

# Hydrocephalus Developed after Cranioplasty : Influence of Cranioplasty on the CSF Circulation

Seok Won Kim, M.D., Seung Myung Lee, M.D., Ho Shin, M.D.

Department of Neurosurgery, College of Medicine, Chosun University, Gwangju, Korea

Hydrocephalus is usually defined as the condition of ventricular dilatation due to the overproduction of cerebrospinal fluid(CSF) or dysfunction of absorption. The pattern of the CSF circulation may change after a cranioplasty secondary to previous decompressive craniectomy for refractory intracranial hypertension after head injury. The effect of the cranioplasty on CSF hydrodynamics has not been explored exactly. We report two cases of acute hydrocephalus developed after cranioplasty and discuss about the clinical importance with review of literatures.

**KEY WORDS :** Cranioplasty · Hydrocephalus · CSF hydrodynamics.

## Introduction

Traditional views of hydrocephalus unify the varying etiological processes as having in common defect in the absorption of CSF, creating an imbalance between the formation and absorption of CSF<sup>5)</sup>. This depiction of the hydrocephalus implies that the ventricles will dilate inexorably at the expense of brain as excess CSF is stored. The effect of the skull and dura on CSF hydrodynamics has been explored experimentally : the resistance to CSF outflow after craniectomy decreases twofold and brain compliance increases<sup>2)</sup>. This problem is important clinically as cranioplasty may be an causing factor of hydrocephalus. We have experienced two patients who had acute hydrocephalus after cranioplasty procedure. Thus we report these cases and discuss about the clinical importance with review of the literatures.

## Case Report

### Case 1

A 27-year-old man fell downstairs and was admitted with a Glasgow Coma Score(GCS) of 8. His mental status was stuporous. Brain computed tomography(CT) reveals

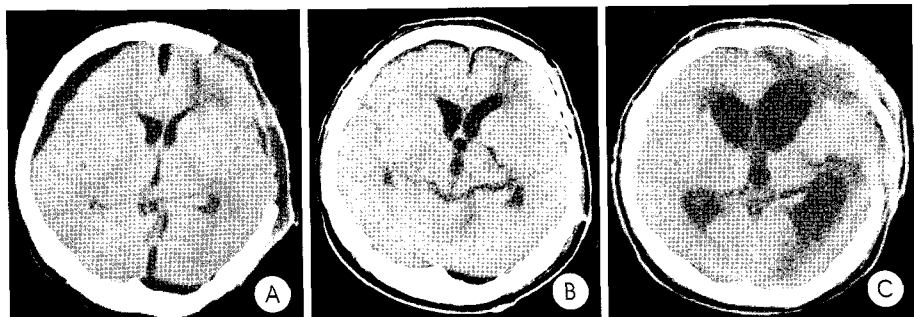
an subdural hematoma, which required decompressive craniectomy to control raised intracranial pressure(ICP).

Two months later he recovered fully with GCS of 15 (Fig. 1A).

Cranioplasty was carried out with autologous bone without any difficulty (Fig. 1B). However, 3 days later, he complained of gait disturbance, slurred speech and urinary incontinence. Brain CT showed a progressive severe ventricular dilatation with widening of cortical sulci (Fig. 1C). He was treated by lumbar-peritoneal shunt and fully recovered one day after the operation.

### Case 2

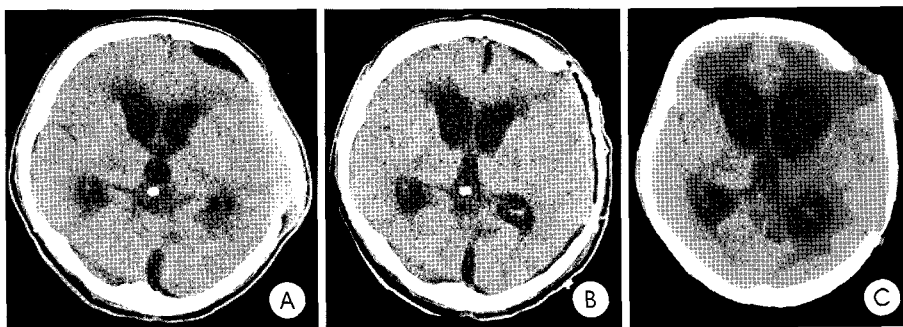
A 32-year-old man stroke by a car and was admitted with semicomatose mentation. His GCS was 4. Brain CT disclosed



**Fig. 1.** A : Brain computed tomography scan shows slight dilatation of ventricle and craniectomy state. B : Postoperative computed tomography scan shows good approximation of bone flap. C : Third postoperative day computed tomography scan shows projection of bone flap due to hydrocephalus.

