ISSN 2005-7881
Journal of Neurosonology 5(Suppl. 2):32-34, 2013

TCD as an unique diagnostic tool vascular reserve

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Acetazolamide (Diamox)

- Causes a arteriolar dilatation
  - mediated by an increase in carbon dioxide concentration secondary to an inhibition of erythrocyte carbonic anhydrase
  - with no change in diameter of the basal cerebral arteries insonated with TCD (eg, the M1 segment of the MCA)
- After acetazolamide-induced arteriolar dilatation
  - a relative increase in the mean MCA blood flow velocity
  - a relative decrease in the PI

Acetzolamide vs. CO_2 inhalation (4~6%)

- Acetazolamide lacking several of current contraindications for induced hypercapnea
  - history of cardiac arrhythmia
  - unstable angina
  - chronic obstructive pulmonary disease
  - uncontrolled hypertension
- Acetazolamide
  - less changes in heart rate and blood pressure than CO_2 inhalation
  - irreversible for about 30 min
  - a vascular steal phenomenon

Acetazolamide TCD

- Good correlation (r = 0.63) between the elevation of regional cerebral blood flow by SPECT and increase in flow velocity by TCD, using acetazolamide as the vasodilatory stimulus (Dahl et al. Stroke 1992)
- 15 ~ 20 minutes before and 15 ~ 20 minutes after the intravenous injection of 500 mg ~ 1 g of acetazolamide
- Increments of CBF start 2 minutes after acetazolamide injection and attain a maximal effect at 20 minutes; this lasts for 30 minutes after injection of acetazolamide

Vasomotor reactivity was calculated as the percentage change of MBFV by means of the formulation described in previous studies [100 × (MBFV2 – MBFV1)/MBFV1][11–13], where MBFV1 was MBFV obtained from MCA during rest, and MBFV2 was MBFV obtained from MCA 20 minutes after acetazolamide infusion. As described in previous studies, an increase of more than 30% in VMR was accepted as normal; less than 30% was considered abnormal.[13]

Breath-holding

- Inexpensive, popular alternative
- Lower sensitivity

Breath-Holding Index

The BHI was obtained according to a previously reported protocol.[4] Once stable baseline velocities and CO_2 measurements were obtained, subjects were instructed to hold their breath for 30 seconds after a normal inspiration (to avoid a Valsalva maneuver). Mean flow velocities were then recorded. If a breath could not be held for 30 seconds, the abbreviated time of breath-holding was documented. BHI was calculated by dividing the percentage increase of time-averaged mean flow velocity (MFV) after breath-holding by seconds of apnea. We considered values of BHI <0.69 as impaired.[4]

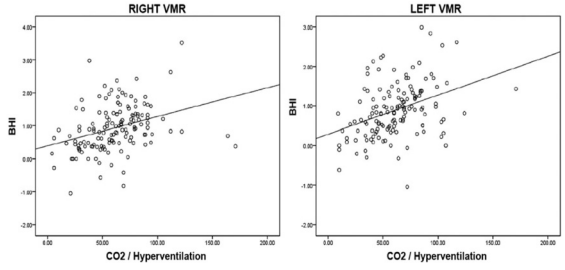

FIGURE 1. Simple linear regression for the correlation of the quantitative estimates of cerebrovascular reactivity (VMR) as evaluated by breath-holding index (BHI) and CO_2 inhalation/hyperventilation (%).

Hemodynamic reserve

- Decreased cerebral perfusion pressure from carotid occlusive disease sequentially followed by
  - promotes a compensatory arteriolar dilatation and recruitment of collateral blood flow
  - chronically predilated arterioles (ie, an exhausted autoregulatory reserve)
  - creates a state of impaired responsiveness in the cerebral vasculature to incraesed carbon dioxide that promotes arteriolar dilation
  - a smaller change in mean MCA velocity and the PI after acetazolamide challenge → may indicate a greater risk for hypoperfusion ischemia

Table 4. Comparison of Initial VMR and 6-Month VMR of Both Groups

Finding	Intracranial Stenosis	Extracranial Stenosis	P*
Contralateral hemisphere: initial VMR	18.81 ± 12.00	15.96 ± 10.49	>.05
Ipsilateral hemisphere: initial VMR	22.44 ± 9.15	13.44 ± 12.87	<.05
Contralateral hemisphere: 6-month VMR	17.55 ± 9.56	22.87 ± 13.85	>.05
Ipsilateral hemisphere: 6-month VMR	17.55 ± 8.16	22.47 ± 13.91	>.05

Values are mean ± SD.
*Independent *t* test.

J Ultrasound Med 2007; 26:179–185

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Objective. In patients with large artery disease, determining the cerebral hemodynamic state and following its alterations may be a good marker for predicting long-term outcome. The aim of our study was to compare the changes in vasomotor reactivity (VMR) of patients with symptomatic intracranial and extracranial artery stenosis. We also examined whether VMR after stroke influences the long-term prognosis for these patients. ***Methods.*** Forty-one patients were included in the study. To determine the cerebral hemodynamic state, transcranial Doppler ultrasound examinations and acetazolamide tests were performed after acute stroke and repeated after 6 months. We compared the VMR on admission and at 6 months, together with changes in VMR, of the patients with symptomatic intracranial and extracranial artery stenosis. By calculating the Barthel index at 6 months, we examined whether VMR had an effect on an improvement in their quality of life. ***Results.*** We observed a significantly higher initial VMR of the ipsilateral hemisphere in patients with intracranial stenosis (22.4 ± 9.1 versus 13.4 ± 12.8; $P = .013$). At 6 months, the VMR obtained from the ipsilateral hemisphere was better in patients with extracranial stenosis than in the patients with intracranial stenosis ($P = .01$). The ipsilateral VMR measured on admission showed a positive correlation with the Barthel index at 6 months ($P = .007$; $r = 0.434$). ***Conclusions.*** Our study showed that VMR in patients with acute stroke who have extracranial and intracranial artery stenosis measured by using a transcranial Doppler examination may have value in predicting long-term outcome. ***Key words:*** large artery atherothrombosis; transcranial Doppler examination; vasomotor reactivity.

Implications of measuring hemodynamic reserve

- Prediction of stroke
  - candidacy of asymptomatic patients with internal carotid artery stenosis for endarterectomy
    - increased risk of ischemic strokes in patients with severe carotid stenosis and impaired autoregulation
  - assessment of potential risk for cerebral ischemia in patients going for major anesthesia and surgery
  - follow-up (TCD) tests: cerebral hemodynamics may spontaneously progress or regress over time
- ? Intracranial atherosclerotic disease