Acta Neurochir Suppl (2008) 102: 15–20

Cranioplasty effect on the cerebral hemodynamics and cardiac function

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Abstract
Background Cranioplasty is usually performed for aesthetic, protective and patient comfort reasons. The objective of this study is to examine the effects of cranioplasty on the cerebral hemodynamics and cardiovascular system.
Methods Twenty-seven patients who had undergone cranioplasty after extensive skull bone removal to prevent uncontrollable intracranial hypertension were included in this study. Arterial blood flow velocities in the middle cerebral artery (MCA) and internal carotid artery (ICA) were assessed by transcranial doppler (TCD). The cardiac functions were evaluated using the echocardiogram. And cerebral blood flow were measured by perfusion CT.
Findings The blood flow velocity at the MCA ipsilateral to the cranioplasty was decreased from 50.5±15.4 cm/s preoperative to 38.1±13.9 cm/s following cranioplasty ($p<0.001$) and from 33.1±8.3 cm/s to 26.4±6.6 cm/s at the ICA ($p<0.001$). The stroke volume was increased from 64.7±18.3 ml/beat, to 73.3±20.4 ml/beat ($p<0.001$), while the cardiac output and mean arterial blood pressure were unchanged. The cerebral blood flow was increased from 39.1±7.2 ml/100g/min to 44.7±8.9 ml/100g/min on the cranioplasty side (P=0.05).
Conclusions Cranioplasty can get rid of the atmospheric pressure on the brain and increase the cerebral blood flow as well as improve the cardiovascular functions. A skull defect should be corrected, because cranioplasty has not only aesthetic or protective effects but also improves the cardiovascular functions.

Keywords Cranioplasty · Stroke volume · Atmospheric pressure · craniectomy

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Introduction

Cranioplasty is usually indicated for cosmetic or protective, but recent reports have suggested that its also has therapeutic effects [2, 4, 7, 12, 14, 16, 19]. Most neurosurgeons agree that the skull defect should be corrected after the offending pathologic problems had been resolved. But in patients with a poor neurological status, neurosurgeons should consider the surgical risks.

We hypothesized that, in patients with skull defect, the atmospheric pressure compresses the brain and this may have effects on the cardiovascular functions as well as the cerebral hemodynamics. This study was designed to investigate the cerebral blood flow velocity, cerebral blood flow and the cardiac function before and after cranioplasty.

Patients and methods

Patient population

Twenty-seven patients with skull bone defect larger than 100 cm^2 were included in this study. Clinical data on the patients are presented in Table 1. All patients underwent bilateral (17 cases) or unilateral (ten cases) craniectomy. After the offending pathologic problems had been resolved, cranioplasty with freeze autologous bone were performed.

Table 1 Clinical data and outcome obtained in 27 patients who underwent cranioplasty

Case No.	Age (years), sex	Reason for craniectomy	Laterality of craniectomy	Time Lapse (weeks)	GCS score cranioplasty		Changed neurologic symptoms
					Before	After	
1	44, F	Massive brain swelling due to Rt. contused ICH	Bilateral	6	6	6	No change in neurologic condition
2	43, M	Rt. acute SDH with brain swelling	Bilateral	6	14	15	Improved headache and dizziness
3	61, F	Rt. acute SDH with brain swelling	Bilateral	8	8	10	Brisk eye opening
4	65, M	Brain swelling post-SAH, Lt. A-comm. aneurysm	Bilateral	8	8	10	Brisk eye opening
5	26, M	Rt. acute SDH with brain swelling	Right	6	15	15	Improved restlessness
6	52, F	Lt. putaminal H-ICH	Left	6	15	15	Improved right hemiparesis, paresthesia
7	57, F	Vasospasm post-SAH, Lt. A-comm. aneurysm	Bilateral	16	8	8	No change in neurologic condition
8	40, M	Rt. acute SDH with brain swelling	Bilateral	5	6	8	Brisk eye opening
9	16, M	Lt. EDH	Left	12	6	8	Brisk eye opening
10	62, F	Vasospasm post-SAH, Lt. MCA aneurysm	Left	5	12	14	Improved headache, orientation
11	78, F	Rt. acute SDH with brain swelling	Bilateral	3	6	6	No change in neurologic condition
12	25, M	Massive brain swelling due to Lt. contused ICH	Bilateral	5	12	15	Improved orientation
13	63, M	Massive brain swelling due to Lt. contused ICH	Left	4	12	14	Improved orientation
14	42, M	Rt. acute SDH with brain swelling	Bilateral	14	10	12	Improved verbal response, orientation
15	46, F	Rt. putaminal H-ICH	Bilateral	13	6	6	No change in neurologic condition
16	41, F	Rt. putaminal H-ICH	Bilateral	8	8	13	Improved verbal response, orientation
17	48, M	Rt. acute SDH with brain swelling	Right	5	15	15	Improved headache, fine movement
18	57, F	Rt, putaminal H-ICH	Right	10	8	12	Improved mentality, orientation
19	62, F	Lt. acute SDH with brain swelling	Left	7	15	15	Improved restlessness
20	41, M	Vasospasm post-SAH, Rt. A-comm. aneurysm	Bilateral	8	6	6	No change in neurologic condition
21	60, F	Massive brain swelling due to Rt. contused ICH	Bilateral	8	8	13	Improved verbal response, orientation
22	66, F	Brain swelling post-SAH, Rt. MCA aneurysm	Bilateral	6	6	6	No change in neurologic condition
23	69, F	Rt. putaminal H-ICH	Bilateral	4	13	15	Improved orientation, restlessness
24	69, F	Rt. putaminal H-ICH	Bilateral	6	13	14	Improved orientation, headache
25	45, M	Brain swelling post-SAH, Lt. A-comm. aneurysm	Bilateral	10	9	12	Improved verbal response, orientation
26	19, F	Massive ICH due to Lt. parietal AVM	Left	3	14	15	Improved right hemiparesis, dizziness
27	72, M	Cerebral infarction due to Lt. ICA occlusion	Left	5	12	13	Improved right hemiparesis, orientation

