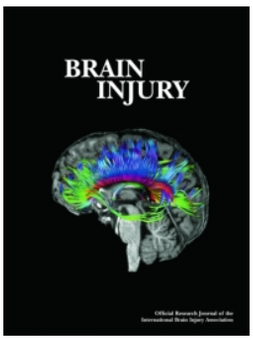

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ORIGINAL ARTICLE

The impact of cranioplasty on cerebral blood perfusion in patients treated with decompressive craniectomy for severe traumatic brain injury

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Abstract

Background: A large cranial defect following decompressive craniectomy (DC) is a common sequela in patients with severe traumatic brain injury (TBI). Such a defect can cause severe disturbance of cerebral blood flow (CBF) regulation. This study investigated the impact of cranioplasty on CBF in these patients.

Methods: Patients who underwent DC and secondary cranioplasty were prospectively studied for a severe TBI. CT perfusion was used to measure CBF before and after cranioplasty. The basal ganglia, parietal lobe and occipital lobe on the decompressed side were chosen as zones of interest for CBF evaluation.

Results: Nine patients representing nine cranioplasty procedures were included in the study. Before cranioplasty, CBF on the decompressed side was lower than that on the contralateral side. During the early stage (10 days) after cranioplasty, CBF on the decompressed side was increased and this increase was significant in the parietal and occipital lobe. CBF was also increased on the contralateral side. In addition, the difference in CBF between the contralateral side and the decompressed side was reduced after cranioplasty. Further, the CT perfusion showed that the CBFs decreased again 3 months post-cranioplasty among four cases, but was still higher than those before cranioplasty.

Conclusions: This study indicates that cranioplasty may increase CBF and benefit the recovery in patients with DC for TBI.

Keywords

Cranioplasty, cerebral blood perfusion, decompressive craniectomy, traumatic brain injury

History

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Introduction

Recently, decompressive craniectomy (DC) has been advocated as an effective treatment for refractory intracranial hypertension, particularly in patients with severe traumatic brain injury (TBI). It has been proven that DC is able to reduce the elevated ICP (intracranial pressure) effectively and this procedure has been recommended as a second-tier therapy for the refractory intracranial hypertension after severe TBI [1, 2]. However, application of DC remains controversial, partly due to the high incidence of complications [3]. Whether this procedure can improve the patients' outcomes is still doubtful. On the other hand, a large cranial defect is a definite sequela following DC. With the improvement of brain oedema, some patients develop severe sinking skin flap syndrome with neurologic symptoms of headache, dizziness, irritation and psychiatric presentations. Over the last few decades, increasing evidence has demonstrated that cranioplasty can improve cerebral blood flow (CBF) and cerebral metabolism following DC [4].

CBF following TBI plays an important role in the clinical outcome in these patients. Specifically, DC is believed to be able to disturb CBF self-regulation and result in clinical symptoms. Moreover, it has been reported that a large cranial defect causes low CBF in the hemisphere of the decompressed side and is responsible for clinical symptoms following DC [5, 6]. Cranioplasty is a cosmetic surgery that is applied for brain protection as well. In addition, it can rectify some pathophysiologic changes following DC and subsequently improves clinical outcomes in these patients. In the present study, in order to study the effect of cranioplasty on CBF, CBF was assessed before and after cranioplasty in patients undergoing DC for a severe TBI via computed tomography (CT) perfusion (CTP).

