

Original Article

Clinical significance of dynamic monitoring by transcranial doppler ultrasound and intracranial pressure monitor after surgery of hypertensive intracerebral hemorrhage

Zaiming Liu, Qianxue Chen, Daofeng Tian, Long Wang, Baohui Liu, Shenqi Zhang

Department of Neurosurgery, Renmin Hospital of Wuhan University, Wuhan 430060, Hubei, China

Received March 13, 2015; Accepted June 8, 2015; Epub July 15, 2015; Published July 30, 2015

Abstract: The aim of this study was to investigate the surgical method of hypertensive intracerebral hemorrhage (HIH) and how to control the postoperative blood pressure. 96 HIH patients were performed the craniotomic hematoma dissection (CHD) and the hematoma-cavity drilling drainage (HCDD), respectively. Meanwhile, the intracranial pressure and mean arterial pressure of each patient were continuously monitored for 7 days, the postoperative 1st, 3rd, 7th and 14th-day average flow velocities and pulsatility indexes of the bilateral middle cerebral arteries were monitored. CHD exhibited the significant difference in the long-term quality of life (ADL classification 6 months later) of patients with hematoma >50 ml than HCDD; furthermore, the postoperative 1st, 3rd, 7th and 14th-day TCD parameter analysis revealed that CHD exhibited better results in relieving the intracranial pressure and improving the cerebral blood flow than HCDD, and the postoperative ICP and MAP monitoring towards all patients could effectively control the blood pressure and prevent the further bleeding. The patients with hematoma >50 ml should choose CHD, and all HIH patients should be routinely performed the ICP and MAP monitoring.

Keywords: Hypertensive intracerebral hemorrhage, intracranial pressure monitoring, transcranial doppler ultrasound

Introduction

The incidence of HIH exhibited a clear upward trend in recent years, and its mortality and morbidity were high, the mortality rate within one month of cerebral hemorrhage was 35% to 52% [1], among who about 50% died within 2 days of the onset, and only 20% survivors could take care of themselves six months later [2]. The postoperative bleeding of HIH patients was the leading cause of significantly high mortality and morbidity. There were 2 surgical treatments towards HIH, trans-cranial hole hematoma-cavity urokinase rinsing-drainage and CHD. The transcranial Doppler (TCD) could detect the basal cerebral blood flow, pulsatility index (PI) and Vm MCA, which might reflect the changes of cerebral hemodynamics. The cranial TCD detection indexes were closely related to the intracranial pressure [3]. Currently, ICP had been widely used into the treatment and res-

cue of traumatic brain injuries [4]. Olivecona [5] considered that ICP was a reliable indicator to determine the prognosis of patients with cerebral hemorrhage. Dale et al [6, 7] reported that the intracranial pressure monitoring could provide the information that could not be provided by the clinics and imaging, thus providing the objective quantitative standard indicators for the selection of surgical methods towards the HIH patients. The intracranial pressure monitor could provide the accurate basis for the reasonable and effective treatment of intracranial hypertension, and would help to determine the disease conditions and the operation time. To investigate the surgical method of hypertensive intracerebral hemorrhage (HIH) and how to control the postoperative blood pressure. In recent years, most studies suggested that the simultaneous monitoring of ICP and CPP had much more clinical significance than the simple ICP monitoring [8, 9]. Bullock's studies showed that

[10, 11] when the intracranial pressure was controlled <20 mmHg, the patients' outcomes could be greatly improved, meanwhile, he pointed out that [12] when the intracranial pressure was increased to >5.3 kPa, the intracranial hematoma might exist. According to the research we conclude that the patients with hematoma >50 ml should choose CHD, and all HIH patients should be routinely performed the ICP and MAP monitoring.

