Intracranial angioplasty and stent placement for direct cerebral revascularization of nonacute intracranial occlusions and near occlusions

Clinical article

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Object. The authors reviewed their experience in 7 cases of nonacute intracranial occlusions and near occlusions in which the patients underwent intracranial angioplasty and stent implantation for direct cerebral revascularization.

Methods. Between 2005 and 2008, 4 men and 3 women underwent direct cerebral revascularization of nonacute intracerebral occlusions or near occlusions. Five patients had chronic angiographically documented occlusion and 2 patients had chronic angiographically documented near occlusions. The locations of the treated vessels included 2 supraclinoid internal carotid arteries, 4 middle cerebral arteries, and 1 vertebral artery. Prior to intervention, all patients were symptomatic and experienced strokes ipsilateral to their occlusions. In addition, all patients had clinical or radiographic evidence of ongoing hemodynamic compromise. Five patients underwent successful intracranial angioplasty and stent placement and 2 patients underwent successful intracranial balloon angioplasty alone. The mean time from documented vessel occlusion to treatment was 35 days. All patients had successful revascularization determined using the Thrombolysis in Cerebral Infarction (TICI) scale: TICI Score 3, 2b, and 2a in 4, 2, and 1 patient, respectively, and the mean residual stenosis was 38%.

Results. After uneventful technical procedures, 1 patient suffered a perforator vessel stroke and 1 patient suffered a fatal hemorrhage. Mean modified Rankin Scale score of 2 (range 1–5) and mean Glasgow Outcome Scale score of 4 (range 1–6) were achieved during a mean clinical follow-up period of 399 days (range 1–840 days). Asymptomatic restenosis was documented in 4 patients, 1 underwent bypass retreatment, and 1 patient received repeated balloon angioplasty.

Conclusions. Combined intracranial angioplasty and stent placement is a potential treatment option in selected patients for the direct revascularization of nonacute intracranial occlusions and near occlusions. Whether this represents a substantial risk reduction compared with the best medical therapy or a long-lasting treatment option is unknown. (DOI: 10.3171/2008.12.FOCUS08269)

KEY WORDS • chronic occlusion • intracranial angioplasty and stent placement • intracranial atherosclerosis

WITH the advent of intracranial self-expandable stents for neurointerventional use, there has been a widespread increase in the endovascular treatment of patients with intracranial stenosis of 50–99%. Several studies have reported on the feasibility of intracranial angioplasty and stent placement for this indication;^{3,9,10,15,32,39,40} however, no studies have evaluated in-

tracranial angioplasty and stenting in those patients with near or complete occlusions (> 99 to 100%) as well as loss of cerebrovascular reserve, or stage II hemodynamic failure, a group with high risk of future stroke.

Physicians have been reluctant to treat these lesions with angioplasty and stent placement because of technical feasibility, as well as the risk of showering emboli, perforating fragile intracranial arteries, or reperfusing a territory with potential for hemorrhage. This small case series represents a paradigm shift in the way certain selected patients with nonacute near and complete occlusion can be treated. This report also demonstrates the feasibility of directly revascularizing nonacute intracranial occlusions with current technology.

Abbreviations used in this paper: CBF = cerebral blood flow; CVR = cerebrovascular reserve; EC-IC = extracranial-intracranial; ICA = internal carotid artery; MCA = middle cerebral artery; MTT = mean transit time; TIA = transient ischemic attack; TICI = Thrombolysis in Cerebral Infarction; VA = vertebral artery; WASID = Warfarin-Aspirin Symptomatic Intracranial Disease.

Fig 1. Diamox-based CT perfusion scans obtained in a 74-year-old man with a history of intracranial atherosclerosis and MCA stenosis, on Coumadin since 2001, who presented with recurrent episodes of speech arrest. No evidence of acute infarction was detected on MR imaging. The scan reveal steal phenomenon in the left hemisphere with prolongation of MTT (*left*) and decrease in the CBF (*right*).

Methods

We performed a retrospective chart review (2005–2008) of all patients treated at our center with angioplasty and/or stent surgery for nonacute intracranial near occlusions or complete occlusions.

Patient Selection

This method of treatment was reserved for selected patients with documented stroke and progression of symptoms despite the best medical therapy. Hemodynamic TIAs were demonstrated in 3 patients. Loss of cerebral autoregulatory reserve, as demonstrated on CT perfusion scanning with acetazolamide, was observed in 4 patients.