Methods

Patients and outcome measures

The impact of cranioplasty on CBF was prospectively studied. In total, nine patients who underwent DC and cranioplasty for

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a TBI at the First Affiliated Hospital, College of Medicine, Zhejiang University between January 2013 and May 2013 were enrolled in this study. One patient had undergone two cranioplasty procedures because of a former bilateral decompressive craniectomy. The study was approved by the Ethics Committee of the First Affiliated Hospital, College of Medicine, Zhejiang University. Written informed consent was obtained from all patients. Patients who developed other severe complications, including hydrocephalus and large cerebral infarction, secondary to TBI at the time of cranioplasty or had other baseline neurological disorders prior to TBI were excluded from this study. The modified Rankin scale (mRS) and Glasgow Outcome Scale (GOS) were used to evaluate clinical outcomes in these patients at 1 year after TBI. mRS is an ordinal hierarchical scale that describes grades of disability from 0–6 (0 = no symptoms; 1 = no significant disability despite symptoms: able to carry out all usual duties and activities; 2 = slight disability: unable to carry out all previous activities but able to look after own affairs without assistance; 3 = moderate disability: requiring some help, but able to walk without assistance; 4 = moderately severe disability: unable to walk without assistance and unable to attend to one's own bodily needs without assistance; 5 = severe disability: bedridden, incontinent and requiring constant nursing care and attention; and 6 = death), as indicated previously [7].

Surgical procedure of cranioplasty

All patients underwent unilateral fronto-temporo-parietal DC for management of malignant intracranial hypertension induced by severe TBI. They were treated by cranioplasty at the department 2–8 months after their first DC. A three-dimensional, digitally manufactured titanium plate was used in the repair of the large cranial defect for all patients.

CTP imaging and measurement of CBF

To measure CBF, CTP imaging was performed during the time frame of 1 week before to 10 days after cranioplasty. Specifically, the Philips Brilliance iCT 256-layer spiral CT scanner was used. All patients had baseline non-contrast brain CT, which was conducted at 120 kV, 300 mA and 5 mm per layer with a layer spacing of 5 mm. Brain CTP was conducted on top of CT imaging by conducting a perfusion scan of 16 layers. The scan was conducted at 80 kV, 100 mA and 5 mm per layer, with the layer spacing of 5 mm. The scan started after a delay of 5 seconds with injection of non-ionic contrast medium (iohexol 300 mg I ml^{-1}) with a high-pressure syringe through the cubital vein with a flow rate of 6.0 ml s^{-1} and dose of 50 ml. The frequency of the tube was once per second, with data collection of 16 images once per 1.5 seconds. The scan continued for 45 seconds for collection of a total of 480 images.

CTP raw data were transferred to the workstation. Specifically, the contralateral anterior cerebral artery was used as the inflow artery and the superior sagittal sinus at the same layer as the outflow vein. By software analysis, maps for CBF, cerebral blood volume (CBV), mean transit time (MTT) and time to peak (TTP) were generated. The region of interest (ROI) area was regarded as parameter measurement. Also, the aim was to avoid major blood vessels, the skull and cerebrospinal fluid. Specifically, each ROI was placed in the ipsilateral side and then mirrored symmetrically on the opposite side using the centre of the brain as the axis. Each ROI was placed in four continuous layers of the maps to record CBF, CBV, MTT and TTP parameter values in bilateral symmetrical areas. The values of the four ROI parameters taken from the ipsilateral side were compared to those from the symmetrical location on the non-operated side. Ratios were calculated from the average values to obtain four relative parameter values: the relative CBF (rCBF), the relative CBV (rCBV), the relative MTT (rMTT) and the relative TTP (TTP).

Given the fact that a cerebral contusion would be largely replaced by encephalomalacia without detectable CBF during the chronic phase of TBI, the basal ganglia, parietal lobe and occipital lobe on the decompressed side were chose as the zones of interest to avoid any potential influence on the results. In addition, the CBF of the corresponding zone on the contralateral hemisphere was measured.

Data analysis and statistical methods

All data are presented as mean ± standard deviation (SD) values. The ipsilateral CBFs on the decompressed side and the contralateral side were recorded. Specifically, CBF was compared between the decompressed side and the contralateral side. All results were compared before and after the procedure. Furthermore, the differences in CBF between the contralateral side and the decompressed side before and after the procedure were compared. Two-tailed paired *t*-tests were used to analyse paired samples. An error probability of less than 0.05 was considered statistically significant.

