Prevalence and Risk Factors Associated With Reversed Robin Hood Syndrome in Acute Ischemic Stroke

Andrei V. Alexandrov, MD; Huy Thang Nguyen, MD; Marta Rubiera, MD; Anne W. Alexandrov, PhD; Limin Zhao, MD; Ioannis Heliopoulos, MD; Alice Robinson, RVT; Jennifer DeWolfe, DO; Georgios Tsivgoulis, MD, FESO

Background and Purpose—Early deterioration can occur after acute stroke for a variety of reasons. We describe a hemodynamic steal and associated neurological deterioration, the reversed Robin Hood syndrome (RRHS). We aimed to investigate the frequency and factors associated with RRHS.

Methods—Consecutive patients with acute cerebral ischemia underwent serial National Institutes of Health Stroke Scale and bilateral transcranial Doppler monitoring with breathholding. Steal magnitude (%) was calculated from transient mean flow velocity reduction in the affected arteries at the time of velocity increase in normal vessels. Excessive sleepiness and likelihood of sleep apnea were evaluated by the Epworth Sleepiness Scale and Berlin Questionnaire.

Results—Among 153 patients (age, 61 ± 14 years; 48% women; 21% transient ischemic attack) admitted within 48 hours from symptom onset, 21 (14%) had steal phenomenon (median steal magnitude, 20%; interquartile range, 11%; range, 6% to 45%); and 11 (7%) had RRHS. RRHS was most frequent in patients with proximal arterial occlusions (17% versus 1%; P<0.001). The following factors were independently (P<0.05) associated with RRHS (multivariate logistic regression model): male gender, younger age, persisting arterial occlusions, and excessive sleepiness (P<0.001). A 1-point increase in the Epworth Sleepiness Scale was independently related to an increased likelihood of RRHS of 36% (95% CI, 7% to 73%).

Conclusions—RRHS and hemodynamic steal can be found in 7% and 14%, respectively, of consecutive patients with stroke without other known causes for deterioration. Patients with persisting arterial occlusions and excessive sleepiness can be particularly vulnerable to the steal. (Stroke. 2009;40:2738-2742.)

Key Words: arterial occlusion | reversed Robin Hood syndrome | sleep apnea | stroke | transcranial Doppler

Neurological deterioration can occur in approximately 15% of patients with acute stroke.1–3 Several mechanisms can lead to ischemic lesion extension and subsequent neurological worsening, including reocclusion, edema progression, and cardiovascular instability.4–6 However, these long-recognized mechanisms do not account for all cases of neurological deterioration or symptom recurrence. Changes in cerebral hemodynamics can be detected in real time using transcranial Doppler (TCD), and several groups, including ours, deployed this modality to determine predictors of neurological deterioration.5,6,8

We observed paradoxical decreases in flow velocity during episodes of hypercapnia in vessels supplying ischemic areas of the brain at the time of expected velocity increase in nonaffected vessels.8 Hypercapnia triggered vasodilation more effectively in normal vessels, thus producing arterial blood flow steal toward the path of least resistance. The steal magnitude was linked to severity of neurological worsening in patients with acute stroke.8,9 We termed this “reversed Robin Hood” for an analogy with “rob the poor to feed the rich.”8 In the first documented cases of reversed Robin Hood syndrome (RRHS), neurological worsening was also more pronounced in patients with sleep apnea,8 a condition that can trigger a perfect storm in a patient with acute stroke, whereas apnea correction can reduce the chances of new vascular events.10 There is a growing interest in obstructive sleep apnea and noninvasive ventilatory correction in acute stroke,11,12 and RRHS may provide a missing link between the respiratory status and neurological worsening.

However, it remains unknown how often the steal and clinical syndrome can be detected. We therefore set out to prospectively determine the prevalence and risk factors associated with intracranial hemodynamic steal phenomenon and RRHS in patients with acute cerebral ischemia. We hypothesized that RRHS would be positively associated with the likelihood of obstructive sleep apnea syndrome and with...
greater neurological deterioration in the setting of acute cerebral ischemia.