Materials and methods

General data

A total of 96 study subjects were enrolled, and divided into traditional CHD group (56 cases) and the HCDD group (40 cases) according to the treatment methods, among who 69 cases were males, and 27 cases were females, aged 35 to 86 years old (with the mean age as 60.8 ± 13.1) years old. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Wuhan University. Written informed consent was obtained from all participants. The systolic blood pressure when admission was 130~240 mmHg, with the average as (175.76 ± 30.13) mmHg, the diastolic blood pressure was 70~160 mmHg, with the average as (115.15 ± 15.12) mmHg. When admitted, 95 cases had the headache, 90 cases had the vomiting, 86 cases exhibited the hemiplegia, 8 cases had the clear consciousness, 14 cases exhibited the lethargy, 9 cases were in the blurred consciousness, 9 cases exhibited the drowsiness, 18 cases were in the light coma, 32 cases were in the moderate coma, and 6 cases were in the deep coma; 7 cases had the bleeding site under the cortex, 73 cases were in the putamen area, 6 cases were in the thalamus, 5 cases were in the cerebellum, 5 cases were in the brainstem, and 12 cases broke into the ventricle. The hematoma volume (including the upper and lower entorium), the Tada formula was used to calculate the hematoma volume (hematoma volume = $\pi/6 \times \text{length}_{\text{max}} \times \text{width}_{\text{max}} \times \text{height}_{\text{max}}$) (the patients that broke into the ventricle were not calculated the intraventricular hemorrhage volume) [13], the HCDD group was (42.8 ± 11.8) ml, the CHD group was (53.3 ± 11.2) ml. The case selection criteria: all the cases were consistent with the diagnostic criteria of brain hemorrhage developed by the 4th National

Conference of the brain blood vessels in 1995, and confirmed by CT, while the patients without temporal window of TCD examination were excluded. The patients with such clear evidence-induced hemorrhage as aneurysm, cerebral vascular malformations, AVM trauma, cancer and stroke were excluded.

Research methods

All the patients were divided into the CHD groups and the HCDD group. The HCDD group: after the CT positioning, the scalp was performed the straight incision under the local anesthesia, the skull was then drilled, which was then used for the slight enlargement of cranial bone hole (which would be conducive for the placement of ICP monitoring probe), the cerebral dura mater was then crossly cut, the hematoma cavity was then direct punctured, and placed the drainage tube after sucked 40% to 60% of hematoma, the diluted urokinase was then used to rinse the hematoma cavity 2~3 times daily with postoperative 3 days, and 3 days later, the head CT was re-checked for the withdraw of drainage tube, and the ICP monitoring was continued until the postoperative 7th day. The CHD group: the patients were performed the general anesthesia and intubation, followed by the converse scalp incision (or horseshoe scalp incision) on the frontotemporal part, after cut the dura mater, one 3~4 cm cortex incision was performed at the sulcus of superior or middle temporal gyrus, the bipolar coagulation was used for the hemostasis, then deepened 2~3 cm further and entered the hematoma cavity, one sucker was used to gently suck the blood clots, and the tumor-pliers could be used to remove the loosen clot if necessary. The drainage tube was then placed inside the hematoma cavity, and whether the bone flaps were restored should be depended on the specific situations, the postoperative continuous drainage was performed, meanwhile, the bone window-surrounding epidurum was placed the continuous ICP monitoring until the postoperative 7th day.

The intracranial pressure monitoring used the PM-9000 multifunctional intracranial pressure monitor (Shenzhen Mindray Co.), which could monitor MAP (mean arterial pressure). All the patients were performed the epidural catheterization for the 24 h continuous monitoring, and for a total of 7 days. The ICP monitoring results were shown in **Tables 1, 2**.

Clinical significance of dynamic monitoring

Table 1. ICP results of the 2 group at different postoperative times (hematoma volume 30~50 ml)

Group	N	Immediately after the surgery	1 st d	3 rd d	7 th d
CHD	30	2.02 ± 0.48	3.01 ± 0.32	3.40 ± 0.55	1.98 ± 0.72
HCDD	27	2.04 ± 0.37	2.96 ± 0.58	3.60 ± 0.78	2.03 ± 0.68

Table 2. ICP results of the 2 group at different postoperative times (hematoma volume >50 ml)

Group	N	Immediately after the surgery	1 st d	3 rd d	7 th d
CHD	26	2.02 ± 1.28	3.11 ± 1.31	3.80 ± 1.56	2.80 ± 1.64
HCDD	13	4.65 ± 1.35	4.98 ± 1.49	5.24 ± 1.84	3.79 ± 1.89

