Reversed Robin Hood Syndrome in Acute Ischemic Stroke Patients

Andrei V. Alexandrov, MD; Vijay K. Sharma, MD; Annabelle Y. Lao, MD; Georgios Tsivgoulis, MD; Marc D. Malkoff, MD; Anne W. Alexandrov, PhD

Background and Purpose—Recurrent hemodynamic and neurological changes with persisting arterial occlusions may be attributable to cerebral blood flow steal from ischemic to nonaffected brain.

Methods—Transcranial Doppler monitoring with voluntary breath-holding and serial NIH Stroke Scale (NIHSS) scores were obtained in patients with acute middle cerebral artery or internal carotid artery occlusions. The steal phenomenon was detected as transient, spontaneous, or vasodilatory stimuli-induced velocity reductions in affected arteries at the time of velocity increase in normal vessels. The steal magnitude (%) was calculated as [(MFVm−MFVb)/MFVb]×100, where m=minimum and b=baseline mean flow velocities (MFV) during the 15- to 30-second period of a total 30 second of breath-holding.

Results—Six patients had steal phenomenon on transcranial Doppler (53 to 73 years, NIHSS 4 to 15 points). Steal magnitude ranged from −15.0% to −43.2%. All patients also had recurrent neurological worsening (>2 points increase in NIHSS scores) at stable blood pressure. In 3 of 5 patients receiving noninvasive ventilatory correction for snoring/sleep apnea, no further velocity or NIHSS score changes were noted.

Conclusions—Our descriptive study suggests possibility to detect and quantify the cerebral steal phenomenon in real-time. If the steal is confirmed as the cause of neurological worsening, reversed Robin Hood syndrome may identify a target group for testing blood pressure augmentation and noninvasive ventilatory correction in stroke patients. (Stroke. 2007;38:3045-3048.)

Key Words: arterial occlusion ■ hemodynamics ■ sleep apnea ■ stroke ■ transcranial Doppler

The concept of blood flow steal with arterial occlusions is well known.1 In brain, hemodynamic steal and shunts were documented with angiomas.2,3 Neurological symptoms were linked to cerebral blood flow reduction with arteriovenous malformations2 or rare cases of the subclavian steal syndrome.4 However, the concept of arterial steal has not been evaluated in real-time in acute ischemic stroke.

Clinical deterioration after improvement can occur in 15% of acute stroke patients most commonly with arterial recanalization.5 Although possible mechanisms of neurological fluctuations include a broad differential, one could be vasodilation of the nonaffected brain that can steal blood flow from ischemic tissues.

Subjects and Methods

Our routine clinical and diagnostic protocol for consecutive ischemic stroke patients includes serial NIHSS scores, standard diagnostic transcranial Doppler (TCD) and TCD monitoring with voluntary breath-holding index (BHI).6 Patients are instructed not to take breath in or strain at the beginning of breath-holding. We measured the mean flow velocity (MFV), thrombosis in brain ischemia flow grades,7 and BHI in the affected and normal middle cerebral artery (MCA). All patients also had persisting MCA or internal carotid artery occlusions by our validated criteria.7

Hypercapnia induces vasodilation mainly at the arteriolar level. This decrease in resistance produces an increase in flow velocities in the proximal intracranial vessels, and normal BHI values are >0.69. In patients with impaired vasomotor reactivity, hypercapnia induces less vasodilation with BHI ≤0.69. With exhausted vasomotor reactivity, there is no vasodilation during hypercapnia and BHI is 0. BHI assesses velocity changes at the end of 30-sec breath-holding and does not take into account possible transient velocity decreases. If steal occurs during breath-holding, it should manifest as velocity decrease at the time of initial normal vessel dilation (that could be expected at 15 to 30 second) as pressure gradient shifts toward vessels that can dilate more in response to hypercapnia.

Therefore, steal was defined as mean flow velocity (MFV) decrease in the affected vessel at the time of spontaneous or hypercapnia-induced velocity increase in the normal MCA. The steal magnitude was quantified as the maximum negative percent velocity reduction during breath-holding: steal magnitude=[(MFVm−MFVb)/MFVb]×100, where m indicates minimum and b indicates baseline MFV. We evaluated continuous TCD data for minimum MFV appearance at the time of initial hypercapnia-induced vasodilation during a 15- to 30-second period of a total of 30 seconds of breath-holding. Steal was considered present when steal magnitude was <0 in the affected vessel. After steal was documented on TCD,
Table. Baseline Characteristics

<table>
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<th>Age</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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<tr>
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<td>M</td>
<td>M</td>
<td>F</td>
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<td>ICA/MCA</td>
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<td>8</td>
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<td>LVA</td>
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<td>No</td>
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<td>3</td>
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<tr>
<td>DWI/PWI mismatch</td>
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<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<td>Yes</td>
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<tr>
<td>Fluctuation (NIHSS)</td>
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<td>4</td>
<td>4</td>
<td>3</td>
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<tr>
<td>BHI affected side</td>
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<td>0.2</td>
<td>–0.17</td>
<td>–0.2</td>
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<tr>
<td>BHI nonaffected side</td>
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<td>0.8</td>
<td>0.8</td>
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<td>0.8</td>
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</tr>
</tbody>
</table>

CE indicates cardioembolism; DWI, diffusion-weighted imaging; LVA, large vessel atherothrombosis; PWI, perfusion-weighted imaging.

reversed Robin Hood syndrome was suspected if recurrent neurological worsening by $\geq 2$ NIHSS points were observed without concurrent changes in blood pressure or arterial patency.