Results

The patients' clinical characteristics, including demographic data, clinical presentations of TBI and outcomes, are shown in Table I. As mentioned before, finally nine cranioplasty procedures were included (six males and three females; mean age = 41.6 years [range = 19–62 years]). All cranial defect sizes were larger than 10 × 12 cm. Eight patients underwent cranioplasty during the first 6 months after TBI and only one case underwent cranioplasty 8 months after TBI. Before cranioplasty, except the two patients who underwent cranioplasty 2 months post-DC, there were seven patients who developed severe sinking skin flap. Seven patients had severe disability (GOS 3) prior to cranioplasty and, at 1 year after TBI, six patients still needed help with activities of daily living. However, the mRS scores of five patients were improved following cranioplasty. The procedure went well in almost all patients, with only one patient developing hydrocephalus 2 months after cranioplasty and requiring subsequent treatment with a ventriculo-peritoneal shunt.

The CBFs of the basal ganglia, parietal lobe and occipital lobe on the operated side before and 10 days after cranioplasty were measured and compared (Table II). The results showed that CBF of all three domains increased following cranioplasty and the increase was statistically significant in the parietal lobe and occipital lobe. In the parietal lobe, CBF

Table I. The patients' information about demographics, head trauma and outcomes.

Case no.	Gender	Age	Injury mechanism	Main diagnosis	GCS	Time	Outcome before cranioplasty GOS	Outcome before cranioplasty mRS	Outcome after cranioplasty GOS	Outcome after cranioplasty mRS
1*	F	48	Traffic accident	Right ICH (temporal lobe) & ASDH	5	5M	3	5	3	4
2*	M	62	Traffic accident	Left ASDH & AEDH & cerebral contusion (frontal and temporal lobe)	5	6M	3	5	3	5
3	M	50	Fall	Right ASDH & cerebral contusion (temporal lobe)	6	3M	3	5	3	5
4	M	19	Fall	Right ASDH & cerebral contusion (frontal and temporal lobe)	8	2M	3	4	3	2
5	M	29	Traffic accident	Right ICH & ASDH & cerebral contusion (frontal and temporal lobe)	8	5M	4	2	5	1
6	M	48	Traffic accident	Right ICH (temporal lobe) & ASDH	5	8M	3	5	3	5
7*	M	45	Fall	Right ASDH & cerebral contusion (temporal lobe)	6	2M	4	2	4	2
8*	F	30	Fall	Left ASDH & cerebral contusion (frontal and temporal lobe)	4	4M	3	5	4	2
9	F	61	Fall	Right ICH & ASDH & cerebral contusion (frontal and temporal lobe)	4	3M	3	4	3	3

GCS, GCS before decompressive craniectomy; ASDH, acute subdural haematoma; ICH, intracerebral haematoma; AEDH, acute epidural haematoma; Outcome after cranioplasty, the patient's outcome was assessed at the time 1 year after head trauma; *, patient whose CBF was measured at the time of 1 year after TBI.

Table II. Comparison of CBF (cerebral blood flow) before and after cranioplasty.

		Before cranioplasty			After cranioplasty		
	n	Parietal lobe	Occipital lobe	Basal ganglia	Parietal lobe	Occipital lobe	Basal ganglia
CBF of decompressive side ($cm^3/100\,g\,min^{-1}$)	9	46 ± 19	42 ± 20	59 ± 29	76 ± 38*	59 ± 16*	77 ± 35
CBF of contralateral side ($cm^3/100\,g\,min^{-1}$)	9	55 ± 34	49 ± 24	72 ± 39	81 ± 43*	68 ± 50	68 ± 26

Two-tailed paired *t*-test, *$p < 0.05$ (comparing with CBF before cranioplasty).