Table 3. TCD parameters of the 2 groups (hematoma volume 30~50 ml)

Group	N	Diseased side VmMCA	Healthy side VmMCA	Diseased side PI	Healthy side PI
CHD					
1 d	30	54.21 ± 7.90	59.12 ± 8.09	1.26 ± 0.13	1.16 ± 0.13
3 d	30	51.01 ± 7.87	56.98 ± 7.01	1.35 ± 0.12	1.26 ± 0.15
7 d	30	59.60 ± 8.73	64.21 ± 8.03	1.01 ± 0.15	0.62 ± 0.13
14 d	30	66.72 ± 8.07	69.07 ± 7.01	0.86 ± 0.15	0.80 ± 0.11
HCDD					
1 d	27	53.18 ± 7.63	60.09 ± 8.01	1.25 ± 0.14	1.17 ± 0.15
3 d	27	52.01 ± 7.90	57.98 ± 7.12	1.36 ± 0.13	1.25 ± 0.15
7 d	27	61.01 ± 8.70	65.21 ± 8.03	1.01 ± 0.17	0.63 ± 0.16
14 d	27	67.81 ± 7.72	70.01 ± 6.09	0.85 ± 0.13	0.79 ± 0.13

Table 4. TCD parameters of the 2 groups (hematoma volume >50 ml)

Group	N	Diseased side VmMCA	Healthy side VmMCA	Diseased side PI	Healthy side PI
CHD					
1 d	26	53.16 ± 8.01	58.66 ± 7.99	1.24 ± 0.14	1.16 ± 0.11
3 d	25	50.19 ± 8.17	57.29 ± 6.78	1.36 ± 0.11	1.24 ± 0.13
7 d	23	58.77 ± 9.12	63.77 ± 8.13	1.01 ± 0.16	0.61 ± 0.14
14 d	22	65.98 ± 8.66	68.09 ± 6.03	0.87 ± 0.13	0.83 ± 0.13
HCDD					
1 d	13	48.72 ± 6.72	53.77 ± 6.78	1.04 ± 0.15	0.52 ± 0.10
3 d	11	43.17 ± 6.16	50.22 ± 5.78	1.06 ± 0.12	0.94 ± 0.15
7 d	11	42.78 ± 7.13	50.01 ± 7.12	1.07 ± 0.19	0.93 ± 0.11
14 d	9	65.78 ± 6.67	68.11 ± 5.99	0.82 ± 0.11	0.78 ± 0.12

TCD monitoring used the EMS-9U-type monitor (Shenzhen Delicate Co.), the patients were monitored the TCD detection on the postopera-

tive 1st, 3rd, 7th and 14th day, respectively, the bilateral MCA Vm and bilateral PI values were collected. The TCD parameters of the 2 groups (hematoma volume 30~50 ml) were shown in **Table 3**, and those with hematoma volume >50 ml were shown in **Table 4**.

Efficacy evaluation, neurological deficits scoring, the short-term indicators postoperative-2-week GOS score, among which good (5 points) and mild disability (4 points) were classified as the good prognosis, severe disability (3 points), vegetal survival (2 points) and death (1 point) were classified as the poor prognosis. There were 17 and 13 cases in the CHD group with the hematoma volume as 30~50 ml, and 14 and 13 cases in the HCDD group with the hematoma volume as 30~50 ml; there were 15 and 11 cases in the CHD group with the hematoma volume >50 ml, and 4 and 9 cases in the HCDD group with the hematoma volume >50 ml. The long-term efficacy was assessed by the abilities of daily living (ADL) classification method, the situations of full recovery of daily living (ADL1), partial recovery of daily living (ADL2) and walkable under the help of someone and crutches (ADL3) after 6-month treatment were classified as the good prognosis, severely disabled-lying in bed but remained the consciousness (ADL4), vegetal survival (ADL5) and death (ADL6) were classified as the poor prognosis. There were 11 and 18 cases in the CHD group with the hematoma volume as 30~50 ml, and 13 and 15 cases in the HCDD group

with the hematoma volume as 30~50 ml; there were 17 and 10 cases in the CHD group with the hematoma volume >50 ml, and 4 and 8

cases in the HCCD group with the hematoma volume >50 ml.

