

# Cortical Pressure Injury: A Hypothesis to Explain the Incongruity of Clinical and Radiologic Improvement in Decompressive Craniectomy

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## Abstract

### Keywords

- ▶ decompressive craniectomy
- ▶ cortical pressure injury
- ▶ hypothesis

It has astonished neuroscientists since the advent of decompressive craniectomy as to why a seemingly successfully achieved goal of reduction in intracranial pressure (ICP), by removing a portion of the cranial vault and the resultant intracranial volume augmentation, fails to give the desired beneficial clinical outcome in every case and in fact, at times, proves to be deleterious in some conditions with a shared problem of refractory raised ICP. The authors propose a hypothesis based on the understanding of the anatomy and physiology of the brain that can explain the fallacy.

## Introduction

Decompressive hemicraniectomy is often resorted to deal with, and sometimes in anticipation of, a refractory raised intracranial pressure (ICP) produced by a wide range of pathologies causing cytotoxic and interstitial edema in the brain.<sup>1–4</sup> It is well documented that opening of the box causes a fall in the ICP or at least renders it more amenable to medical measures.<sup>5,6</sup> However, the clinical improvement does not seem to be proportionately reflected in every case, and often it is the primary pathology *responsible for the rise in ICP*, which determines this variability. Explanations available so far, proven or presumed, are either global in nature, for example, failure of cerebral vascular autoregulation or alteration of cerebrospinal fluid (CSF) dynamics, or are equally applicable to cases of any etiology who have undergone a decompressive craniectomy (DC) to tackle a refractory raised ICP. They fail to explain why DC routinely gives better result with malignant middle cerebral artery (MCA) infarct,<sup>1</sup> as compared with the cases of traumatic brain injury (TBI)<sup>7</sup> and the extent of brain damage due to the primary insult is the only reasoning we are often left with.

Why has the DC been unable to universally give the benefit it promises? Is there something very basic we are not doing correctly? In this article, we have made an attempt to

formulate a hypothesis on the basis of the existing understanding of the anatomy and physiology of the brain and known physical and hemodynamic changes in DC, documented in clinical and experimental studies to explain these fallacies from a viewpoint unexplored so far.

## Hypothesis

Decompressive craniectomy, on one hand, reduces ICP by providing the swollen brain additional space to expand and thereby corrects the hemodynamics in the white matter and causes resolution of brain shift, whereas on the other hand, it jeopardizes the blood supply of the cerebral cortex directly underlying the craniectomy defect, by compressing the pial vessels and cortical microvasculature at the brain scalp interface—a phenomenon similar to that of the pressure sore formation in soft tissues.

## Rationale

### Cranial Cavity and Brain

Brain is housed in the cranium protected from the physical forces of the world by the calvaria. The cerebral vessels bathe in CSF in the subarachnoid space on the brain surface and Virchow Robin spaces in the brain parenchyma. In the face of

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increasing brain volume secondary to any pathology, when no more compensation is possible by shifting CSF or blood, compliance of the brain reduces drastically and there is an exponential rise in ICP. DC is the solution available to open the box.

### Peculiarities of Cerebral Microvasculature That Make Cerebral Cortex Especially Vulnerable to Mechanical Forces

At any given point in time, all the cerebral capillaries are perfused with blood,<sup>8</sup> and practically every neuron in the brain has its own capillary.<sup>9</sup> Structurally, compared with the systemic arteries, cerebral arteries have no external elastic lamina and have a very thin adventitia.<sup>10</sup> In the cerebral cortex, penetrating arterioles descend from the cortical surface approximately 2.5 mm deep into the cortex reaching down to the gray white matter intersection.<sup>11</sup> In contrast, deep white matter (DWM) arteries pass through the cerebral cortex with a few branches to the cortex and run straight through the DWM, concentrated ventriculopetally to the white matter around the lateral ventricle. The subcortical and DWM arteries have thicker adventitial sheaths as compared with the cortical vessels.<sup>12</sup>

### Vector of Forces Acting on Brain under Craniectomy Scalp Flap

With the cranium removed from the way, there are two opposing set of forces that start working against each other. Centrifugal ICP has to push against the combination of centripetal forces of atmospheric pressure and the tensile strength of the scalp to create space for itself: the promised “volume augmentation.” After the craniectomy, as the brain shifts laterally through the craniectomy defect, basal cisterns fill up. However, at the pia/scalp interface, where the opposing forces described above come to act against each other, the cortical vessels are likely to be subjected to prolonged compression compromising the cortical blood supply.