Results

The first 6 patients met TCD and clinical criteria for steal/syndrome (Table). Spontaneous, transient, and recurrent residual flow velocity decreases (Figure A) occurred without changes in blood pressure, thrombolysis in brain ischemia grades, flow resistance, or early re-occlusion. In all patients, MFV in the affected vessels decreased by $\geq 20\%$ for periods ranging 10 to 20 second, and with variable frequency from 1 to 5 min for up to every 2 hours.

Voluntary hypercapnia produced transient MFV decrease by $\geq 10\%$ at 18 to 25 sec with gradual but incomplete MFV recovery by 30 to 34 seconds. The steal magnitude ranged from $-15\%$ to $-43.2\%$ (Figure B). Contralateral MCA response to hypercapnia was normal in all patients (Table, Figure B).

As part of routine neuroimaging, the steal was also documented on CT-perfusion before and after Diamox (patient 5, Figure B). All patients had a diffusion-less-than-perfusion mismatch of $\geq 20\%$ on magnetic resonance within 4 to 24 hours after symptom onset (Figure A).

All patients had recurrent fluctuation of aphasia or hemiparesis by 3 to 6 points the same day TCD showed the steal phenomenon. In 3 of 5 patients with sleep apnea, neurological symptoms were worse on awakening and improved with return to daily activities. No further MFV or NIHSS score changes were observed in 3 of 5 patients placed on positive airway pressure bi-level.

Discussion

We report paradoxical changes in cerebral hemodynamics occurring spontaneously or in response to vasodilatory stimuli. Although velocity does not equal flow, MFV changes at a constant insonation angle reflect changes in flow volume. Observed velocity fluctuations even without breath-holding may simply reflect the instability of vasomotor tone caused by ischemic insult. With persisting proximal arterial occlusions, hypercapnia can paradoxically decrease the residual flow velocity in the affected vessel at the expected time of normal brain vasodilation when blood pool is shifted to nonischemic areas. We termed this “reversed Robin Hood” for analogy with “rob the poor to feed the rich.”

The cerebral blood steal phenomenon, if associated with desaturation of oxygen, a common occurrence in sleep apnea, and with the natural blood pressure dip observed in sleep, would create the “perfect storm” during nocturnal sleep to further damage brain tissues with poor hemodynamic reserve. Insufficient collaterals that could also be seen in patients with tandem lesions can further contribute to hemodynamic compromise. It remains to be determined whether the observed velocity changes truly identify the steal from transcortical and other collaterals, or they coincide with other phenomena responsible for clinical changes. Nevertheless, these stroke patients may potentially benefit from noninvasive ventilatory correction, shown to reduce new vascular events after stroke. Although we did not actively change blood pressure in our patients, we hypothesize that experimentally induced hypertension may also be tested in these circumstances.

Transient velocity decreases and incomplete velocity recovery with breath-holding were not previously described. In fact, zero BHI value could be misleading. A velocity decrease in the affected vessel at the time of normal vessel dilation indicates steal because blood follows the path of least resistance. Normal vessels probably are able to dilate to a greater extent than already dilated vessels in the ischemic region. However, subsequent partial velocity recovery suggests some additional dilation in the affected vessel. Therefore, BHI=0, as measured at 30 to 34 seconds of breath-holding, may not always mean “exhausted” or absent vasomotor reactivity. Instead, a limited compensatory attempt to balance hemodynamics in the affected vessels can be present in response to a shift in flow volume. The key is to examine velocity behavior at the time of expected normal vessel dilation (15 to 30 seconds) that is usually overlooked because BHI formula focuses on 30- to 34-second velocities and BHI is often performed with a single transducer, not with bilateral simultaneous monitoring. Recent improvements in digital multi-gate Doppler will enable simultaneous assessment of vasomotor reactivity in the MCA and ipsilateral anterior or posterior cerebral arteries to determine which arterial territory may profit from this steal.

Our report has obvious limitations because a causative link between TCD and clinical findings cannot be established with small numbers of patients. No normative data are available for BHI in these circumstances, and our steal magnitude calculations would be difficult to compare to cerebral blood flow imaging modalities that take longer acquisition times. What could be other mechanisms for neurological fluctuations are uncertain. Finally, the noticed symptom improvement while on bi-level may not be mediated by correction of cerebral hemodynamics.

In conclusion, our report describes criteria to suspect and quantify a cerebral steal phenomenon in real-time on TCD. If confirmed in subsequent studies, reversed Robin Hood syn-
Figure. A, Spontaneous velocity fluctuation with thrombolysis in brain ischemia 3 residual flow (patient 2) with embolic occlusion (MRA insert) and diffusion-perfusion mismatch (MRI insert). Bottom left, Thrombolysis in brain ischemia 3 waveform and flow diversion. Steal magnitude = [25 cm/sec–44 cm/s)/44 cm/sec] × 100 = – 19/44] × 100 = –43.2%. B, Bilateral MCA monitoring during BHI (patient 5) with a proximal internal carotid artery occlusion. Affected MCA MFV decreased at 18 s (middle frame) Steal magnitude = [34 cm/s–40 cm/s)/40 cm/s] × 100 = [–6/40] × 100 = –15.0%. Increased flow on left nonischemic hemisphere and decreased right MCA flow indicating vasoparalysis on CT perfusion after Diamox. In this situation the pressure gradient over leptomeningeal collaterals may decrease and manifest as a deterioration of the patient’s neurological status.
drome may identify a target group of patients for testing blood pressure augmentation and noninvasive ventilatory correction in stroke treatment and prevention.

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**Disclosures**

None.

**References**

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