Data analysis

All the data were used the SPSS13.0 statistical software for the analysis, the measurement data were expressed as $\bar{x} \pm s$, and performed the t-test or F-test, as well as ANOVA multi-sample single-factor analysis of variance and linear correlation analysis; the counting data and constituent ratio were performed the χ^2 test, with $P < 0.05$ considered as the statistical significance; the multi-factor impacts were performed the logistic regression analysis.

Results

The efficacies of two surgical methods were compared, the logistic regression analysis revealed that the gender did not significantly affect the prognosis ($P = 0.561$), while the age, GCS score, blood pressure when admitted, hematoma volume, hematoma site and surgery-starting time significantly affected the prognosis ($P = 0.019, 0.016, 0.021, 0.016, 0.007$ and 0.021 , respectively), the treatment method had the significant effects towards the prognosis ($P = 0.001$). The cumulative logistic gradual regression model was used for the screening and analysis, which excluded the impacts of age, and eventually found that the blood pressure, hematoma volume, hematoma site, GCS score, surgery-starting time and surgical method were the most important factors that would affect the prognosis. There was no significant difference in age, blood pressure, hematoma volume, hematoma site, GCS score and surgery-starting time between the 2 groups, while the two surgical methods had the significant differences in the short-term quality of life (GOS1-month later) and long-term quality of life (ADL classification 6-month later) towards the patients with hematoma volume >50 ml. Furthermore, the TCD parameters on the post-operative 1st, 3rd, 7th and 14th-day revealed that the craniotomy was significantly better in relieving the intracranial pressure, improving the cerebral blood flow and reducing the brain tissue damage than HCDD. Therefore, we believed that the patients with hematoma volume >50 ml should choose the craniotomy.

Discussion

The most important pathological changes of HIH were the hemorrhage-occupying effects-

induced mechanical damages and such secondary damages of surrounding tissues as cerebral ischemic edema [14]. Shaheen et al [15] thought that the damages of cerebral tissues caused by the cerebral ischemic edema after the cerebral hemorrhage were as much as the hematoma itself. The patients with sharp rising of intracranial pressure in a short time might also occur the acute cerebral edema induced by some reasons, and the mortality of these patients was high, and the effective appropriate treatments should be performed clinically [16]. Over the years, the surgical treatment of HIH had been widely accepted by neurosurgeons, and the surgical treatments used were also diverse, but the clinical results reported varied. The much more consistent trend was the minimal-invasive procedure, because the hematoma size were different, the bone flap craniotomy or minimal-invasive catheterization could not simply be used as the unified procedure, it would be much more objective to choose the different surgical methods according to the severity, hematoma site, hematoma volume and damaged range of brain tissues. The surgical purposes were to remove the hematoma, decrease the intracranial pressure, prevent or reduce the series of post-bleeding secondary pathological changes, and break the vicious cycle that would threaten the lives. We believed that these two surgical methods were both effective, and different patients should choose different surgical procedures. This study grouped 96 patients, the CHD group was namely the hemicraniectomy, with wider vision field, and the hemostasis under the direct vision could be much more thorough, so the opportunity of rebleeding was fewer, the hemicraniectomy could reduce the incidence of cerebral hernia and reduce the neurogenic pulmonary edema; the HCDD group, namely the CT-locating cranial drilling + urokinase dissolution, tried to exchange the maximal removal of hematoma with a smaller brain damages, thus achieving the full decompression, maximally protect the brain tissues and good postoperative recovery of neurological functions. The minimal-invasive craniotomy had smaller trauma, but had such shortcomings as difficult for the deep-site hemostasis, incomplete drainage and easy rebleeding, thus it was much more suitable for the advanced hematoma that was largely liquefied and lytic, while could not achieve the satisfactory effects towards the early hematoma which was mainly the blood

clots. We took both CHD and HCDD for the treatments, and observed the changes of post-operative ICP, bilateral MCA Vm and PI values of the patients with hematoma >50 ml, the results showed from the traditional craniotomy exhibited the significantly lower ICP values than the HCDD group from the exact time of hematoma-removal surgery, to 72th hour and one week after the surgery, the TCD results showed that the diseased-side MCA Vm was significantly increased, while the PI value was decreased significantly from the exact time of hematoma-removal surgery, to 72th hour and one week after the surgery, which of course would help to reduce the brain edema and brain tissue damages, improve cerebral circulation, and create favorable conditions for the early recovery of compressed brain tissues. We believed CHD was better in relieving the intracranial pressure, reducing the brain tissue damages than HCDD. We found no significant difference existed between CHD and HCDD towards the prognosis of patients with hematoma volume within 30~50 ml.

