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test</td>
<td>30</td>
<td>3*</td>
<td>2*</td>
<td>6</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td>Control</td>
<td>26</td>
<td>8</td>
<td>10</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>

*Compared with the control group, Chi-square ($\chi^2$) test P<0.05.

Table 3. Number of patients with different GOS scores after 3 months.

Statistical analysis was made according to: eusemia (grade IV, moderate disability; grade V, good recovery (mild disability)), unfavorable prognosis (grade II, vegetative state; grade III, severe disability; grade IV), and grade I death.

7. Comparative statistics of GOS scores in postoperative 6 months of the two groups of patients (Table 4).

Table 4. Number of patient GOS with different scores after 6 months.

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mild Disability</th>
<th>Moderate Disability</th>
<th>Severe Disability</th>
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<th>Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test</td>
<td>30</td>
<td>3*</td>
<td>3*</td>
<td>5</td>
<td>6</td>
<td>13</td>
</tr>
<tr>
<td>Control</td>
<td>26</td>
<td>9</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

*Compared with the control group, Chi-square ($\chi^2$) test P<0.05.

8. Comparative statistics of GOS scores in postoperative 12 months of the two groups of patients (Table 5).

Indexes such as the GCS scores, the intracranial pressure, the middle line shift, etc. have all shown significant improvement after the new decompressive craniectomy in 7 days. The improvement is more obvious than the standard decompressive craniectomy (P<0.05). The incidence of cephalocele and incision hernia in the test group are significantly lower than in the control group (P<0.05). It was discovered in a follow-up visit, 12 months after the operations, that in the test group, there were 9 cases of good recovery (mild disability), 9 medium disability, 2 severe disability, 1 vegetative state and 5 deaths. While in the control group, there were 3 cases of good recovery (mild disability), 4 medium disability, 4 severe disability, 6 vegetative state and 13 deaths; the rate of favorable prognosis of the test group (69.2%, 18/26) is significantly higher than the control group (23.3%, 7/30); the mortality rate of the test group (19.2%, 5/26) is significant lower than the control group (43.3%, 13/30).
Sometimes a "T" shaped incision needs to be added to expand the
within the application of standard decompressive craniectomy.
Temporal contusion, the Labbe vein, the transverse sinus, or the
inadequate in the following aspects: 1) Damage to the combined
In clinical practice, the standard decompressive craniectomy is still
decompressive craniectomy and standard decompressive craniectomy
Comparison of the advantages and disadvantages of the new
hematoma [9-11].
and transverse sinus, causing massive haemorrhage or intracranial
infratentorial hematoma>10 ml; (6) Rupture of superior sagittal sinus
decrease of GCS scores (5) supratentorial hematoma>30 ml or
subdural hematoma combined with brain contusions, significant
cistern, and the disappearance of the sulcus and the gyrus [7,8]; (3)
(2) Diffuse brain injury (cerebral axonal injury, brain swelling), head
indications of decompressive craniectomy for patients with severe and
The necessity of decompressive craniectomy for severe and extra
severe craniocerebral injury
In order to relieve damage to the brain caused by malignant high
intracranial pressure of severe traumatic brain injuries, currently
the main STBI treatment options include conservative treatment,
surgery, and other special treatment (such as mild hypothermia, brain
protection, etc.). Conservative treatment is rather ineffective, among
which decompressive craniectomy is the key to the cure [4]; surgical
indications of decompressive craniectomy for patients with severe and
extra severe craniocerebral injury: (1) Patients with GCS<8, in a coma
or a deep coma, with one side of pupils dilated and cerebral hernia;
(2) Diffuse brain injury (cerebral axonal injury, brain swelling), head
CT showing significant compression or disappearance of the ambient
cistern, and the disappearance of the sulcus and the gyrus [7,8]; (3)
patients with bilateral hematoma, middle line shift>5 mm; patients
with unilateral hematoma, middle line shift>10 mm, significant
compression or occlusion of ipsilateral or bilateral paraceles; (4)
subdural hematoma combined with brain contusions, significant
decrease of GCS scores (5) supratentorial hematoma>30 ml or
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and transverse sinus, causing massive haemorrhage or intracranial
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Comparison of the advantages and disadvantages of the new
decompressive craniectomy and standard decompressive craniectomy
In clinical practice, the standard decompressive craniectomy is still
inadequate in the following aspects: 1) Damage to the combined
temporal contusion, the Labbe vein, the transverse sinus, or the
superior sagittal sinus, which are often found rather difficult to deal
with in the application of standard decompressive craniectomy.
Sometimes a "T" shaped incision needs to be added to expand the
bone window backward. This not only increases operation time,
the intracranial pressure not having been mitigated timely, but also
veins on the surface and depth of the brain such as Labbe vein,
the sagittal sinus, transverse sinus and other veins remain under
pressure, affecting cerebral venous reflux, leading to sustained brain
ischemia, hypoxia, a corresponding change in the pathophysiology of
intracranial hypertension, and an increasingly malignant high cranial
pressure [12]; 2) Since the brain tissue is in a swelling state after the
brain injury, and the size of the bone window is definite when applying
standard decompression craniectomy, the thin layer of potential
subdural hematoma in the skull base cistern and other places cannot
be cleared timely, causing cerebrospinal fluid circulation disorder,
increasing the incidence of hydrocephalus, and worsening the
patient's condition and prognosis; 3) Since the bone window is small
in the operation of standard decompressive craniectomy, the temporal
lobe, the frontal lobe, and the parietal lobe are not decompressed
adequately, leading to cerebral ischemia and hypoxia, disorder of
cerebrospinal fluid circulation, obvious secondary swelling of brain
tissue after the operation, and incision hernia, further exacerbating
the circulation disorder of cerebrospinal fluid and blood, softening the
brain tissue in the decompressed area and causing vascular necrosis,
increasing intracranial pressure, the rate of cerebral infarction and
vascular occlusion, later causing cerebromalacia and cerebral atrophy
of surviving patients, and increasing the incidence of epilepsy, and
worsening prognostic [13]; 4) After decompressive craniectomy for
severe brain injury, changes of electrolytes and osmotic pressure in
extracellular fluid and blood cause intracerebral edema, also known
as osmotic pressure brain edema. Since brain swelling is still evident
after standard decompressive craniectomy, the dose of dehydration
drugs such as mannitol increases significantly, which is reported
to cause up to 20% impairment of renal function. Large doses of
mannitol may lead to heart and kidney damage, increase the incidence
of complications such as water, electrolyte imbalance, and also lead
to hyponatremia and hypokalemia, which at the same time is also a
major cause of secondary intracranial cerebral edema.
Considering the disadvantages of standard decompressive
craniectomy, we designed this new decompressive craniectomy to
make up for the disadvantages of standard decompressive craniectomy
to some extent. Its advantages include: 1) Full exposure of the lobe,
the temporal lobe, and the parietal lobe, making the decompression
more complete; removing about 95% of the unilateral supratentorial
acute intracranial hematoma, and removing more effectively the
hematoma in the anterior skull base and the middle concave bottom,
which is conducive to the self-healing of hernia. Full exposure of
the skull base, and the anterior longitudinal can clear skull base
cistern hemorrhage more precisely, which is conducive to smooth
cerebrospinal fluid circulation [14]; 2) Full exposure of the veins on the
surface and in the depth of the brain such as veins of the Labbe and
the lateral fissure veins. Meanwhile the lateral fissure cistern and the
skull base cistern are fully opened in surgery. Removing the inferior vena
hemorrhage, releasing the bloody cerebrospinal fluid, and relieving
vasospasm will smoothly venous return, relieve cerebral edema and
infarction, while reducing the incidence of arachnoid adhesions and