increased from $46 \pm 19\,cm^3/100\,g\,min^{-1}$ to $76 \pm 38\,cm^3/100\,g\,min^{-1}$; whereas, in the occipital lobe, CBF increased from $42 \pm 20\,cm^3/100\,g\,min^{-1}$ to $59 \pm 16\,cm^3/100\,g\,min^{-1}$. Besides, among four of these patients the CBFs were measured again at the time of 3 months after cranioplasty. The CBFs of the parietal lobe, occipital lobe and basal ganglia on the operated side were $47 \pm 6\,cm^3/100\,g\,min^{-1}$, $53 \pm 10\,cm^3/100\,g\,min^{-1}$ and $59 \pm 24\,cm^3/100\,g\,min^{-1}$, respectively. CBFs of the parietal lobe, occipital lobe and basal ganglia on the contralateral side were $47 \pm 10\,cm^3/100\,g\,min^{-1}$, $60 \pm 17\,cm^3/100\,g\,min^{-1}$ and $49 \pm 18\,cm^3/100\,g\,min^{-1}$, respectively. It seems that the CBFs increased in the early stage after cranioplasty, but decreased again 3 months later (Figures 1 and 2).

This study also compared CBF on the side contralateral to the cranioplasty before and after the surgical procedure (Table II). CBF was increased in the parietal and occipital lobes and decreased in the basal ganglia. The changes of CBF in the parietal lobe were statistically significant, increasing from $55 \pm 34\,cm^3/100\,g\,min^{-1}$ to $81 \pm 43\,cm^3/100\,g\,min^{-1}$.

In addition, CBF was compared between the decompressed side and the contralateral side before and after cranioplasty (Table III). Prior to cranioplasty, the CBF values in all three domains on the decompressed side were lower than those on the non-operated side, but these differences were without statistical significance. Following cranioplasty, the CBF values in both the parietal and occipital lobes on the decompressed side remained lower than those on the non-operated side. The changes of CBF intervals between the non-operated hemisphere and the decompressed hemisphere on these two zones were without statistical significance. The CBF of the basal ganglia of the decompressed side was higher than that on the contralateral side after cranioplasty, although the change was not statistically significant either.

In summary, after cranioplasty the CBFs of the basal ganglia, parietal lobe and occipital lobe increased on the decompressive hemisphere and the changes had statistical significance in the parietal lobe and occipital lobe. On the contralateral hemisphere CBF was increased in the parietal and occipital lobes after cranioplasty either, but decreased in the basal ganglia. The changes of CBF in the occipital lobe were statistically significant. Besides, before cranioplasty the CBF values on the decompressed side were lower than those on the non-operated side, but without statistical significance.

