

# Prediction of Cerebral Hyperperfusion Syndrome with Velocity Blood Pressure Index

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## Abstract

**Background:** Cerebral hyperperfusion syndrome is an important complication of carotid endarterectomy (CEA). An >100% increase in middle cerebral artery velocity (MCAV) after CEA is used to predict the cerebral hyperperfusion syndrome (CHS) development, but the accuracy is limited. The increase in blood pressure (BP) after surgery is a risk factor of CHS, but no study uses it to predict CHS. This study was to create a more precise parameter for prediction of CHS by combined the increase of MCAV and BP after CEA.

**Methods:** Systolic MCAV measured by transcranial Doppler and systematic BP were recorded preoperatively; 30 min postoperatively. The new parameter velocity BP index (VBI) was calculated from the postoperative increase ratios of MCAV and BP. The prediction powers of VBI and the increase ratio of MCAV (velocity ratio [VR]) were compared for predicting CHS occurrence.

**Results:** Totally, 6/185 cases suffered CHS. The best-fit cut-off point of 2.0 for VBI was identified, which had 83.3% sensitivity, 98.3% specificity, 62.5% positive predictive value and 99.4% negative predictive value for CHS development. This result is significantly better than VR (33.3%, 97.2%, 28.6% and 97.8%). The area under the curve (AUC) of receiver operating characteristic:  $AUC_{VBI} = 0.981$ , 95% confidence interval [CI] 0.949–0.995;  $AUC_{VR} = 0.935$ , 95% CI 0.890–0.966,  $P = 0.02$ .

**Conclusions:** The new parameter VBI can more accurately predict patients at risk of CHS after CEA. This observation needs to be validated by larger studies.

**Key words:** Blood Pressure; Carotid Endarterectomy; Cerebral Hyperperfusion Syndrome; Prediction; Transcranial Doppler

## INTRODUCTION

Carotid endarterectomy (CEA) has been demonstrated as the most effective treatment for patients with symptomatic high-grade stenosis of the internal carotid artery (ICA). Operation is superior to another treatment for patients that are younger than 75 years old with an asymptomatic high-grade stenosis of the ICA. However, the benefits of CEA are limited by various operation-related complications, which lead to perioperative death or stroke in up to 5% of patients.<sup>[1,2]</sup>

Cerebral hyperperfusion syndrome (CHS) is a rare but important perioperative complication of CEA. Impaired cerebral autoregulation and the postoperatively elevated systemic blood pressure (BP) are the two interlinked and synergistic mechanisms causing CHS.<sup>[3]</sup>

Severe ipsilateral headache, deterioration of consciousness, confusion, seizures and focal neurological deficits are the most common symptoms of CHS. Most symptoms are transient and mild, however, if CHS cannot be recognized

and treated adequately, catastrophic events such as intracerebral hemorrhage (ICH) can occur. The reported incidence of CHS after CEA in most studies is less than 3%, the majority of CHS cases develop within the first 7 days after the operation.<sup>[4]</sup> Several clinical risk factors of CHS are taken into consideration, including poorly-controlled preoperative and postoperative hypertension, advanced age, bilateral ICA stenosis, and a previous neurological ischemic event.<sup>[3,5]</sup> If CHS can be predicted early, stricter management can be implemented earlier during the perioperative period to prevent the catastrophic results of CHS.

Transcranial Doppler sonography (TCD) has been used to predict CHS development. The criterion of a  $\geq 100\%$  increase in the middle cerebral artery velocity (MCAV) after de-clamping/postoperation compared with the preoperative baseline value is widely used clinically to identify high-risk patients for the development of CHS.<sup>[6-11]</sup> However, this method has low accuracy.

Postoperatively elevated BP is closely related to CHS because cerebral perfusion pressure is directly related to mean arterial pressure. Postoperative hypertension usually occurred in the early stage after surgery, and the incidence

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of severe hypertension was significantly higher in CHS cases than in non-CHS control groups.<sup>[12]</sup> Hypertension usually peaks in the first few hours after surgery and is related to impaired baroreceptor function.<sup>[13]</sup> According to these, the increase in BP immediately after surgery is a risk factor of CHS, and which can be used to predict the occurrence of CHS.

Therefore, we attempted to combine the two risk factors, the increase in MCAV and the increase in BP after CEA, to create a more precise parameter to predict the patients at high risk of CHS.