### Subjects and Methods

Consecutive patients with symptoms of both posterior and anterior acute cerebral ischemia admitted within 48 hours from symptom onset to our tertiary hospital stroke service were prospectively evaluated. Patients who met inclusion criteria were adults ≥19 years of age, had an ischemic stroke or transient ischemic attack (TIA), had temporal windows for TCD examination, and consented to participation in the study. According to the Trial of Org 10172 in Acute Stroke Treatment criteria, ischemic strokes were classified based on etiopathogenic mechanisms into the following groups: large artery atherosclerotic stroke, cardioembolic stroke, small artery occlusion or lacunar stroke, and infarct of undetermined cause. Because we aimed to detect the prevalence of RRHS in consecutive patients with acute cerebral ischemia, all ischemic stroke subtypes were included in the present study. Patients with known causes of clinical or neurological deterioration, including reocclusion, continuing embolization, edema with mass effect, cardiovascular instability, and hemorrhagic transformation were excluded from the study. Consecutive TCD evaluations were performed on a daily basis during hospitalization to document the presence of reocclusion as a cause of deterioration. The project was approved by our Institutional Review Board.

Neurological deficits were measured by serial National Institutes of Health Stroke Scale (NIHSS) scores obtained by certified stroke team members. Neurological deficits were assessed using NIHSS evaluations on a daily basis during hospitalization. Standard diagnostic TCD and bilateral TCD monitoring with voluntary breathholding were performed by registered vascular technologists or registered vascular technologist-eligible sonographers. TCD monitoring of the symptomatic and asymptomatic side was performed simultaneously. As an indirect measure of the effectiveness of breathholding in producing hypercapnia, we used the increase in MFV measured at the end of 30 seconds breathholding.

To quantify this steal phenomenon, we used a methodology to measure the MFV changes during 15 to 30 seconds of breathholding.8 Persistence of an arterial occlusion was documented from CT or MR angiography. Instead, we focused on the initial velocity changes at the time when breathholding just induced an initial rise in CO2. Our rationale for choosing this methodology to quantify this steal phenomenon has been recently described. Briefly, previous steady-state methodologies such as the BHI assess velocity changes at the end of 30 seconds breathholding and therefore do not take into account possible transient velocity decreases. If steal occurs during breathholding, it may also manifest as a velocity decrease at the time of initial normal vessel dilation (that could be expected at 15 to 30 seconds) as pressure gradient shifts toward vessels that can dilate more in response to hypercapnia. Hypercapnia induces vasodilatation mainly at the arteriolar level. This is accompanied by a decrease in resistance in feeding vessels. In turn, blood flow moves faster into a dilated vascular bed, thus increasing velocities in the proximal intracranial vessels. This is consistently seen in normal vessels on the unaffected side of the brain. If a hemodynamic steal occurs during breathholding, it manifests as a velocity decrease in the affected vessel at the time of normal vessel dilation due to transient pressure gradient shifts toward vessels that can dilate more in response to hypercapnia.

Therefore, steal was defined as an MFV decrease in the affected vessel at the time of hypercapnia-induced velocity increase in the normal MCA. The vascular steal phenomenon had to occur in the vascular territory considered responsible for the patient’s ischemic stroke or TIA for the patient to be classified as having RRHS. The steal magnitude (SM, %) was quantified as the maximum negative percent velocity reduction during breathholding: SM = (MFV in normal vessel − MFV in affected vessel)/MFV in normal vessel × 100, where m is minimum and b is baseline MFV. Steal was considered present when SM was negative, ie, SM < 0 in the affected vessel.

In patients with brainstem strokes referable to the basilar artery, bilateral transtemporal insonation was performed. We monitored
posterior circulation (top of the basilar artery; ipsilateral P1 posterior cerebral artery and contralateral P1 posterior cerebral artery)\(^1\)\(^{19}\) with one probe, whereas with the other one, anterior circulation was monitored (ipsilateral MCA). Steal phenomenon was documented when there was reduction in P1 posterior cerebral artery (ipsilateral to the probe monitoring posterior circulation) MFV during breathing with simultaneous increase in MCA MFV (ipsilateral to the probe monitoring anterior circulation) during breathing.

After the steal was documented on TCD, reversed Robin Hood syndrome was suspected if new or recurrent neurological worsening by ≥2 NIHSS points were observed without concurrent changes in blood pressure or arterial patency.\(^3\) Given the current American Heart Association recommendations that advocate against proposed reductions of blood pressure (exceeding ≥20%) in the setting of acute cerebral ischemia,\(^2\) a range of blood pressure oscillations of ≤20% was used for considering blood pressure as stable. Changes of arterial patency were evaluated using consecutive TCD evaluations on a daily basis during hospitalization. For the definition of neurological deterioration in patients with TIA, we used a cutoff of ≥2 points in NIHSS score in the serial NIHSS assessments, because all patients with TIA had a baseline NIHSS score of 0 on hospital admission.

Demographics and common risk factors were documented from routine stroke workup. Patients or family members answered the Epworth Sleepiness Scale (ESS) at the baseline assessment within the first 24 hours from hospital admission, a subjective measure of sleep propensity with scores in 2 or 3 of 3 categories were defined as high risk for excessive sleepiness (40% versus rest 13%; \(P<0.003\)).