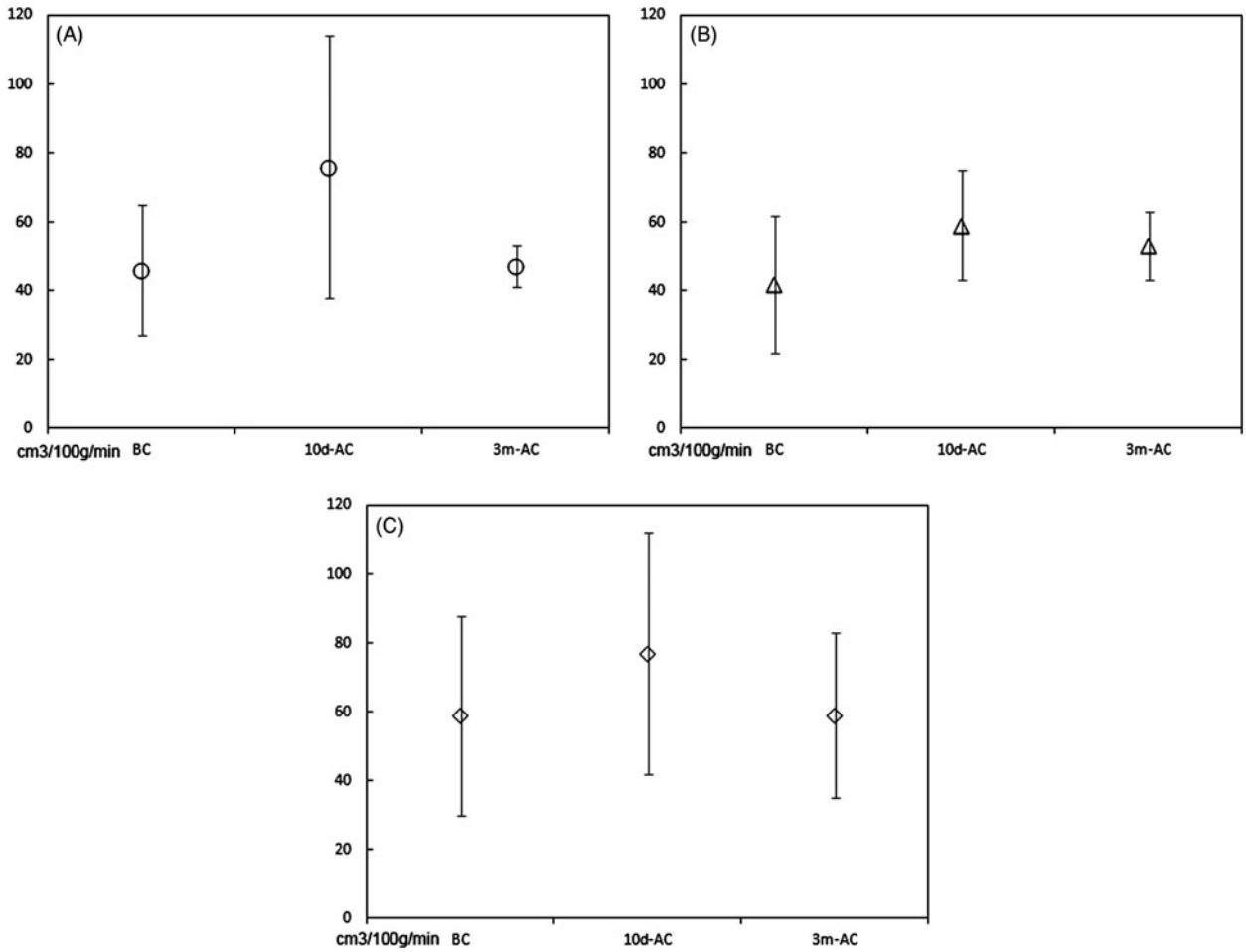

Figure 1. CBF (cerebral blood flow) changes on the decompressive hemisphere after the cranioplasty among patients who underwent decompressive craniecotmy for a severe head trauma (BC, before cranioplasty; 10 d-AC, 10 days after cranioplasty; 3m-AC, 3 months after cranioplasty). (A) CBF changes of parietal lobe on the decompressive side. (B) CBF changes of occipital lobe on the decompressive side. (C) CBF changes of basal ganglia on the decompressive side. From these figures (A, B, C), after the cranioplasty the CBF on the decompressive hemisphere seemed to increased on the acute phase, but decreased again 3 months post-cranioplasty.

After cranioplasty, the CBF interval between the non-operated hemisphere and the decompressed hemisphere was decreased; and the CBF of the basal ganglia of the decompressed side was even higher than that on the contralateral side.

Discussion

Currently, DC is an important treatment for malignant intracranial hypertension and increasing evidence demonstrates that it can be used to decrease elevated intracranial pressure rapidly and effectively. Thus, this procedure has been widely performed to save patients' lives, especially those with severe TBI. Also, it has been demonstrated that DC can improve CBF dysfunction resulting from intracranial hypertension [8]. Based on a meta-analysis, Bor-Seng-Shu et al. [9] reported that the post-operative cerebral perfusion pressure (CPP) is significantly higher than the pre-operative CPP with reduced intracranial pressure. However, it must be noted that patients who undergo DC have to live with a large cranial defect. Large cranial defects can lead to dynamic changes in CBF and contribute to a number of neurologic symptoms. Following the acute phase of TBI, most patients who complain of headache, dizziness, irritation, discomfort and psychiatric illness, a condition referred to as 'syndrome of the trephined', have a poor clinical outcome [10]. Almost all of the patients would have such complications 2 or 3 months following large DC and, among the patients, except two patients having early cranioplasty, all of the remaining patients developed a severe sinking skin flap. At this stage, the low CBF in the decompressed hemisphere may be responsible for such clinical symptoms [5, 6]. Therefore, cranioplasty following DC is performed not only for cosmetic reasons, but more importantly for brain protection and neurologic function improvement.