Computed Tomography Perfusion

Dynamic contrast-enhanced CT perfusion scanning was performed before and after administration of acetazolamide (with 100 ml Optiray-320 contrast and 1 g acetazolamide in those patients with no obvious clinical signs of hemodynamic failure). Computed tomography perfusion scanning was performed using a standard technique, and functional maps were calculated. Cerebral blood flow, cerebral blood volume, and MTT were calculated by manually drawing regions of interest to avoid areas of infarction and cortical blood vessels. The percentage change for CBF and MTT was calculated: percentage change parameter = (the parameter post-acetazolamide administration – the parameter pre-acetazolamide administration)/parameter pre-acetazolamide administra-

tion $\times 100$. Deranged CVR was demonstrated as $< 30\%$ increase in CBF and a prolongation of MTT. Computed tomography perfusion scanning before acetazolamide administration demonstrated decreased CBF and prolonged

Fig 2. Catheter angiogram demonstrating near occlusion of the left M₁ segment. A microcatheter with an exchange microguidewire was used to catheterize the left MCA.

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Fig 3. Supraselective angiogram demonstrates the anatomy.

MTT in the diseased territories. Derangement of the CVR was classified as mild (10–30% percentage change), moderate (0–10% percentage change) and severe (negative percentage change) in CBF (in other words, the steal phenomenon).

Definitions of Complete Occlusion and Near Occlusion

Complete occlusion was defined by catheter-based angiography as an abrupt cutoff of the index vessel with no continued antegrade flow in the downstream territory or a TICI Scale score of 0 flow. Near occlusion was defined by catheter angiography as stenosis > 99% with minimal or trickle antegrade flow past the initial obstruction and limited distal branch filling with little or slow distal perfusion (that is, TICI Scale score of 1).¹⁶ In near occlusion, the caliber of the distal vessel was significantly narrowed and difficult to characterize with the vessel walls collapsed. The standard WASID Trial method was used to measure degree of stenosis.³⁰

The term nonacute occlusion or near occlusion was defined as an occlusion or near occlusion well outside the traditional window of opportunity for acute chemical or mechanical thrombolysis, 6–8 hours for an anterior circulation occlusion, and 24 hours for a posterior circulation occlusion.^{11,14,23,31} In all patients the diagnosis of intracranial occlusion or near occlusion was made by catheter angiography before treatment.

Management Options

The management options, including the risks and benefits of each treatment modality, were reviewed with all the patients. The treatment options included continued best medical therapy with statins and antiplatelet agents, EC-IC bypass if possible, or attempt at direct revascularization with angioplasty and/or stent placement. The patient and family were counseled that both surgical bypass

Fig 4. Two balloon inflations were performed with a 2 × 9-mm Gateway balloon.

and angioplasty/stent placement are considered investigational treatment options. In addition, they were informed that scientific evidence is lacking to support the benefits of intracranial angioplasty and stent implantation compared with best medical therapy or surgical bypass. After multidisciplinary analysis, with the goal of preventing future stroke and minimizing procedural complications, direct revascularization with off-label use of intracranial angioplasty and/or stent placement was recommended in these select patients.

Operative Technique

The Wingspan stent system was used in compliance with internal review board approval at our center. The endovascular procedures were performed after induction of general anesthesia. All patients were pretreated 5 days prior to the procedure with aspirin (325 mg/day) and clopidogrel (75 mg/day or loaded with 600 mg clopidogrel) as well as aspirin (325 mg) 6 hours prior to the procedure. Preprocedural platelet function or platelet inhibition was not tested. The procedures were performed with systemic intraprocedural heparinization, with bolus dose 60 U/kg followed by 12 U/kg/hour. A 6 Fr guide catheter was placed in the ICA or VA via a transfemoral approach. Using a coaxial technique with a Prowler (Cordis) or Excelsior SL 10 (Boston Scientific) microcatheter and Agility 0.014 microguidewire (Cordis), the occlusion or near occlusion was gently traversed. In all cases, the occlusions were easily crossed without significant microguidewire manipulation or force. Selective microcatheter injection distal to the occlusion was performed to confirm anatomical position and rule out extravasation, perforation, or dissection. An exchange length 0.014 Transend 300 ES Floppy microguidewire (Boston Scientific) was used to

Fig 5. Angiogram obtained after deployment of a 3 × 15-mm Wingspan stent, showing revascularization of the MCA territory flow (TICI Scale score of 3) and 30% residual stenosis.

exchange the microcatheter for the angioplasty balloon, Gateway (Boston Scientific) or Sprinter (Medtronic), for predilation. The predilation balloon diameter was generally 1.5 or 2 mm and was purposely undersized. The balloon was then exchanged for the stent system. In 2 cases, balloon angioplasty was performed alone without insertion of a stent. In the case of near occlusion or occlusions, the distal vessel diameter and distal landing zones were often unknown. This information was predicted by the

distal microcatheter injection. The stent diameter was generally oversized, usually 0.5–1.0 mm of the predicted vessel diameter. The proximal one-third to one-half of the stent was positioned at the point of occlusion. As indicated, postdilation of the stent was performed with a balloon sized to 80% of the predicted vessel diameter. Delayed angiographic runs were performed to rule out thrombosis, spasm, or dissection. The exchange wire and the guide catheter were then removed. The femoral puncture site was closed with the Perclose or Starclose device (Abbott Vascular).