The likelihood of obstructive sleep apnea was evaluated with the Berlin Questionnaire at the baseline assessment within the first 24 hours from hospital admission. Patients or family members answered the questionnaire were positive.

<table>
<thead>
<tr>
<th>Variable</th>
<th>RRHS (n=11)</th>
<th>Non-RRHS (n=142)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years 51 ± 9</td>
<td>62 ± 14</td>
<td>0.003*</td>
<td></td>
</tr>
<tr>
<td>Gender, female 1 (9%)</td>
<td>72 (51%)</td>
<td>0.007*</td>
<td></td>
</tr>
<tr>
<td>Race, white 8 (73%)</td>
<td>87 (61%)</td>
<td>0.737</td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus 1 (9%)</td>
<td>29 (20%)</td>
<td>0.324</td>
<td></td>
</tr>
<tr>
<td>Dyslipidemia 9 (82%)</td>
<td>103 (73%)</td>
<td>0.394</td>
<td></td>
</tr>
<tr>
<td>Atrial Fibrillation 1 (8%)</td>
<td>9 (6%)</td>
<td>0.537</td>
<td></td>
</tr>
<tr>
<td>CAD 4 (36%)</td>
<td>22 (16%)</td>
<td>0.093</td>
<td></td>
</tr>
<tr>
<td>LVA 10 (91%)</td>
<td>50 (35%)</td>
<td>&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Baseline NIHSS 2 (0–19; IQR, 3)</td>
<td>3 (0–13; IQR, 9)</td>
<td>0.321</td>
<td></td>
</tr>
<tr>
<td>SM, % 24 ± 10</td>
<td>1:4</td>
<td>&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>ICA 50%–100% stenosis 4 (36%)</td>
<td>25 (18%)</td>
<td>0.131</td>
<td></td>
</tr>
<tr>
<td>Reversed OA 4 (36%)</td>
<td>17 (12%)</td>
<td>0.046*</td>
<td></td>
</tr>
<tr>
<td>Anterior crossfilling 5 (46%)</td>
<td>17 (12%)</td>
<td>0.010*</td>
<td></td>
</tr>
<tr>
<td>PcomA flow 3 (27%)</td>
<td>7 (5%)</td>
<td>0.026*</td>
<td></td>
</tr>
<tr>
<td>ESS score &gt;12 5 (50%)</td>
<td>18 (13%)</td>
<td>0.008*</td>
<td></td>
</tr>
<tr>
<td>Berlin Questionnaire 2+ 3 (38%)</td>
<td>65 (51%)</td>
<td>0.359</td>
<td></td>
</tr>
</tbody>
</table>

CAD indicates coronary artery disease; LVA, large vessel atheromatous stroke mechanism; ICA, internal carotid artery; OA, ophthalmic artery; PcomA, posterior communicating artery; IQR, interquartile range; 2+, at least 2 components of the questionnaire were positive. \(*P<0.05.\)

Statistical analyses were performed with the SPSS 15.0 software (SPSS Inc). The 2-tailed Fisher exact test or Pearson \(\chi^2\) test for categorical variables and Student t test or Mann–Whitney U test for continuous variables were used to assess intergroup differences. Correlations between continuous variables were assessed by the Spearman correlation coefficient. We evaluated the interrater reliability for detection of RRHS using our newly developed dynamic criteria\(^6\) and the more traditional steady-state criteria such as the BHI\(^15\) using Cohen’s \(\kappa\) statistic. Initially, univariable analyses of potential predictors (demographic characteristics, stroke risk factors, admission NIHSS score, systolic blood pressure and serum glucose levels, presence of proximal arterial obstruction on baseline TCD assessment, excessive sleepiness, and likelihood of sleep apnea syndrome on the basis of the ESS and Berlin Questionnaire, respectively) were performed. To maximize sensitivity, those variables with a univariable association of \(P<0.1\) were included as candidates into a multivariable logistic regression model and then removed by the backward stepwise selection procedure. To confirm the robustness of multivariable models, we repeated all multivariable analyses using a forward selection procedure. Predictor variables that were significant at \(P<0.05\) were retained in the multivariable model. A level of \(P<0.05\) was accepted as statistically significant.

**Results**

We studied 153 patients who met inclusion criteria: age 61 ± 14 years, women 48%, and 21% with TIA. We found 21 (14%) patients who had steal phenomenon (median SM, 20%; interquartile range, 11%; range, 6% to 45%) on TCD. The median elapsed time between the beginning of breathholding and documentation of the SM was 23 seconds (range, 17 to 29 seconds). Their baseline characteristics are summarized in Table 1.