Overall, cranioplasty provides a significant benefit for patients undergoing DC. Stiver et al. [8] reported that 26% of patients ($n = 10$) who underwent DC experienced delayed weakness of contralateral upper extremities several months following DC. Also, all patients experienced markedly improved motor strength in their extremities following cranioplasty. Moreover, CTP imaging in two patients identified an improvement in CBF following cranioplasty. As mentioned earlier, with its transient increase in the decompressed hemisphere, CBF will gradually decrease to a low level beneath the sinking skin flap, which worsens clinical outcomes in those patients.

Several published studies focused on the effects of cranioplasty on CBF. However, most of them used

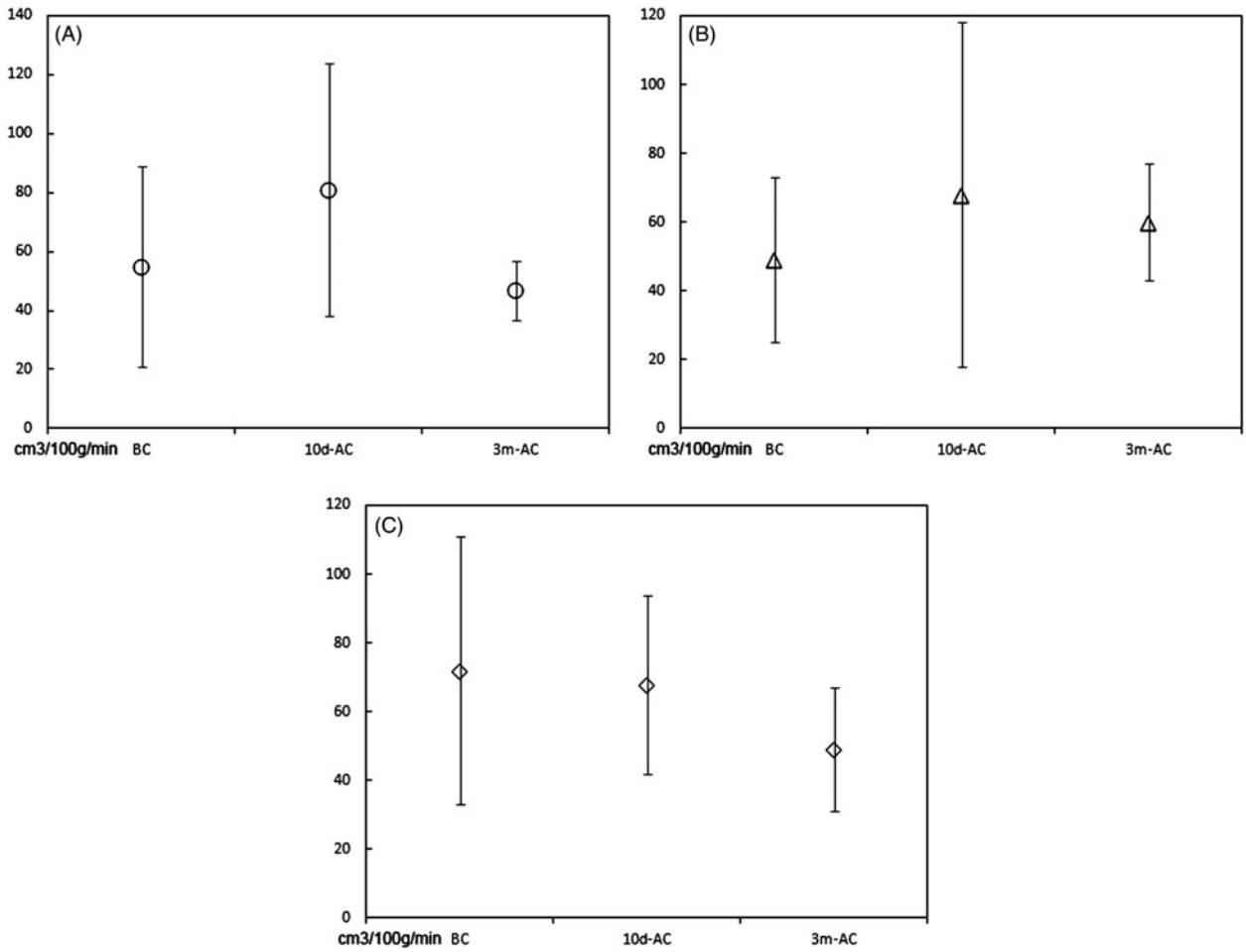

Figure 2. CBF (cerebral blood flow) changes on the hemisphere contralateral to decompressive side after the cranioplasty among patients who underwent decompressive craniecotmy for a severe head trauma (BC, before cranioplasty; 10d-AC, 10 days after cranioplasty; 3m-AC, 3 months after cranioplasty). (A) CBF changes of parietal lobe on the contralateral side. (B) CBF changes of occipital lobe on the contralateral side. (C) CBF changes of basal ganglia on the contralateral side. From these figures (A, B), the CBF changes on the contralateral hemisphere was similar with those on decompressive hemisphere except those of the basal ganglia (C).

Table III. Comparison of CBF (cerebral blood flow) between decompressive side and contralateral side before and after cranioplasty.

		Before cranioplasty			After cranioplasty		
	n	Parietal lobe	Occipital lobe	Basal ganglia	Parietal lobe	Occipital lobe	Basal ganglia