### Dural Opening and Hydraulics

Dural opening, the most effective step in reducing ICP, neutralizes the mechanical advantage of floating inside CSF that the cerebral vessels enjoy being inside the subarachnoid space as surface vessels and in Virchow Robin spaces as perforating branches. With this the second line of defense is also lost.

Pulsatile nature of the brain can render protection against this cortical pressure injury by intermittently taking the pressure off; however, in face of refractory raised ICP, this also may lose its effectiveness.

### The hypothesis explains the following common observations on DC:

1. Decompressive hemicraniectomy in malignant MCA infarct: DC reduces ICP and improves the perfusion in the DWM. The contralateral brain shift is reversed in accordance with the volume expansion achieved. This restricts further damage to the tissues in the penumbra as well as that part of the brain which has not been affected by the primary pathology but being compromised because of the raised ICP and brain shift. This is manifested as reduced mortality and improved outcome. In the clinical

conditions in which the cerebral cortex underlying the craniectomy defect has lost its vitality due to the primary pathology, for example, malignant MCA infarct, cortical microvascular compression has no additional ill effect. Only the positive effect of the DC is manifested.

2. Decompressive craniectomy in traumatic brain injury and ICH: In pathologic states in which the pathology is more focal in nature or located in white matter with relative cortical sparing, for example, cerebral contusion and intracerebral hemorrhage (ICH), the craniectomy must overlie healthy (uninvolved by primarily pathology) parts of the brain and in these cases, both the arms of the DC manifest. Though the ICP reduces, DWM hemodynamics stabilize, and brain shifts resolve, compression of the pial vessels and cortical microvasculature cause the underlying otherwise healthy gray cells to become ischemic or infarct. This thin and patchy layer of cortical infarct is not detectable by routine neuroimaging presently in vogue but manifests clinically as poor outcome.
3. Craniectomy size and clinical benefit: The shape and volume of brain parenchyma herniating out of a craniectomy defect depend on the size of the craniectomy and determines the extent of postcraniectomy ICP reduction and presence of certain vascular complications. The ICP reduction and improvement in clinical outcome are hence expected to be proportionate to the size of the craniectomy defect.<sup>13</sup> This hypothesis, however, predicts the possibility of existence of an optimal craniectomy size, which ensures a correct titration of the two arms of the hemodynamics. A very large craniectomy is likely have a deleterious effect on the outcome by exposing healthy cerebral cortical surface to the atmospheric pressure resulting in its vascular jeopardy, disproportionately more than that required to achieve adequate volume expansion.
4. Sinking skin flap syndrome: There have been many hypothesis forwarded to explain this phenomenon.<sup>14</sup> The neurologic damage it produces has been variously ascribed to direct cortical compression, hydrodynamically disturbed CSF parameters, hemodynamically reduced cerebral blood flow (CBF), cerebrovascular reserve capacity, venous return due to pressure on the vasculature and brain tissue, and disturbed metabolism. However, the pathophysiologic process described to be causing the cortical vascular impairment has been the obliteration of the subarachnoid space over the cortex by scarring of the pseudo-dura or reapproximated dura, thus compromising any vessel, especially the veins on the cortex that extends into the subarachnoid space.<sup>15</sup> We propose that as the scalp flap sinks and presses against the pial surface, cortical vessels get compressed, and flow gets compromised irrespective of the scarring referred to above. The surface area of the effected scalp–brain interface being disproportionately more in symptomatic sinking scalp flap syndrome, as compared to that of the causative craniectomy defect itself, the neurological derangements are even more extensive and profound. The proportion of cortical neuronal cells that are ischemic to those that are infarcted, determines the prospect of postcranioplasty neurological recovery.

### Can the Hypothesis Be Validated?

The hypothesis can be tested by postoperative magnetic resonance imaging (MRI) of the brain looking for features similar to cortical lamellar necrosis, albeit patchy, in DC. *Single-photon emission computed tomography (SPECT) for evaluating the cortical vascular status is still evolving and may become an important tool in future.*

### Conclusion

With all the emphasis on the correction of the white matter changes, obvious clinically and on routine neuroimaging, the ill effects of DC on the microvasculature of the cortical neurons have been ignored so far. While the first arm has been useful in improving the outcome of patients in a select group, its optics have blinded the neuroscientists toward the existence of the second arm. It is this second arm that explains the fallacy of a poor neurologic outcome in the face of documented correction of ICP and brain shift. Taking this second aspect of the hemodynamic changes *and devising surgical techniques to take the pressure off the brain surface and thereby from the cortical vessels, while ensuring reduction in ICP, are likely to prove vital in future patient management.*

#### Conflict of Interest

None.

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