## METHODS

A prospective study was held at the vascular surgery center of Peking Union Medical College Hospital (PUMCH). The study was approved by the ethics committee of PUMCH. All participants provided their written informed consent to participate in this study.

### Patients

Patients included were those who met the following criteria:

1. Recruited for CEA in our center from October 2010 to January 2013
2. Had a middle-grade symptomatic carotid stenosis (more than 50%) or a high-grade asymptomatic carotid stenosis (more than 70%)
3. Underwent CEA 30 days after his/her last ischemic cerebrovascular event in the case of symptomatic carotid stenosis
4. Signed the ethical information consent form
5. Underwent pre-and post-operative TCD monitoring.

Patients excluded from database met the following criteria:

1. Underwent emergency CEA
2. No temporal windows to measure the MCAV by TCD
3. Stenosis caused by nonatherosclerotic diseases
4. Refusal to sign the ethical information consent form.

Patients were divided into two groups: Patients in whom CHS occurred (CHS group) and patients in whom CHS did not occur (non-CHS group).

### Carotid endarterectomy

All the patients were treated with antiplatelet and lipid-lowering agents for at least 7 days before the CEA. General anesthesia was performed, and all patients received the same anesthetic drugs. CEA was performed by experienced vascular surgeons.

### Definition of cerebral hyperperfusion syndrome

Cerebral hyperperfusion syndrome was diagnosed as follow: (1) Patient developed ipsilateral severe headache, seizures, confusion, focal neurological deficits after surgery; (2) presence of normal carotid anatomy on color Duplex ultrasound, the absence of focal middle cerebral

artery territory infarction on computed tomography (CT) scanning or magnetic resonance imaging (MRI) results; (3) an independent neurologist identified the symptoms were not secondary to cerebral ischemia according to the clinical presence and the imaging results.<sup>[4,14]</sup>

### Management protocols of perioperative blood pressure

Generally, we did not change the long-term BP control strategy of hypertensive patents. Anti-hypertensive agents at the morning of the surgery were administrated. But if the patient's hospitalized systolic BP is more than 180 mmHg or the anesthesiologist thought the patient's BP is too high and too dangerous to accept the surgery, the surgery would be delayed until it was adequately controlled. The systolic arterial pressure was maintained from 0 to + 20% of the preoperative baseline value in the clamping phase.

Postoperative hypertension was defined as an absolute high BP threshold (BP >160 mmHg systolic) or as a relatively high BP (20% above the preoperative baseline BP). All patients with postoperative hypertension were treated with intravenous nicardipine (first choice) or sodium nitroprusside (second choice). Intravenous anti-hypertensive agents were tapered as soon as possible when the BP was within the required limit; administration of an oral beta-blocker (labetalol or metoprolol) was initiated as an extended treatment.<sup>[13]</sup>

All the BP were measured by the noninvasively BP meter.

### Timeframes

- Preoperation: The MCAV ( $V_{pre}$ ) and BP<sub>pre</sub> were registered 10 min before anesthetic admitted.
- Postoperation: The postoperative MCAV ( $V_{post}$ ) and BP<sub>post</sub> was measured at the end of the CEA (30 min after extubation).

### Transcranial Doppler sonography method

For the TCD measure and registration, a pulsed Doppler transducer gated at a focal depth of 45–60 mm was placed over the temporal bone to insonate the main stem of the ipsilateral MCA; the TCD transducer was fixed with a head frame, and the peak systolic MCAV was recorded continuously. The indicated data points to gather the values were described above.

### Parameters to predict cerebral hyperperfusion syndrome were defined and calculated as follows

- Velocity ratio (VR) (postoperative increase ratio of MCAV) =  $V_{post}/V_{pre}$
- Blood pressure ratio (BPR) (postoperative increase ratio of BP) =  $BP_{post}/BP_{pre}$
- Velocity BP index (VBI) = BPR × VR.

### Statistical analysis

Patients were classified according to the occurrence or absence of CHS. The sensitivities, specificities, positive predictive values (PPV) and negative predictive values (NPV) of VR and VBI were calculated. Differences

in hemodynamic parameters between the CHS and non-CHS groups were compared using the Chi-square test or Fisher's exact test for categorical variables and the Mann-Whitney U-test for continuous variables, as appropriate.