CBF of decompressive side ($cm^3/100\,g\,min^{-1}$)	9	46 ± 19	42 ± 20	59 ± 29	75 ± 38	59 ± 14	77 ± 35
CBF of contralateral side ($cm^3/100\,g\,min^{-1}$)	9	55 ± 34	49 ± 24	72 ± 39	81 ± 43	68 ± 50	68 ± 26
CBF interval ($cm^3/100\,g\,min^{-1}$)	9	8 ± 23	8 ± 27	14 ± 29	6 ± 33	9 ± 39	-9 ± 27

Two-tailed paired *t*-test, $*p < 0.05$ (comparing with CBF of decompressive side), $\#p < 0.05$ (comparing with CBF interval before cranioplasty). CBF interval, CBF interval between contralateral side and decompressive.

transcranial Doppler ultrasonography to investigate CBF regulation. Winkler et al. [11] evaluated the blood flow velocities in the middle cerebral artery (MCA) and the extracranial internal carotid artery (ICA) before and 7 days after cranioplasty by transcranial Doppler ultrasonography during postural maneouvres (supine and sitting positions) and during stimulation with 1 g acetazolamide for the interpretation of cerebrovascular reserve (CVR) capacity in 13 patients with extensive craniectomy. Their study showed that CVR capacity is increased significantly following cranioplasty. Song et al. [12] reviewed 43 patients undergoing early (<12 weeks) or late (≥12 weeks) cranioplasty following DC. The CBF velocity in the MCA ipsilateral to cranioplasty was significantly increased in both groups. However, on the side contralateral to cranioplasty, the CBF in the MCA was increased only in the early procedure group. Moreover, the increase in CBF velocity on the ipsilateral side following cranioplasty was more significant in the early procedure group. These results have been confirmed by other groups using the method of transcranial Doppler ultrasonography [13, 14].