All patients were admitted to the neurointensive care unit for neurological and hemodynamic monitoring. The intraprocedural heparinization was allowed to normalize, and dual antiplatelet agents were continued for a minimum of 3 months.

Clinical Outcomes

Follow-up information was obtained in each case from postprocedural clinical evaluation and the medical records. The following clinical outcomes were evaluated: any stroke or death within 24 hours and 30 days of the procedure, and ischemic stroke in the territory of the stented artery 30 days postprocedure. Stroke was defined as any hemorrhagic or ischemic event associated with a neurological deficit lasting longer than 24 hours. All procedurally related complications were documented. Technical success of the procedure was defined as improvement of blood flow after balloon angioplasty and/or stent placement across the target lesion. Postprocedural TICI grading was performed.¹⁶ Preprocedural and postprocedural modified Rankin Scale and Glasgow Outcome Scale scores were assessed.^{2,18} Follow-up angiographic data, as clinically indicated, were collected to estimate the rate of restenosis. Restenosis was defined as > 50%

Fig 6. Posttreatment CT perfusion scan demonstrating marked improvement in CBF (left) and MTT (right).

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TABLE 1: Summary of demographic, treatment, and clinical outcome data

Age (yrs)	Vessel Treated	Near Occlusion	Occlusion	TICI Score		% Residual stenosis	Angioplasty Balloon (mm)	Stent (mm)	% Stenosis at Follow-Up (mos)	Complications	Retreatment
				Pretreatment Flow	Post-treatment Flow						
66	lt supraclinoid ICA	no	yes	0	3	40	Gateway 1.5 x 9	Wingspan 3 x 15	0 (14)	none	
74	lt MCA M ₁ segment	yes	no	1	3	30	Gateway 2 x 9	Wingspan 3 x 15	60 (14)	none	
54	rt supraclinoid ICA	no	yes	0	2b	40	Gateway 2 x 9	Wingspan 4 x 20	no follow-up imaging	cerebral hemorrhage	
41	lt MCA M ₁ segment	yes	no	1	2a	0	Gateway 1.5 x 9	Wingspan 3 x 15	no follow-up imaging	acute infarct	
59	rt MCA M ₁ segment	no	yes	0	3	40	Sprinter 2.5 x 6	angioplasty alone	95 (11)	none	EC-IC bypass
56	lt VA	no	yes	0	3	48		Neuroform 4 x 20	90 (6)	none	angioplasty
64	lt MCA M ₁ segment	no	yes	0	2b	65	Gateway 2 x 9	angioplasty alone	80 (4)	none	

luminal narrowing or interval worsening from immediate posttreatment result (Figs. 1–7).

Results

Four men and 3 women were included in this study. The mean age was 59.3 years. All patients were symptomatic having experienced documented strokes despite best medical therapy. Clinical or radiological evidence of hemodynamic compromise was suspected in all 7 patients, with angiographically proven occlusion in 5 patients and angiographically proven near occlusion in 2 patients. The treated vessels included 2 supraclinoid ICAs, 4 MCAs, and 1 VA.

Three patients experienced clear hemodynamic symptoms—for example, limb-shaking TIAs or posturally related and reproducible symptoms. Four patients did not have clear hemodynamic events and therefore underwent CT perfusion scanning with and without acetazolamide. In all 4 patients, CT perfusion scanning demonstrated severe derangement of CVR. An underlying focal high-grade intracranial stenosis that led to occlusion or near occlusion was suspected in all patients. Despite proximal intracranial near occlusion or complete occlusion, all patients exhibited some distal collateral flow via leptomeningeal retrograde collaterals, or flow was predicted by preserved neurological function in the downstream territory. All patients treated had chronic occlusions or near occlusions. The mean time from radiographic documentation of occlusion/near occlusion to treatment was 35 days, which most likely underestimated the chronicity of occlusion. Five of 7 patients underwent angioplasty and stent placement. Two of 7 patients underwent angioplasty alone. All patients had successful revascularization with a TICI score of 3 in 4 patients, 2b in 2 patients, and 2a in 1 patient; the mean immediate post-treatment residual stenosis was 38% (range 0–65%). One