For the assessment of the accuracy of each parameter in discriminating CHS from non-CHS patients, we performed a receiver operating characteristic (ROC) analysis. For the ROC analysis, we used MedCalc version 17.0.0 (MedCalc Software, Mariakerke, Belgium). Other statistical analyses were performed using the Statistical Package for Social Sciences version 13.0 (SPSS Inc., USA). A confidence level of less than 5% ( $P < 0.05$ ) was considered significant.

## RESULTS

### Patient characteristics

We recruited 185 cases (148 men, 37 women; median age 66 years, interquartile range [IQR] 59–73 years) of patients that underwent CEA from October 2010 to January 2013. There was a hypertension history for 79.5% of the cases (147/185). A past medical history of stroke or TIA was present for 70.8% of the cases (131/185), among which 87 cases had a recent TIA attack and 31 cases had a recent stroke attack (within 180 days). An ipsilateral stenosis with  $\geq 70\%$  ICA had occurred for 79.5% of the cases (147/185). A contralateral stenosis with  $\geq 70\%$  ICA had occurred for 16.8% of the cases (31/130). A bilateral stenosis with  $\geq 70\%$  ICAs had occurred for 15.7% of the cases (29/185).

After stratification according to the occurrence of CHS, the patients' characteristic parameters were compared. The two groups were similar with respect to age, sex, hypertension, hypercholesterolemia, diabetes, smoking history, coronary artery disease history, past TIA or stroke history, recent TIA or stroke occurrence (within 180 days) and the stenosis degree of ipsilateral ICA. The contralateral stenosis degree  $\geq 70\%$  in the two groups are similar, but there is a higher trend in the CHS group because the  $P$  value is 0.06 [Table 1].

### Clinical outcome

Six patients (6/185, 3.2%) developed CHS. Ipsilateral severe headache presented in all 6 cases of CHS. Confusion presented in two cases, motor disturbances presented in 3 cases, and heteroptics occurred in 1 case. After symptom occurrence, all the patients underwent a brain CT test to exclude ICH. MRI-diffusion-weighted imaging and perfusion-weighted imaging were also used to exclude the ischemic lesions causing the symptom. Finally, the CHS diagnosis was given by an independent neurologist according to the symptoms, physical examinations, and imaging results. All patients fully recovered following the administration of intravenous antihypertensive medicines and glycerin fructose. One CHS occurred on the 1<sup>st</sup> day after the surgery, and the other 5 CHS cases occurred from the 3<sup>rd</sup> to 7<sup>th</sup> day after the surgery.

In the CHS group, the median BP on the occurrence day of CHS was 161 mmHg, with an IQR from 148.75 to 165 mmHg. All postoperative hypertension temporally preceded CHS.

There are 47 patients (47/185, 25.4%) complained of different levels of headache. Most of them recovered after observation or symptomatic treatment. One patient occurred motor disturbance were diagnosed TIA by the neurologist according to the MRI. No stroke patient.

### Hemodynamic parameters

Preoperative BP in CHS group and non-CHS group has no difference. Median BP<sub>pre</sub> in the CHS group is 110 mmHg (IQR from 99 to 133 mmHg), which in the non-CHS group is 117 mmHg (IQR from 106 to 130 mmHg),  $P = 0.436$ . Postoperative BP in CHS group is higher than which in non-CHS group. Median BP<sub>post</sub> in the CHS group is 130 mmHg (IQR from 120 to 149 mmHg), which in the non-CHS group is 118 mmHg (IQR from 110 to 127 mmHg),  $P = 0.022$ .

A more obvious increasing BP ratio (BPR) after the surgery was noticed in CHS group than which in non-CHS