Different from previous reports, this study evaluated CBF regulation by CTP. It found that the CBF on both the ipsilateral and contralateral sides to cranioplasty was

increased following the procedure. The difference in CBF between hemispheres ipsilateral and contralateral to surgical decompression appeared to decrease following cranioplasty. CTP imaging is a simple and accurate method to study CBF, especially for patients who experience consciousness problems following TBI. Won et al. [15] assessed CBF in 24 patients by CTP and found that the CBF of the operated hemisphere was increased from 39.1 ± 7.2 ml/100 g min^{-1} to 44.7 ± 8.9 ml/100 g min^{-1}, although this difference was not statistically significant. Sarubbo et al. [16] studied CBF in six patients by CTP before cranioplasty and at 7 days and 3 months following the procedure. Their results suggest that cortical perfusion increased shortly after cranioplasty and then progressively declined in the operated hemisphere value even lower than that prior to the procedure, but remained stable in the contralateral hemisphere following the procedure. These two studies chose the cortex beneath the cranial defect as the zone of interest for evaluating CBF. However, most of cerebral contusions occur in the temporal and frontal lobes, which are beneath the cranial defect from DC. Such cerebral contusions would be largely replaced by encephalomalacia with extremely low or even no CBF, which may affect study results. In this study, the basal ganglia, parietal lobe and occipital lobe were chosen as the zones of interest for assessing CBF. Similarly to Sarubbo et al.'s [16] study, the results showed that, after cranioplasty, the CBF on the operated hemisphere increased in the early stage but decreased 3 months post the procedure. However, in this study the CBF 3 month post-cranioplasty seemed to be still higher than that prior to cranioplasty; and the change of the CBF of the contralateral hemisphere was like that of the operated hemisphere. The results showed that cranioplasty may improve cerebral perfusion. Several published case reports involving methods of magnetic resonance imaging or CTP imaging were also in line with these results [5, 6].

After decompressive craniectomy, the brain beneath the cranial defect loses the protection of the cranium and the atmosphere could lead to a positive pressure to it directly through the flap. This pressure may lead to the decrease on the CBF. Besides, when the patient is maintained in orthostatism, the pressure would lead to the shift of brain to the skull base and may affect the blood flow of the internal carotid. The brain shift resulting from the change of position also could lead to the disorder of adjustment of CBF. The cranioplasty is able to recover the integrity of the cranium and provides protection for the brain to avoid direct pressure from the atmosphere. In the meantime, the procedure also avoids brain shift. These changes after cranioplasty may improve cerebral perfusion at last.

Cranioplasty has been recommended to be performed at least 3 months or even 6 months following DC, mainly to prevent the surgical complication of infection. However, after the acute phase of TBI, a large cranial defect can cause pathophysiological disturbances, including cerebrospinal fluid circulation disturbance and CBF dysregulation, which worsen patients' situations. It is plausible that these changes appear early following DC. Recently, increasing evidence suggests the benefits of early cranioplasty [17, 18]. Bender et al. [19] found that patients who undergo an early procedure (<86 days) have significantly better outcomes than those who undergo delayed cranioplasty (>85 days). Furthermore, Song et al. [12] reported that the increase in CBF velocity on the ipsilateral side following cranioplasty is prominent in the early surgery group on transcranial Doppler ultrasonography. These studies on changes in CBF following cranioplasty also suggest its early benefits, especially for patients who have a large cranial defect following surgical decompression. Similar results were discovered in the study introduced by Chibbaro et al. [20]. However, a well-designed study which can approve the early cranioplasty after decompressive cranioplasty is still absent at present.

Conclusion

This study prospectively investigated the changes in CBF following cranioplasty in patients who underwent DC for severe TBI and had a large cranial defect. The results indicated that, after cranioplasty, the cortical CBF was increased in both the decompressed and contralateral sides, and the cortical difference in CBF between the contralateral side and the decompressed side appeared to decrease. It seems that the CBF could increase after cranioplasty in the patients undergoing decompressive craniectomy for TBI and benefit the recovery. Further, the effects of cranioplasty time on the CBF changes after DC are on the plan.

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Declaration of interest

The authors have reported no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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