patient suffered a periprocedural perforator vessel stroke, and one patient suffered a fatal hemorrhage several hours after an uneventful technical procedure. No patient experienced a nonprocedurally related ipsilateral stroke or hemorrhage (mean clinical follow-up of 399 days). We performed post-CT perfusion scanning studies in 3 patients, in all of whom significant improvements in CBF and MTT were observed. Asymptomatic restenosis was documented in 4 (57%) of 7 patients. One of 7 patients underwent retreatment in the form of bypass surgery, and 1 patient underwent repeat angioplasty. Both patients, who initially underwent angioplasty alone, experienced asymptomatic restenosis. A mean posttreatment modified Rankin Scale score of 2 (range 1–6) and mean posttreatment Glasgow Outcome Scale score of 4 (range 1–5) were achieved in a mean clinical follow-up period of 399 days (range 1–840 days). Table 1 provides a summary of the results in our clinical series.

Discussion

New paradigms for the treatment of selected symptomatic patients with nonacute focal intracranial occlusions need to be investigated. Currently, no consensus exists on the optimal treatment paradigm for severe intracranial atherosclerotic disease.

Since the publication in 1985 of the International Cooperative Study of Extracranial/Intracranial Arterial Anastomosis, a certain therapeutic nihilism has been advocated for nonacute intracranial occlusions. Currently, the Carotid Occlusion Surgery Study is reinvestigating whether EC-IC bypass for indirect cerebral revascularization in patients with symptomatic ICA occlusion and ipsilateral increased oxygen extraction fraction measured by PET will reduce future ischemic stroke. However, this study does not evaluate the treatment of VA, basilar artery, or MCA occlusions and near occlusions. These ter-

Fig 7. Follow-up angiogram acquired at 14 months revealing 60% in-stent restenosis. The patient was placed on dual-antiplatelet agents and remains asymptomatic 24 months postprocedure.

ritories are conceivably most suitable for direct revascularization by means of angioplasty and stent placement. Furthermore, there are no published studies on the comparative safety and efficacy of angioplasty and/or stent surgery compared with medical therapy.¹⁹ Stenting and Aggressive Medical Management for Preventing Recurrent stroke in Intracranial Stenosis (SAMMPRIS), a prospective, randomized multicenter clinical trial funded by the National Institutes of Health/National Institute of Neurological Disorders and Stroke, is currently investigating medical compared with endovascular angioplasty and stent placement in symptomatic patients with 70–99% intracranial stenosis.

Angioplasty and stenting of acute intracranial occlusions is emerging as a viable treatment option. In fact, this technique has been shown to result in high recanalization rates with an acceptable degree of safety. Early experience at multiple centers has indicated the feasibility of intracranial self-expanding stents for acute intracranial artery occlusions. In many cases TICI revascularization scores of 2 or 3 have been successfully obtained, often exceeding the recanalization rates currently reported for other means of acute thrombolysis.²⁴

This direct revascularization approach involving angioplasty and stent placement in chronically occluded intracranial vessels has not been rigorously investigated or frequently reported. Chronically occluded arteries in the peripheral circulation such as the subclavian artery have been recanalized successfully with this method.^{4,21} Limited case reports have been published regarding recanalizing chronically occluded intracranial vessels. Thomas et al.³⁵ recently described 2 cases of chronic extracranial carotid artery occlusions recanalized by angioplasty and stent therapy. In addition, Imai et al.¹⁷ reported the suc-

cessful stenting of the intracranial petrous ICA 7 days after an atherothrombotic occlusion. The authors concluded that short-segment atherothrombotic intracranial ICA occlusions could be opened in the subacute stroke stage without distal migration of thrombi; however, no sizeable reports of direct endovascular revascularization for chronic near or complete intracranial occlusions have been published.

This case series illustrates the potential to open intracranial occlusions well outside the standard accepted time window. The efficacy of thrombolytic treatment in acute ischemic stroke is highly time dependent. Based on current recommendations, intravenous thrombolysis can be administered within 3–4.5 hours of symptom onset, intraarterial thrombolysis within 6 hours of symptom onset, and mechanical thrombolysis up to 8 hours after symptom onset.^{1,11,13,26,31}

In this case series, chronic occlusions or near occlusions were demonstrated in all patients, with a mean documented time of occlusion of 35 days. Direct cerebral revascularization was successfully achieved in all 7 patients, illustrating that there is no fixed window of opportunity and considerable variability exists for restoring CBF. The cause of occlusion—acute embolic compared with slowly progressive thrombosis from a tight stenosis—most likely plays an important role in the development of collaterals and influences the window of opportunity for revascularization. In this study, all patients were hypothesized to possess focal stenoses with progressive thrombosis and marginal distal collateral circulation.