**Table 1: Patient characteristics**

Characteristics	CHS* group (n = 6)	Non-CHS* group (n = 179)	P
Male, n (%)	5 (88.3)	143 (79.9)	1
Age, median (IQR), years	72.5 (64–76.5)	66 (59–73)	0.84
Hypertension, n (%)	4 (66.7)	143 (79.1)	0.84
Diabetes, n (%)	1 (16.7)	53 (29.6)	0.67
Hypercholesterolemia, n (%)	3 (50)	63 (35.2)	0.66
Smoking, number (%)	3 (50)	89 (49.7)	1
Coronary artery disease history, n (%)	2 (33.3)	54 (30.2)	1
Stroke history, n (%)	4 (66.7)	127 (70.9)	1
Recent TIA <sup>†</sup> , n (%)	3 (33.3)	84 (46.9)	1
Recent stroke <sup>‡</sup> , n (%)	2 (33.3)	29 (16.2)	0.26
Degree of stenosis (50%–69%) (ipsilateral), n (%)	0 (0)	38 (21.2)	0.35
Degree of stenosis (70%–100%) (ipsilateral), n (%)	6 (100)	141 (78.8)	0.35
Degree of stenosis (70%–100%) (contralateral), n (%)	3 (50)	28 (15.6)	0.06

\*CHS: Cerebral hyperperfusion syndrome; <sup>†</sup>Recent TIA or recent stroke: Patients had a stroke or TIA attack within 180 days; IQR: Interquartile range; TIA: Transient ischemic attack.

group. The median BPR in CHS group is 1.28 (IQR from 1.04 to 1.34), and which in non-CHS group is 1.03 (IQR from 0.92 to 1.12),  $P = 0.014$  [Figure 1].

BPR was also higher in the majority of postoperative hypertension cases. The median BPR of postoperative hypertension patients is 1.18 (IQR from 1.08 to 1.24), and other patients' median BPR is 1.0 (IQR from 0.91–1.09),  $P < 0.001$ .

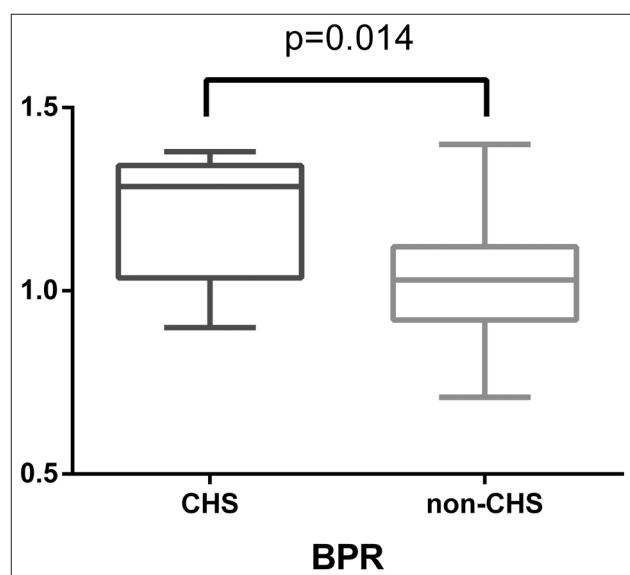
Preoperative velocity in CHS group is lower than which in non-CHS group. Median  $V_{pre}$  in the CHS group is 34.5 cm/s (IQR from 28.5 to 58.75 cm/s), which in the non-CHS group is 68 cm/s (IQR from 50 to 90 cm/s),  $P = 0.007$ . Postoperative velocity in CHS group and in non-CHS group has no difference. Median  $V_{post}$  in the CHS group is 75.5 cm/s (IQR from 50.5 to 117.75 cm/s), which in the non-CHS group is 84 cm/s (IQR from 65 to 105 cm/s),  $P = 0.645$ .

Increasing ratio of velocity after the surgery (VR) was higher in CHS group, median VR is 1.84 (IQR from 1.70 to 2.39) in CHS group, and which is 1.22 (IQR from 1.05 to 1.46) in non-CHS group,  $P < 0.001$  [Figure 2].

We also calculated the VBI values in two groups. VBI in CHS group is significantly higher than which in non-CHS group. The median VBI in CHS group is 2.25 (IQR from 2.14 to 2.60), the median VBI in non-CHS group is 1.30 (IQR from 1.06 to 1.55),  $P < 0.001$  [Figure 3].

### Prediction power

Figure 4 shows the ROC plots, the area under the curves (AUCs), and the 95% confidence intervals (95% CI) of all investigated parameters. For each parameter, the AUCs



**Figure 1:** Blood pressure ratio (BPR) in cerebral hyperperfusion syndrome (CHS) and non-CHS groups. It shows that blood pressure increased higher after surgery in CHS group than in non-CHS group. The median BPR in CHS group is 1.28 (interquartile range [IQR] from 1.04 to 1.34), and in non-CHS group is 1.03 (IQR from 0.92 to 1.12),  $P = 0.014$ .

and the corresponding 95% CI were greater than 0.5. For VBI, the AUC was also significantly higher than the AUC of VR ( $AUC_{VBI} = 0.981$ , 95% CI 0.949–0.995;  $AUC_{VR} = 0.935$ , 95% CI 0.890–0.966),  $P = 0.02$ .

