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.4-6

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.”9 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 etiopathogenetic mechanisms into the following groups: large artery atherosclerotic stroke, cardioembolic stroke, small artery occlusion or lacunar stroke, and infarct of undetermined cause.13 Because we aimed to detect the prevalence of RRHS in consecutive patients with acute ischemic stroke, all ischemic stroke subtypes were included in the present study. Patients with known causes of clinical or neurological deterioration, including reoxygenation, 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 breathholding14 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 mean flow velocity (MFV) in the asymptomatic side, which was quantified as a breathholding index (BHI) of \( \frac{\text{mean flow velocity in the asymptomatic side}}{\text{mean flow velocity in the affected side}} \times 100 \). The presence of a proximal arterial occlusion was diagnosed when a Thrombolysis in Brain Ischemia flow grade of 0 to 3 was identified in any of the following vessels: M1 middle cerebral artery (MCA), M2 MCA, terminal internal carotid artery, anterior cerebral artery, posterior cerebral artery, vertebral artery, and basilar artery.17–20

Our TCD assessment of vasomotor reactivity differs from previously proposed techniques that focus on steady-state conditions achieved either at the end of breathholding or a plateau response to dose-controlled CO2 inhalation or Diamox injection. 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 arterial 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 nonaffected 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 as 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 velocity decrease in the affected vessel at the time of normal vessel dilation, and therefore do not take into account possible transient velocity changes. Collaterals on TCD were quantified using Thrombolysis in Brain Ischemia flow grades in addition to measuring MFV changes. Collaterals on TCD were identified using previously validated criteria.17–20 The steal magnitude (SM, %) was quantified as the maximum negative velocity decrease in the affected vessel at the time of normal vessel dilation, and therefore do not take into account possible transient velocity changes. Collaterals on TCD were identified using previously validated criteria.17–20 The presence of a proximal arterial occlusion was diagnosed when a Thrombolysis in Brain Ischemia flow grade of 0 to 3 was identified in any of the following vessels: M1 middle cerebral artery (MCA), M2 MCA, terminal internal carotid artery, anterior cerebral artery, posterior cerebral artery, vertebral artery, and basilar artery.17–20

Our TCD assessment of vasomotor reactivity differs from previously proposed techniques that focus on steady-state conditions achieved either at the end of breathholding or a plateau response to dose-controlled CO2 inhalation or Diamox injection. 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 arterial 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 nonaffected 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 as 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 (Figure):

\[
\text{SM} = \left( \frac{\text{MFV}_{m} - \text{MFV}_{b}}{\text{MFV}_{b}} \right) \times 100
\]

where \( m \) is minimum and \( b \) is baseline MFV. Steal was considered present when SM was negative, i.e., SM < 0 in the affected vessel.

In patients with brainstem strokes referable to the basilar artery, bilateral transtemporal insonation was performed. We monitored

Figure. Velocity changes indicating intracranial steal during breathholding in a patient with RRHS. SM was calculated at 20 seconds after the initiation of breathholding. SM = \left( \frac{\text{mean flow velocity in the asymptomatic side}}{\text{mean flow velocity in the affected side}} \right) \times 100 = \left( \frac{-8}{51} \right) \times 100 = -16\%.

Baseline

<table>
<thead>
<tr>
<th></th>
<th>Affected side</th>
<th>Non-affected side</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Affected MCA MFV</strong></td>
<td>51 cm/s</td>
<td>62 cm/s</td>
</tr>
<tr>
<td><strong>Normal MCA MFV</strong></td>
<td>53 cm/s</td>
<td>62 cm/s</td>
</tr>
<tr>
<td><strong>Baseline</strong></td>
<td>71 cm/s</td>
<td>62 cm/s</td>
</tr>
</tbody>
</table>
sleep propensity with scores on the Epworth Sleepiness Scale (ESS) at the baseline assessment within routine stroke workup. Patients or family members answered the questionnaire referred to the patient’s condition before the stroke. Positive responses using a more steady-state methodology such as the BHI were obtained from patients with proximal arterial occlusions. No steal phenomenon was identified in the following cases: M1 MCA, M2 MCA, and terminal internal carotid artery. In all of these cases, the steal phenomenon was documented when there was reduction in P1 posterior cerebral artery (ipsilateral to the probe monitoring anterior circulation) during breathholding.

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. Given the current American Heart Association recommendations that advocate against profound reductions of blood pressure (exceeding ≥20%) in the setting of acute cerebral ischemia, 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 ≥10 defined as excessive sleepiness. The likelihood of obstructive sleep apnea was evaluated with the Berlin Questionnaire at the baseline assessment within the first 24 hours from hospital admission. Both the ESS and Berlin Questionnaire referred to the patient’s condition before the stroke. Positive scores in 2 or 3 of 4 categories were defined as high risk for obstructive sleep apnea.