Time Lapse = duration from craniectomy to crnioplasty

EDH epidural hematoma; *GCS* Glasgow coma scale; *H-ICH* hypertensive intracerebral hematoma; *ICH* intracerebral hematoma; *SDH* subdural hematoma;

Table 2 Mean values in echocardiogram before and after the cranioplasty

Case no.	Cardiac output (ml/min)		Stroke volume (ml/beat)		Heart rate (beat/min)	
	Before	After	Before	After	Before	After
1	3,942	5,154	41.1	50	96	103
2	5,849	8,789	77.9	113	75	78
3	5,601	5,081	64.4	69.6	87	73
4	6,749	7,058	61.4	65.4	110	108
5	5,111	5,512	74.1	75.5	69	73
6	7,201	6,276	84.7	89.7	85	70
7	6,588	6,334	64.6	67.9	97	98
8	4,414	4,193	58.1	67.6	76	62
9	3,590	3,281	34.9	38	103	86
10	5,627	4,415	64.6	62.6	87	66
11	8,675	9,165	92.3	119	94	77
12	6,080	6,337	78	84.5	77	75
13	8,610	7,200	106	112	81	64
14	6,328	5,390	64	67.9	99	79
15	3,169	3,690	47.3	61.7	67	60
16	5,160	5,560	57.9	69	89	80
17	4,290	5,550	47.7	68.3	90	81
18	3,340	3,650	33.9	44.9	99	81
19	7,895	7,880	84.9	98.5	93	80
20	6,800	6,500	62.1	56.6	120	104
21	6,960	6,070	80.3	74.8	93	76
22	8,750	8,520	97.2	99	90	86
23	6,150	8,240	57.8	67.6	106	122
24	8,240	6,030	65.2	67.2	126	90
25	5,228	5,450	61.5	67.3	85	81
26	3,370	3,770	55.9	49.3	68	67
27	4,580	5,110	52.4	44.5	103	98
Mean±SD	5,862±1,705	5,933±1,621	65.6±17.9	72.3±21.2	91.4±14.7	82.2±15.1