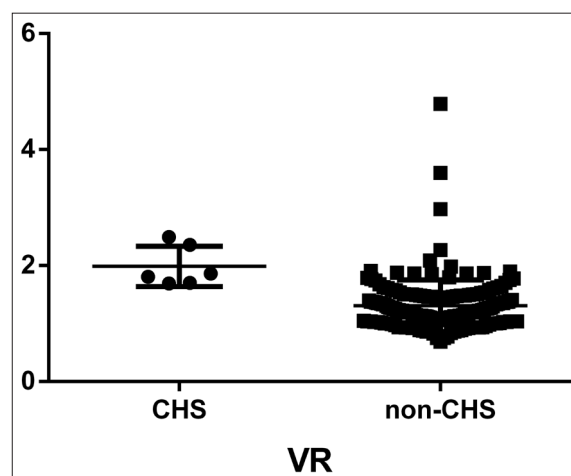
With respect to the VBI cut-off values, 5/8 patients with a VBI  $\geq 2$  developed CHS, this results in 83.3% sensitivity, 98.3% specificity, 62.5% PPV and 99.4% NPV. Other cut-off values of VBI are worse than VBI  $\geq 2$  in predicting CHS development. So the best cut-off value of VBI was identified as 2.

According to previous publications, we also chose 2 as the cut-off value for VR (which means a 100% increase of MCAV compared with the preoperative value). Seven patients with VR  $\geq 2$ , only two of them developed CHS, which has 33.3% sensitivity, 97.2% specificity, 28.6% PPV and 97.8% NPV, respectively.

Table 2 shows the detailed cut-off values of VBI and VR, and the relative sensitivities, specificities, and PPV and NPVs for predicting CHS development.

## DISCUSSION

This study was designed to identify the power of VBI, which combining BP and velocity changes in the perioperative phase, in predicting CHS development. The results demonstrate that when we chose 2 as the best cut-off value of VBI, which has a sensitivity of 83.3%, PPV of 62.5%. So the VBI has a great power to predict the development of CHS. Due to the catastrophic result of CHS, an ideal predictive parameter should include as many positive patients as possible, and the accuracy must be high, so the sensitivity and PPV are very important. According to this parameter, the majority of CHS patients can obtain more rigorous and earlier BP monitoring and control, and no patient developed ICH. Meanwhile, the specificity and

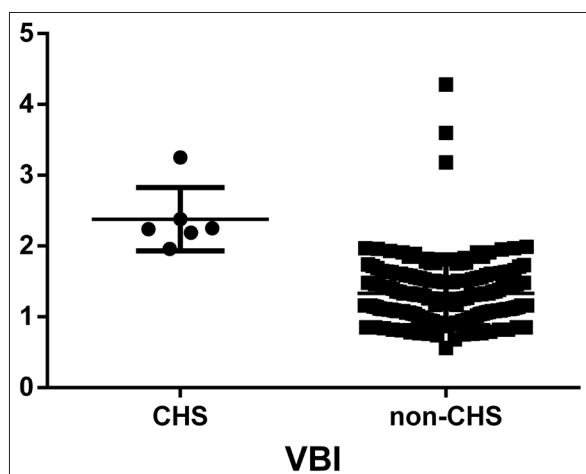


**Figure 2:** Velocity ratio (VR) in cerebral hyperperfusion syndrome (CHS) and non-CHS groups. It shows that velocity also increased higher after surgery in CHS group than which in non-CHS group. The median VR in CHS group is 1.84 (interquartile range [IQR] from 1.70 to 2.39), and which in non-CHS group is 1.22 (IQR from 1.05 to 1.46),  $P < 0.001$ .