Statistical analyses were performed with the SPSS 15.0 software (SPSS Inc). The 2-tailed Fisher exact test or Pearson χ² 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 and the more traditional steady-state criteria such as the BHI using Cohen’s κ 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.

RRHS was documented in 11 cases (7% of the study population) using our recently developed criteria. The RRHS prevalence using a more steady-state methodology such as the BHI was 6% (n=9). The measure of agreement between the 2 sets of criteria was satisfactory (Cohen’s κ=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 ESS 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

Table 1. Baseline Characteristics

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

* P<0.05.

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>

P=0.05.
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 persisting 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 hypercarbia, factors that can compromise cerebral perfusion. Patients with symptomatic steal had a greater need for collateral flow recruitment, because the underlying pathogenic mechanism of cerebral ischemia in the vast majority of patients with RRHS was large artery atherosclerotic stroke (91%; Table 1). We hypothesize that these collaterals 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 sleep phenomenon. The question is whether a confirmation of sleep apnea with a formal sleep study is necessary to initiate ventilatory correction or, because these hemodynamic changes are so “front-loaded” and common with sleepiness, there is little time, if any, to do a formal study. Perhaps a prospective documentation of sleep apnea using unattended stroke unit-based polysomnography or even simple nocturnal oximetry (as valid proxies for full-scale sleep laboratory polysomnography) would be sufficient to better understand the association between hypercapnia during sleep apnea and neurological deterioration caused by RRHS.

Our study has limitations. First and most important, because we aimed to document the prevalence of RRHS in consecutive patients with symptoms of acute cerebral ischemia, we included in the present study both patients with posterior and anterior circulation symptoms. Therefore, it may be argued that is unclear whether the effect of hypercapnia is exerted directly through the arterial vessels or else through the response of brainstem chemoceptors. Also, the simultaneous TCD monitoring of posterior (top of the basilar) and anterior circulation (MCA) is technically challenging and this may be related to the fact that we did not document any steal phenomenon in strokes referable to the basilar artery. Second, we performed no monitoring of respiratory function and subsequently we are unable to evaluate a potential relationship between hypercapnia and neurological worsening. Our group plans to address this question in a future study. Third, we used only one Doppler test that provides only a snapshot and does not answer the question what happens to the steal over time. Fourth, because only proximal vessels were studied, we cannot state how often the steal can affect more distal vessels (ie, if an occlusion is more distal than M2). Of note, however, our inability to detect this phenomenon in distal MCA branches would result only in underestimating the prevalence of RRHS. Fifth, our study did not show an independent association between greater likelihood of obstructive sleep apnea syndrome and RRHS/steal likely due to the sample size of the patient population studied. However, an alternative explanation could be given. Sleepiness is more common with larger strokes likely leading to hypventilation and hypercapnia that can have a more profound effect producing symptomatic steal with or without sleep apnea. Because the volume of ischemic strokes was not documented in the present report, the potential relationship between ischemic stroke size and excessive sleepiness as well as occurrence of RRHS remains to be determined in a future study. Furthermore, we plan to further investigate the trend toward less neurological improvement during hospitalization in patients with proximal arterial occlusions and RRHS compared with cases with proximal arterial occlusions and absent vascular steal phenomenon, because the documented 1% difference in neurological improvement is hardly strong evidence for a meaningful clinical impact. Sixth, the introduction of novel dynamic ultrasonographic criteria for the detection of RRHS may constitute another study limitation. Interestingly, the interrater agreement between our newly developed and more traditional steady-state criteria such as the BHI for detecting RRHS was satisfactory (\(\kappa=0.89\)). Nevertheless, we consider that our results regarding RRHS prevalence should be cautiously interpreted and replicated in a larger series of patients by other independent investigators using both recent dynamic and more traditional steady-state ultrasonographic criteria. Finally, it should be acknowledged that ESS and the Berlin Questionnaire are designed for outpatient ambulatory use and may not be applicable in patients with acute stroke. This is the reason why, in cases of noncooperative patients (sustaining a severe stroke), we asked the relatives to answer the questionnaires. Also, the ESS and Belin Questionnaire have been previously used in studies evaluating sleep apnea in the setting of acute ischemic stroke.

In summary, RRHS and hemodynamic steal can be found in a substantial number of consecutive patients without other...
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

Prevalence and Risk Factors Associated With Reversed Robin Hood Syndrome in Acute Ischemic Stroke
Andrei V. Alexandrov, Huy Thang Nguyen, Marta Rubiera, Anne W. Alexandrov, Limin Zhao, Ioannis Heliopoulos, Alice Robinson, Jennifer DeWolfe and Georgios Tsivgoulis

Stroke. 2009;40:2738-2742; originally published online May 21, 2009;
doi: 10.1161/STROKEAHA.109.547950
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2009 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/40/8/2738

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/