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Address for reprints : Seung Myung Lee, M.D., Department of Neurosurgery, College of Medicine, Chosun University, 588 Seoseok-dong, Dong-gu, Gwangju 501-717, Korea Tel : +82-62-220-3120, 3126, Fax : +82-62-227-4575, E-mail : chosunns@hanmail.net



**Fig. 2.** A : Brain computed tomography scan reveals slight dilatation of ventricle and craniectomy state. B : Postoperative computed tomography scan reveals good approximation of bone flap. C : Seventh postoperative day computed tomography scan shows periventricular low density and projection of bone flap due to hydrocephalus.

an subdural hematoma with midline shift, which required decompressive craniectomy to control raised ICP. Three months later he remained disabled but recovered to drowsy mentation (Fig. 2A). Cranioplasty was performed without any difficulty (Fig. 2B). But 7 days later, he showed neurological deterioration with increasing spasticity. Brain CT showed a hydrocephalus (Fig. 2C). Lumbar-peritoneal shunt was performed and he recovered to the state before the cranioplasty.

## Discussion

Hydrocephalus is defined as the condition of ventricular dilatation due to the overproduction of CSF and dysfunction of absorption.

One way of analyzing steady-state CSF pressure is to relate the sagittal sinus venous pressure(SSVP), rate of CSF formation and CSF absorption by the formula : CSF pressure = (CSF formation  $\times$  CSF absorption) + SSVP<sup>1,2</sup>.

In the present series of experiments, we have shown that changes in the tissues enveloping the brain cause minimal alteration in SSVP and CSF pressure while markedly decreasing resistance to CSF absorption. Using this steady-state equation and assuming relatively constant CSF production, the contribution of the (CSF formation  $\times$  CSF absorption) product to total CSF pressure decreases from 22% in the intact cat to 13% after craniectomy and 6% after durectomy<sup>6</sup>. Although SSVP remains constant after each alteration of the container, the progressive decrease in resistance to CSF absorption demonstrated by both bolus and infusion techniques makes the relative contribution of SSVP to total CSF pressure greater with each alteration of the container. For the most part, SSVP determines CSF pressure in the steady state. In settings with large changes in CSF hydrodynamics, the apparent effect on CSF pressure is minimal so long as the sinus pressure remains stable. Intriguingly, one would expect that maneuvers that increase the neural axis volume-buffering capacity would lower CSF pressure. Conceptually, the increase in pressure-volume index(PVI)

following craniectomy with or without durectomy should favor the accumulation of volume within the neural axis. The sequential decrease in resistance to CSF absorption following each of these maneuvers facilitates the absorption of CSF and maintains steady-state CSF pressure. The depiction of steady-state CSF pressure cited above does not include a parameter of volume storage. This equation relates changes of CSF hydrodynamics to CSF

pressure in a system described by a fixed neural axis pressure-volume curve. If volume is added to the system at steady state, CSF pressure will change transiently along the pressure-volume curve, but will return to a steady-state CSF pressure as volume is absorbed along outflow pathways described by the resistance to CSF absorption parameter. Thus, the steady state is re-established over time. While both the PVI and resistance to CSF absorption parameters are independent of one another, the changes in each after the container of the brain is altered are compatible with maintenance of a stable balance between volume buffering and the circulation of CSF. If the resistance to CSF absorption did not decrease with poening of the skull and dura, CSF volume would accumulate because of the ease of volume storage induced by the increase in PVI.

The patient had developed an hydrocephalus, possibly as a result of cranioplasty. Craniectomy state was a factor allowing compensation of CSF circulation in the early stages. Therefore, a mechanistic increase in compliance after craniectomy tends to be followed by a decrease in the resistance to CSF outflow. This process may be reversed after cranioplasty. That is, a decrease in PVI may be followed by an increase in the resistance to CSF outflow<sup>3,6</sup>. A large craniectomy may facilitate irreversible ventricular dilatation over weeks or months<sup>4</sup>. Thus, after cranioplasty, the expanded ventricles may, via the cerebral mantle, obstruct the lumen of the cortical subarachnoid space and increase the resistance to CSF outflow. Although the head injury itself can induce the development of hydrocephalus, cranioplasty procedure influence the CSF dynamics by increase the resistance to CSF outflow. So volume buffering- capacity and CSF dynamics study are necessary in the future.

## Conclusion

Our cases demonstrate that cranioplasty may be an causing factor of hydrocephalus by increasing resistance to CSF absorption. We must keep in mind the possibility of hydrocephalus after cranioplasty procedure.

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