**Table 2: Cut-off values of VBI and VR**

Values	Best cut-off (%)	Sensitivity (%)	Specificity (%)	PPV <sup>†</sup> (%)	NPV <sup>‡</sup> (%)
VBI <sup>§</sup>	2.0*	83.3	98.3	62.5	99.4
	1.9	100	93.3	33.3	100
VR <sup>  </sup>	2.0	33.3	97.2	28.6	97.8

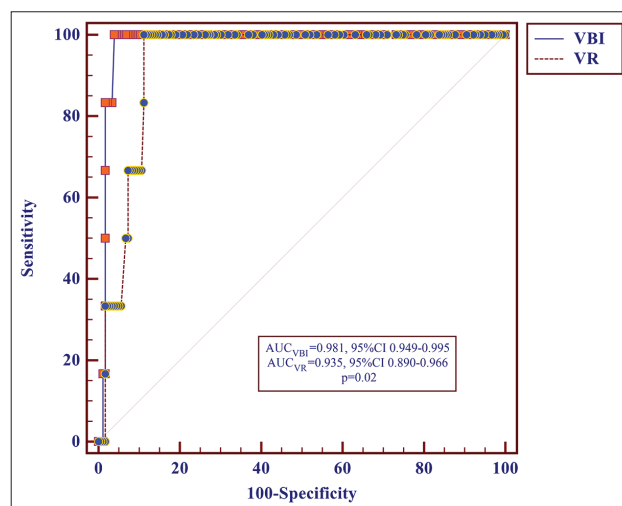
\*Best cut-off value; <sup>†</sup>PPV: Positive predictive value; <sup>‡</sup>NPV: Negative predictive value; <sup>§</sup>VBI: Velocity blood pressure index = BPR×VR; <sup>||</sup>VR: Postoperative increase of middle cerebral artery velocity =  $V_{\text{post}}/V_{\text{pre}}$ . BPR: BP ratio; BP: Blood pressure; VR: Velocity ratio.



**Figure 3:** Velocity blood pressure index (VBI) in cerebral hyperperfusion syndrome (CHS) and non-CHS groups. It shows that VBI value can distinguish CHS patients well. The median VBI in CHS group is 2.25 (interquartile range [IQR] from 2.14 to 2.60), the median VBI in non-CHS group is 1.30 (IQR from 1.06 to 1.55),  $P < 0.001$ . In 8 patients with  $VBI \geq 2$ , 5 patients developed CHS, only 1 patient's VBI value is less than 2. VR: Velocity ratio.

NPV are within high levels; almost no patient will accept the unnecessary treatment of intravenous antihypertensive medication.

The pathophysiological mechanisms of CHS have been identified as impaired cerebral autoregulation combining with postoperative hypertension. These two interactive mechanisms lead to the abnormal increase in cerebral blood flow. TCD can reflect the actual volume flow by measuring the cerebral middle artery velocity. So there are many clinicians use the increased velocity measured by TCD to predict CHS occurrence.<sup>[6,8-10,11,15]</sup> Traditionally, for intra-operative TCD monitoring during CEA under general anesthesia, a  $\geq 100\%$  increase of the velocity immediately after de-clamp is the most widely used parameter for the prediction of CHS.<sup>[6,8,9]</sup> However, the instability of the systemic BP at that time and the operative stimulation of the carotid sinus interfere with the predictive power of this method. Therefore, most studies did not acquire favorable results. Ogasawara *et al.* and Jansen *et al.* found that a  $\geq 100\%$  increase in the velocity at the end of CEA compared to the preclamp was highly suggestive of hyperperfusion but was poor at predicting CHS.<sup>[10,15]</sup> Pennekamp *et al.* introduced an additional TCD measurement at 2 h after the surgery. They used a postoperative mean MCAV increase of  $\geq 100\%$  as the cut-off value. The PPV of this predictor is 41%, which is better than the result of the intra-operative mean MCAV



**Figure 4:** Receiver operating characteristic curve of velocity ratio (VR) and velocity blood pressure index (VBI). It shows that the area under the curve (AUC) of VBI is higher than the AUC of VR (AUC<sub>VBI</sub> = 0.981, 95% confidence interval [CI] 0.949–0.995; AUC<sub>VR</sub> = 0.935, 95% CI 0.890–0.966,  $P = 0.02$ ). VSI: Velocity systolic blood pressure index.

increase; however, the sensitivity of this predictor was only 28%, which limited its use.<sup>[11]</sup>