**Table 2.** Factors Independently Associated With RRHS on Multivariate Logistic Regression Models

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient (SE)</th>
<th>OR (95% CI)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (per 1-year increase)</td>
<td>−0.156 (0.059)</td>
<td>0.86 (0.76–0.96)</td>
<td>0.008</td>
</tr>
<tr>
<td>Presence of proximal arterial occlusion</td>
<td>3.141 (1.524)</td>
<td>23.14 (1.17–458.63)</td>
<td>0.039</td>
</tr>
<tr>
<td>ESS score (per 1-point increase)</td>
<td>0.308 (0.122)</td>
<td>1.36 (1.07–1.73)</td>
<td>0.012</td>
</tr>
</tbody>
</table>

RRHS was documented in 11 cases (7% of the study population) using our recently developed criteria.\(^8\) The RRHS prevalence using a more steady-state methodology such as the BHI\(^15\) was 6% (n=9). The measure of agreement between the 2 sets of criteria was satisfactory (Cohen’s \(\kappa=0.89\), \(P<0.0001\)). The magnitude of steal was directly related with the NIHSS score increase (range, 0 to 13 points; Spearman’s correlation coefficient 0.453; \(P=0.039\)). RRHS was most frequent in patients with proximal arterial occlusions (17% versus 1%; \(P<0.001\)) and those with both arterial occlusions + excessive sleepiness (40% versus rest 5%; \(P=0.003\)). Among the patients with proximal arterial occlusions, a steal phenomenon was identified in the following vessels: M1 MCA, M2 MCA, and terminal internal carotid artery. In all of these cases, the steal phenomenon was identified in the vessels with acute occlusions. No steal phenomenon was identified in patients with basilar artery occlusions (n=2). Half of patients with RRHS had high NIHSS scores of >12 points (50% versus rest 13%; \(P=0.008\)).

The following factors were independently associated with RRHS on multivariate logistic regression model (Table 2): younger age (\(P=0.008\)), presence of proximal arterial occlusions (\(P=0.039\)) and higher ESS scores (\(P=0.012\)). A 1-point increase in the ESS score was independently related to an increased likelihood of RRHS of 36% (95% CI, 7% to 73%). Greater likelihood of
obstructive sleep apnea (Berlin Questionnaire 2 or more positive parts) was more common among patients with excessive sleepiness (85%) versus rest (44%; \( P=0.01 \)). Neurological improvement at discharge was lower if RRHS was present (median NIHSS decrease 4%; interquartile range, 100%) versus rest (5%; interquartile range, 100%; \( P=0.039 \)).

**Discussion**

Our study showed that intracranial steal and an associated clinical syndrome leading to neurological worsening can be found in a substantial number of consecutive patients with stroke or TIA. Our findings indicate that RRHS was associated with a trend toward less neurological improvement at hospital discharge, although it should be noted that the present report was not designed to investigate a temporal and potentially causative association among hypercapnia, vascular steal phenomenon, and neurological deterioration.

Our study confirms our previous observations and provides data on the syndrome prevalence and associated risk factors. Our data highlight the need to pay more attention to patients with persistent arterial occlusions and excessive sleepiness because these 2 conditions may lead to hemodynamic compromise and subsequent neurological worsening. Although we did not measure arterial blood gases, we suspect that sleep can lead to decreased cardiac output, hypoventilation, and at least transient hypercapnia because these 2 conditions may lead to hemodynamic compromise and subsequent neurological worsening. We hypothesize that these collateral vessels appeared insufficient to fully compensate during sleep and transient hypercapnia.

Although the likelihood of obstructive sleep apnea syndrome did not appear as a factor independently associated with the steal, the majority of patients with excessive sleepiness had Berlin scores predictive of obstructive sleep apnea syndrome. Perhaps the next step could be to evaluate an association between hypoventilation during sleep and the hemodynamic steal phenomenon. The question is whether a confirmation of sleep apnea with a formal polysomnography test provides data on the syndrome prevalence and associated risk factors. Our data highlight the need to pay more attention to patients with persisting arterial occlusions and RRHS.

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known causes for deterioration. Patients with persisting arterial occlusions and excessive sleepiness may be particularly vulnerable to the steal because both excessive sleepiness and proximal arterial occlusions were independently associated with a higher likelihood of RRHS in our multivariate analyses. With or without subsequent confirmation of sleep apnea, these patients may represent a target group for early noninvasive ventilatory correction after acute ischemic stroke.

Disclosures

None.

References


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