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<td>30</td>
<td>2*</td>
<td>3*</td>
<td>5</td>
<td>6</td>
<td>13</td>
</tr>
<tr>
<td>Control 26</td>
<td>9</td>
<td>9</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 5. Number of patients with different GOS scores after 12 months

Discussion: Severe and extra severe craniocerebral injuries are serious
and complicated illnesses with rapid progression, poor prognosis
and high disability and mortality rate; post-traumatic cephaloedema
and malignant high intracranial pressure are the main influencing
factors of prognosis, while cerebral ischemia, hypoxia are the main
reasons for secondary brain injury, interacting with other influencing
factors of secondary brain injury (eg: BBB damage, disorder of
cerebrospinal fluid circulation, etc.), further exacerbating brain injury
and cephaloedema, and affecting the prognosis [6]. For patients with
severe and extra severe craniocerebral injury, conservative treatment
is ineffective. Decompressive craniectomy is the key to the treatment
of patients.

In order to relieve damage to the brain caused by malignant high
intracranial pressure of severe traumatic brain injuries, currently
the main STBI treatment options include conservative treatment,
surgery, and other special treatment (such as mild hypothermia, brain
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Disadvantages of the new decompressive craniectomy

The incidence of poor healing of scalp incisions and postoperative intracranial infection of the new decompressive craniectomy are higher than the standard decompressive craniectomy. In the test group, 3 patients had poor scalp healing, and 5 patients had intracranial infection.

By our analysis the possible causes of poor scalp healing are:

1) surgical incision: the scalp is rich in blood supply, mainly from the orbitofrontal artery, the superficial temporal artery and various branches, yet due to the large surgical incision on the scalp, damage to the branches of the superficial temporal artery is inevitable, causing poor or prolonged wound healing on the scalp; 2) Patients of severe brain injury need comprehensive treatment combined with mild hypothermia, during the process of which the head wears an ice cap for cooling. The low temperature reduces the blood circulation of the scalp, increasing the incidence of poor wound healing [13].

By our analysis the causes of increased postoperative infection may be that: severe brain injuries are often accompanied by varying degrees of coma and vomiting, affecting the normal physiological function of patients, weakening immunity, and increasing infection rates. The liquefaction and necrosis of edematous brain tissues offer a very good medium for bacteria. Infections are easy to occur due to intracranial and extracranial communication in open craniocerebral injuries. Related research has found that the number of operations, the operative time, and whether hypoproteinemia and cerebrospinal fluid leakage have occurred are high risk factors of intracranial infection [19,20]. The high risk of intracranial infection in the new decompressive craniectomy is due to: 1) big surgical wound, poor local blood supply, and the incision which is close to the bacteria area through the parietal tuber; 2) long surgical incisions, large amounts of incision sutures, the large area of artificial dura mater required in intraoperative dura mater reparation, as well as the increases of the number and area of foreign matter, increasing the rate of infection; 3) possible open scalp lacerations and open fractures in surgical areas, which needs debridement first, and repeated rinsing by disinfectants such as saline, iodine and 2% of hydrogen peroxide, leading to wet draping, and increasing the chances of infection. In order to reduce postoperative scalp necrosis and the incidence of intracranial infection, surgical incisions and the drainage tube apparatus should be observed closely after the surgery. Dressings and drainage bags should be replaced regularly. The ward should be ventilated and disinfected on time to maintain environmental hygiene. And medical care should be strengthened [2,21].

Conclusion: The new decompressive craniectomy has improved the prognosis, laid a good foundation for comprehensive postoperative treatment, and reduced disability and mortality rates in the treatment of patients with severe and extra severe craniocerebral injury. It is rather significant in clinical promotion and practice.

References