The detection of cerebral blood flow velocity by TCD could basically reflect the speed of cerebral blood flow, PI mainly reflected the changes of cerebral vascular resistance, and the velocity of systolic peak (Vm) was mainly affected by the systolic blood pressure [17, 18], CVR was also decided by the cerebrovascular diameter and ICP. Therefore, these parameters might reflect the changes of cerebral hemodynamics [3]. So far, the determinations of ICP all used the invasive methods, the invasive intracranial pressure monitoring technologies and methods were plenty, the intraventricular monitoring and the subdural monitoring were classified according to the different anatomical locations of intracranial-pressure monitoring probes [19, 20]. The traditional medicine thought that because the cranial cavity was a whole, when the intracranial occupying effect was produced, the cranial contents would be changed, so that the pressures inside the cranial cavity were all increased, and almost the same throughout the intracranium [21]. We used the epidural pressure (EDP) method. CPP was the result of systemic mean arterial pressure (MAP) subtracted by ICP, the cerebral blood flow (CBF) was positively proportional to CPP, while negatively proportional to the cerebral vascular resistance (CVR), i.e. $CBF = (MAP-ICP)/CVR$.

When the cerebrovascular autoregulation functions existed, ICP rose, CPP decreased, the cerebral small arteries expanded, CVR would decrease to maintain the constant of cerebral blood supply, and then the diastolic blood pressure (DBP) would be decreased significantly than the systolic blood pressure (SBP), so the pulse pressure difference was increased, and PI that reflected the pulse pressure difference was increased. TCD made it possible for the bedside noninvasive monitoring of ICP and cerebral perfusion pressure. The occupying effects of intracranial hematoma might lead to the continuous increasing of ICP and the reduction of blood flow. In the present study, the asymmetry of bilateral MCA flow velocities among the 96 HIH patients was decreased, and that in the healthy side was much more significant than the diseased side, consistent with Egido [22], namely the MCA flow velocity was considered to be closely associated with the hematoma volume, thus it could reflect the asymmetry of blood flows in both hemispheres. As for the patients with hemorrhage volume >50 ml, the bilateral MCA flow velocities of the HCDD group exhibited the declining changes within 1 week, while began to increase in the 2nd week, while the PI value was increased continuously in the 1st week, and started to fall in the 2nd week. Compared with the HCDD group, the MCA flow velocity of the CHD group began to increase on the 7th day, while PI was decreased, and there existed the significant difference between the 2 groups on the 14th day. This was because the hematoma-caused secondary cerebral edema of the CHD group was significantly reduced on the 7th day, and the occupying effects were basically eliminated on the 14th day, the gradual eliminations of cerebral edema and metabolite accumulation made ICP approach the normal.

In recent years, most studies suggested that the simultaneous monitoring of ICP and CPP had much more clinical significance than the simple ICP monitoring. Menzel et al [23] considered that the patients with CPP less than 60 mmHg would have poor prognosis, when CPP fell below 60 mmHg, the oxidation of brain parenchyma would decrease, which was the indicator to reflect the balance of cerebral oxygen supply and demand, its decreasing showed the imbalance of brain oxygen supply. Meanwhile, Reinert et al [24] thought that the