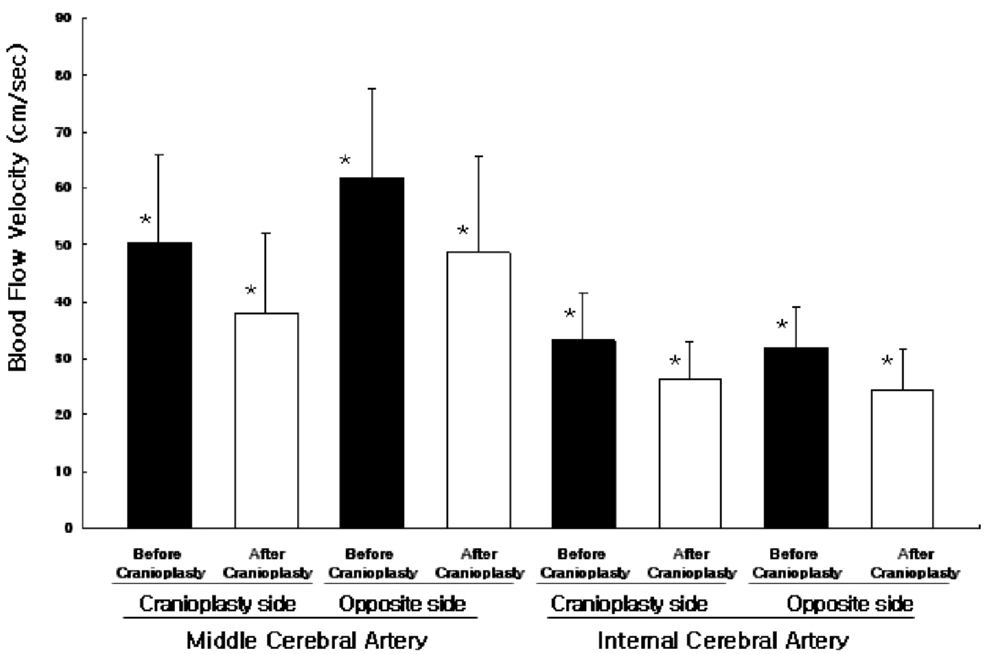

Fig. 1 Bar graph depicting the influence of cranioplasty on the cerebral blood flow velocity obtained in 27 patients (mean ±SD). The *symbols on the left of the bars* show the significance (*$p<0.05$) compared with before cranioplasty

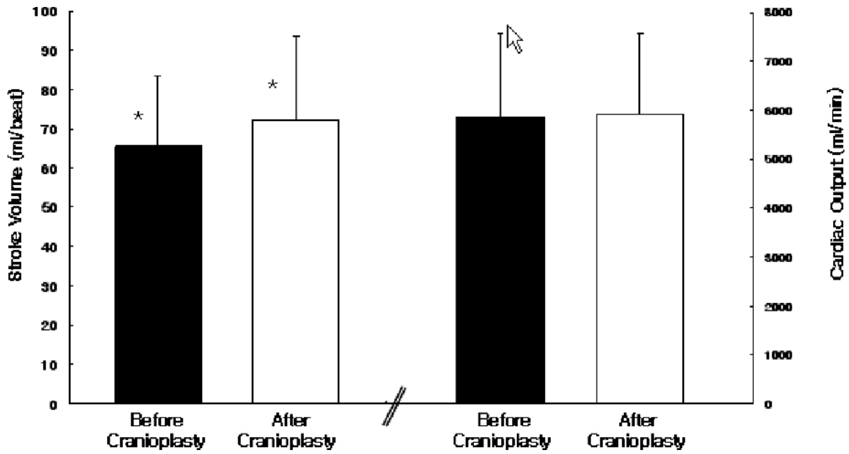

Fig. 2 Bar graph depicting the influence of cranioplasty on the stroke volume and the cardiac output (mean±SD). The *symbols on the left of the bars* show the significance (*$p<0.05$) compared with before cranioplasty

In all patients, echocardiography, cerebral blood fow velocity by transcranial Doppler and Ct perfusion cerebral blood flow studies were performed before and after the cranioplasty.

Data collection

Transcranial doppler ultrasonography (TC2020; Eden Medical Electronics, Lake Constance, Germany) was performed to determine the blood flow velocities in the both middle cerebral arteries (2mm distal from the ICA bifurcation site) and both extracranial intracranial carotid arteries (depth 46 mm from the both madibular angle). From the echocardiography (Sonos 5500; Hewlett Packard, Andover, MA, USA) routine echocardiographic values, cardiac output and stroke volume were measured (Table 2). Perfusion CT (Somatom plus 4; Siemens, Forcheim, Germany) was performed to evaluate the cerebral blood flow changes. All of these studies were performed before and 14±2 days after the cranioplasty operation.

Statistical analysis

All values are presented as the mean ± standard deviation. Comparisons between data groups were computed using Student's t-test. Statistical significance was defined as a probability value of less than 0.05.

Results

Changes of the cerebral blood flow velocity

The blood flow velocity in the MCA ipsilatral to the cranioplasty was decreased from 50.5±15.4 cm/s pre-operatively to 38.1±13.9 cm/s ($p<0.001$) post-operatively and from 33.1±8.3 to 26.4±6.6 cm/s ($p<0.001$) in the ICA. Blood flow velocity on the contralateral side was decreased from 61.9±15.7 to 48.7±16.9 cm/s ($p=0.002$) at the MCA and from 31.8±7.3 to 24.5±7.1 cm/s ($p<0.001$) at the ICA (Fig. 1).

Effect on the cardiac function and systemic blood pressure

The cardiac function evaluation with echocardiogram revealed that the stroke volume was increased from 64.7± 18.3 to 73.3±20.4ml/beat ($p<0.001$). The cardiac output showed no statistical significance ($p=0.731$) before and after the cranioplasty (Fig. 2).