For most patients, there are only moderate asymptomatic increases in ipsilateral cerebral blood flow (20%–40% over baseline) immediately after CEA,<sup>[18,16,17]</sup> even in hyperperfusion patients (most commonly defined as  $\geq 100\%$  increase over baseline) the MCAV requires 3–4 days to reach its maximum.<sup>[8,18,19]</sup> Meanwhile, the cerebral artery autoregulation of CHS patients is a gradually deteriorating process. So at the end of the operation, many CHS patients' MCAV did not have a  $\geq 100\%$  increase over baseline. Newman *et al.* included more than 1000 CEA patients and found that postoperatively a  $>100\%$  increase in mean and peak MCAV only had a PPV of 6.3% and 2.7% respectively.<sup>[14]</sup> Therefore, the method that merely used the MCAV increase to predict the occurrence of CHS is associated with a high occurrence of false negative and false positive results.

The role of BP increase in predicting the occurrence of CHS was ignored. Postoperative hypertension is itself a strong contributor of cerebral perfusion pressure increase because cerebral perfusion pressure is dependent on mean arterial pressure.<sup>[20]</sup> Postoperative hardly controlled hypertension coupled with impaired cerebral autoregulation may result in hyperperfusion of an area of the brain that was previously chronically ischemic.<sup>[21,22]</sup> The study has demonstrated

surgical destruction of the ipsilateral carotid baroreflex mechanisms during CEA. Denervation of baroreceptors leads to an inability of the receptors to alter systemic BP in response to various physiological stimuli; hence, hypertension following CEA coupled with baroreceptor reflex breakdown can result in cerebral hyperperfusion.<sup>[23]</sup>

According to the systematic review published in *Eur J Vasc Endovasc Surg* in 2011, postoperational hypertension temporally preceded CHS in cases where data were available.<sup>[12]</sup> In addition, surgical removal of a carotid plaque caused immediate partial disruption of the baroreceptor activity leading to increased arterial pressure instability.<sup>[24]</sup> Thus, at the end of the surgery, although the BP of some cases did not reach the criteria of hypertension, their BP is higher than preoperational. This mild but noticeable increase in BP did not greatly elevate the velocity, but this hemodynamic instability is definitely a predictive factor of postoperation hypertension, which has a significant relation with CHS.<sup>[13]</sup> This is the reason why BP increasing more at the early stage after surgery in the CHS patients than it in the normal patients. This was also demonstrated in our cohort. In consideration of the theory and phenomenon stated above, the VBI is a reasonable and effective parameter to predict CHS. VBI combines the effect of postoperative BP changes to predict that patients are at risk for CHS, and that is why using VBI as a predictive value of CHS can decrease the number of false negative patients. As far as we know, studies on combining BP and velocity changes in the prediction of CHS have never been performed.

Various authors have supported the diagnostic criteria of CHS based on the combination of the clinical picture with imaging techniques to exclude the ischemic patients.<sup>[4,14,25]</sup> But some authors hold the idea that >100% increase of cerebral blood flow or velocity at the symptom occurred should be added in the criteria of CHS.<sup>[11]</sup> There are studies found that some patients can develop CHS with moderate increases in perfusion.<sup>[26,27]</sup> CHS is still a clinical diagnosis, despite its name; it is not always associated with significantly increased blood flow.<sup>[28]</sup> Accordingly, if we add MCAV increase >100% as a diagnostic criterion in patients with CHS, then it will inevitable bias the results, because of the velocity increase was used in both diagnosis and prediction. So we adopted the recognized criteria for CHS to avoid the bias of the study.

The TCD results can impact by many factors, so we chose the relatively stable time points to measure it, thus can increase the accuracy. It has been demonstrated that after 30 min extubation, cerebral blood flow will tend to be stable, so we chose this time point to record the value.<sup>[13]</sup> In addition, most anesthesia operation began with 15–20 min before surgery, at 30 min there is no anesthesia interference in the patient, this time point can reflect a stable basal status of the cerebral blood flow, so we chose this time point as the baseline record.

In conclusion, at the early stage after CEA, the BP of CHS patients are increased more than for non-CHS patients. In addition, VBI combined velocity and BP changes can be a

new parameter to predict CHS in patients who underwent CEA under general anesthesia. This is an exploring study, so the result should be confirmed in further larger studies.

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