Clinical significance of dynamic monitoring

excessively high CPP would make the brain oxygen level lower than that when CPP was within 70~90 mmHg. So, most scholars thought that the CPP level should be maintained at 70~90 mmHg. Because of the relationships such as $CPP = MAP - ICP$ and others, and CPP was controlled within 70~90 mmHg, ICP was controlled within 6~15 mmHg, the MAP level should then be controlled within 76~105 mmHg. Bullock's studies showed that when the intracranial pressure was controlled <20 mmHg, the patients' outcomes could be greatly improved, meanwhile, he pointed out that when the intracranial pressure was increased to >5.3 kPa, the intracranial hematoma might exist. We firstly guaranteed that ICP was maintained within 6~15 mmHg, then controlled MAP within 76~105 mmHg according to the ICP monitoring results, so that we not only could ensure the adequate cerebral perfusion, reduce the further damages of nerve cells inside the perihematomal penumbra caused by the ischemia and hypoxia, but also would not cause the bleeding because of the excessively high CPP, the specific method was to use the infusion of 20% mannitol and sodium nitroprusside for the maintenance. Therefore, this study believed that it was necessary for HIH patients to dynamically monitor their ICP and MAP, because this monitoring could accurately maintain the intracranial pressure within the normal range, reduce the brain damages caused by the postoperative cerebral edema-induced high intracranial pressure, and could promptly adjust the blood pressure, thus ensuring that cerebral perfusion on one hand, and preventing the high blood pressure-caused rebleeding on the other hand, our group performed the ICP and MAP monitoring towards all the patients, controlled MAP within 70~105 mmHg, and among the 96 cases, 8 patients occurred the rebleeding, among who the blood pressures of 3 cases were well controlled, and the related-reason analysis revealed that, among the 2 cases of the CHD group, 1 case was related with the non-thorough hemostasis, and the other case was related with the coagulation defects; and 1 case of the HCDD group was related with the inaccurate puncture site; the rest 5 cases were all refractory hypertension, so that the blood pressure could not be controlled within the above range.

The HIH patients with hematoma volume >50 ml should choose the craniotomy, and the post-

operative blood pressure should be controlled at the range of MAP 70~105 mmHg.

Disclosure of conflict of interest

None.

Address correspondence to: Qianxue Chen, Department of Neurosurgery, Renmin Hospital of Wuhan University, Wuhan 430060, Hubei, China. Tel: +86 13607141618; E-mail: qianxuechen@163.com

References

- [1] Soustiel JF, Svirgi GE, Mahamid E, Shik V, Abeshaus S and Zaaroor M. Cerebral blood flow and metabolism following decompressive craniectomy for control of increased intracranial pressure. *Neurosurgery* 2010; 67: 65-72.
- [2] Asgari S and Vespa P. Lack of consistent intracranial pressure pulse morphological changes during episodes of microdialysis lactate/pyruvate ratio increase. *Physiol Meas* 2011; 32: 1639-1651.
- [3] Edouard AR, Vanhille E, Le Moigno S, Benhamou D and Mazoit JX. Non-invasive assessment of cerebral perfusion pressure in brain injured patients with moderate intracranial hypertension. *Br J Anaesth* 2005; 94: 216-221.
- [4] Lavinio A and Menon DK. Intracranial pressure: why we monitor it, how to monitor it, what to do with the number and what's the future? *Curr Opin Anaesthesiol* 2011; 24: 117-123.
- [5] Olivecrona M. Use of the CRASH study prognosis calculator in patients with severe traumatic brain injury treated with an intracranial pressure-targeted therapy. *J Clin Neurosci* 2013; 20: 996-1001.
- [6] Dale CH and Jamshid G. Marked improvement in adherence to traumatic brain injury guidelines in United States trauma centers. *J Trauma* 2007; 63: 841-848.
- [7] Romner B and Grande PO. Traumatic brain injury: Intracranial pressure monitoring in traumatic brain injury. *Nat Rev Neurol* 2013; 9: 185-186.
- [8] Junl N, Morris GF, Marshall SB and Marshall LF. Intracranial hypertension and cerebral perfusion pressure: influence on neurological deterioration at long-term outcome in severe head injury. *Neurosurgery* 2000; 92: 1-6.
- [9] Sarrafzadeh AS, Peltonen EE, Kaisers U, Küchler I, Lanksch WR and Unterberg AW. Secondary insults in severe head injury do multiply injured patients do worse. *Crit Care Med* 2001; 29: 1116-1123.
- [10] Bullock MR and Povlishock JT. Guidelines for the management of severe traumatic brain in-