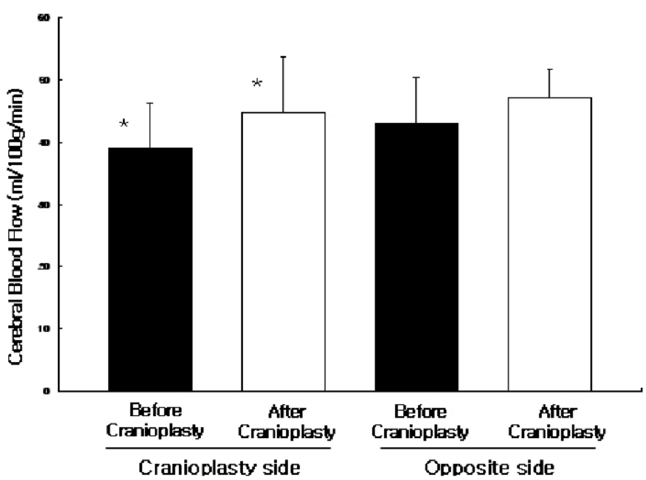

Fig. 3 Bar graph depicting the influence of cranioplasty on the cerebral blood flow (mean±SD). The *symbols on the left of the bars* show the significance (*$p<0.05$) compared with before cranioplasty

Cerebral blood flow changes

The cerebral blood flow, evaluated with perfusion CT, in terms of the hemispheric values were increased on both sides (Fig. 3). On the cranioplasty side it was increased from 39.1±7.2 to 44.7±8.9 ml/100 g/min ($p=0.05$), and on the contralateral side, from 42.9±7.5 to 47.2±4.4 ml/100 g/min ($p=0.137$).

Discussion

Accepted indications for cranioplasty are for aesthetic and for physical protection of intracranial structures. Recently, the potential improvement of neurological function should be added as an acceptable indication for cranioplasty [6–8, 12, 15, 16, 19, 24].

Gardner reported a syndrome characterized by headaches, dizziness, irritability, epilepsy, discomfort, and psychiatric symptoms that he had observed in patients with large cranial defect. He called it the "syndrome of the trephined" and was the first to describe an improvement in the neurological function of some patients who underwent cranioplasty with tantalum [4, 6–8, 10, 15]. The mechanism of "syndrome of trephined" remains controversial. These mechanisms include, cicatrical changes occurring in the cortex, dura, and skin that may exert pressure on the skull contents [6, 8, 15, 24], cerebrospinal fluid hydrodynamic changes [7–9, 15–17], the effects of atmospheric pressure on the brain cerebral blood flow changes [7, 11, 12, 16, 18, 21, 24] and disturbed cerebral energy metabolism [15, 22, 24].

Previous reports have shown, a 15 to 30% increase in cerebral blood flow in the area of cortex adjacent to the cranioplasty [5, 13, 15, 16, 21, 24]. Furthermore cranioplasty after decompressive craniectomy may increase cerebral blood flow in the ipsilateral hemisphere, as well as the contralateal hemisphere [11, 21, 24]. In our study, the cerebral blood flow on the cranioplasty side was increased as observed by others in these previous studies.

Scar tissue and atmospheric pressure produced by skull defect may increase pressure on the brain cortex and subarachnoid space [8, 16, 18, 19]. Consequently, vessels under the skull defect, with added atmospheric compression may alter their cerebral blood flow hemodynamics [5–7, 12, 15, 20, 22, 24]. Under such circumstances, cerebral blood flow velocity of the compressed vessel may increase. Following the law of Laplace, we suggest that the diameter of cerebral blood vessels may be dilated after cranioplasty, based on our observations of a decrease in blood flow velocities and an increase in cerebral blood flow following the cranioplasty procedure [23].

Cardiac output is mathematically equal to the stroke volume multiplied by the heart rate [2, 3]. Stroke volume is influenced by peripheral vascular resistance [1–3]. In echocardiogram studies, stroke volume was increased and the heart rate was decreased after cranioplasty while the cardiac output and systemic blood pressure were unchanged.

Based on the results of our study, cranioplasty may remove the compression that arise from the atmospheric pressure and scar tissue [4, 12]. As a result, the cerebral blood flow was increased and cardiac function improved following cranioplasty. Therefore cranioplasty should be performed even on the poor neurologic patients, because cranioplasty has not only aesthetic and skull protective effects but it also improves the systemic cardiovascular and cerebral hemodynamic functions.

Conflict of interest statement We declare that we have no conflict of interest.

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