Clinical significance of dynamic monitoring

- jury. Editor's Commentary. *J Neurotrauma* 2008; 25: 276-278.
- [11] Adelson P, Bratton S, Carney N, Chesnut RM, du Coudray HE, Goldstein B, Kochanek PM, Miller HC, Partington MD, Selden NR, Warden CR, Wright DW; American Association for Surgery of Trauma; Child Neurology Society; International Society for Pediatric Neurosurgery; International Trauma Anesthesia and Critical Care Society; Society of Critical Care Medicine; World Federation of Pediatric Intensive and Critical Care Societies. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. *Ped Crit Care Med* 2003; 4: S38-S39.
- [12] Maas AI and Schouten JW, Stocchetti N, Bullock R, Ghajar J. Questioning the value of intracranial pressure (ICP) monitoring in patients with brain injuries. *J Trauma* 2008; 65: 966-967.
- [13] Beseoglu K, Etminkan N, Turowski B, Steiger HJ and Hänggi D. The extent of the perihemorrhagic perfusion zone correlates with hematoma volume in patients with lobar intracerebral hemorrhage. *Neuroradiology* 2014; 29: 987-993.
- [14] Zhang X, Zhang Z, Yin X, Zhang K, Cai H and Ling F. Exploring the optimal operation time for patients with hypertensive intracerebral hemorrhage: tracking the expression and progress of cell apoptosis of prehematomal brain tissues. *Chin Med J* 2010; 123: 1246-1250.
- [15] Lakhan SE, Kirchgessner A, Tepper D and Leonard A. Matrix metalloproteinases and blood-brain barrier disruption in acute ischemic stroke. *Front Neurol* 2013; 4: 32.
- [16] Ropper AE and Chi JH. Treatment of traumatic brain injury without direct intracranial pressure monitoring. *Neurosurgery* 2013; 72: N19-N20.
- [17] Kiphuth IC, Huttner HB, Drfler A, Schwab S and Köhrmann M. Doppler pulsatility index in spontaneous intracerebral hemorrhage. *Eur Neurol* 2013; 70: 133-138.
- [18] Thompson BB, Wendell LC, Potter NS, Fehnel C, Wilterdink J, Silver B and Furie K. The use of transcranial doppler ultrasound in confirming brain death in the setting of skull defects and extraventricular drains. *Neurocrit Care* 2014; 10: 89-92.
- [19] Speck V, Staykov D, Huttner HB, Sauer R, Schwab S and Bardutzky J. Lumbar catheter for monitoring of intracranial pressure in patients with post-hemorrhagic communicating hydrocephalus. *Neurocrit Care* 2011; 14: 208-215.
- [20] Staykov D, Speck V, Volbers B, Wagner I, Saake M, Doerfler A, Schwab S and Bardutzky J. Early recognition of lumbar overdrainage by lumboventricular pressure gradient. *Neurosurgery* 2011; 68: 1187-1191.
- [21] Thompson KM, Gerlachs Y, Jom HK, Larson JM, Brott TG and Files JA. Advances in the care of patients with intracerebral hemorrhage. *Mayo Clin Proc* 2007; 82: 987-996.
- [22] Egido JA, Alonso de Leciñana M, Díaz F, Fernández C, Carneado J, González JL. Blood flow in patients with intracerebral hemorrhage. Does there exist the associated hypoperfusion? A transcranial Doppler study. *Rev Neurol* 2001; 31: 179-183.
- [23] Menzel M, Soukup J, Henze D, Clausen T, Marx T, Hillman A, Miko I, Grond S and Rieger A. Brain tissue oxygen monitoring for assessment of auto regulation: preliminary results suggest a new hypothesis. *Neurosurg Anesthesiol* 2003; 15: 33-41.
- [24] Reinert M, Barth A, Rothen HV, Schaller B, Takala J and Seiler RW. Effects of cerebral perfusion pressure and increased fraction of inspired oxygen on brain tissue oxygen, lactate and glucose in patients with severe head injury. *Acta Neurochir* 2003; 145: 341-350.