Through a better understanding of the natural history of intracranial atherosclerotic disease,^{29,33,34,38} we now know that patients with severe intracranial stenosis and loss of cerebrovascular reserve or Stage II hemodynamic failure have a higher stroke risk.^{6,12} Evidence supports a higher stroke risk in patients with severe stenosis > 70%. The WASID Trial investigators identified subgroups of patients with the highest risk of future stroke. Patients with > 70% stenosis and recent symptoms are at particularly high risk of stroke. Risk increased linearly with greater percentage of stenosis. The probability of stroke at 1 year (95% CI) in patients with 70–79%, 80–89%, and 90–99% stenosis and 100% occlusion was 17, 31, 5, and 33%, respectively.^{6,19}

Several well-designed prospective studies have also shown a strong association between preexisting hemodynamic impairment and stroke risk.⁶ The strongest associations have been found with measurements of oxygen extraction fraction on PET and vasodilatory studies using breath-holding and transcranial Doppler measurements of blood velocity.^{6,12,19} Reduced blood flow and reactivity to acetazolamide have also been shown to be predictive of subsequent ischemic stroke in patients with ICA or MCA occlusion.²⁰

Some evidence supports a synergistic effect of embolic and hemodynamic factors. It is likely that preexisting hemodynamic impairment predisposes to ischemic stroke in patients who have an emboligenic, atherosclerotic lesion. Grubb and colleagues¹² have suggested that either hemodynamic failure predisposes to the formation of thromboemboli or that thromboemboli may cause infarction.

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tion more readily in the setting of poor collateral circulation. Others have postulated that hemodynamic failure may lead to delayed washout of microembolic material.⁵ All patients treated in this study were considered to be in these high-risk categories with recent neurological symptoms, severe intracranial stenosis > 99%, or complete occlusion. In 4 patients, acetazolamide-based CT perfusion scanning documented loss of CVR or steal, and 3 patients had characteristic clinical hemodynamic symptoms.

Clearly, not all patients with nonacute intracranial occlusions and near occlusions are candidates for direct endovascular revascularization. Patients with large strokes greater than one-third of the cerebral hemisphere or radiographic evidence of hemorrhagic transformation should not be treated. Patients with arteries thrombosed over a long segment or occluded territories with no distal collateral are also not candidates for direct endovascular revascularization.

Some patients with near occlusions or complete occlusions in whom there is good circle of Willis collateral flow may not need treatment. Rothwell et al.²⁸ have demonstrated that patients with extracranial carotid artery near occlusion may not represent a high-risk group. Reports show that benefit in these patients with extracranial carotid artery near occlusion undergoing carotid endarterectomy is marginal in the short term and uncertain in the long term.

However, the natural history of intracranial and extracranial occlusions or near occlusions may be different. Extracranial occlusions have greater capacity for the development of collateral circulation via the circle of Willis. Intracranial occlusions have less ability for the development of collateral flow. In our cases of intracranial near occlusion, the vascular territories tended to be isolated with less opportunity for circle of Willis collaterals and more reliant on leptomeningeal collaterals. This case series illustrates that as the near occlusion or occlusion approaches the target tissue, the hemodynamic consequences are more severe. For example with a M₁-segment near occlusion, the circle of Willis collaterals are not available, and the deep perforating vessels have limited potential for collateral flow.

The NASCET (North American Symptomatic Carotid Endarterectomy Trial) angiographic criteria for extracranial carotid artery near occlusions predate modern CBF perfusion technology and thus represent an anatomic definition without physiologic correlate.²⁵ The NASCET angiographic criteria for near occlusion are severe stenosis with evidence of reduced flow in the distal ICA (that is, delayed arrival of contrast into the distal ICA, evidence of collateral flow of contrast medium toward the symptomatic cerebral hemisphere from other arterial territories, or both) and evidence of narrowing of the poststenotic ICA (that is, lumen diameter similar to, or less than, the ipsilateral external carotid artery and less than the contralateral ICA). Subsequently, it has been shown that anatomical imaging studies are not sufficient to determine the hemodynamic status in individual patients.⁶ The presence of complete arterial occlusion does not reliably predict hemodynamic impairment in individual patients.^{7,27} Certain patterns of collateral flow

have been correlated with hemodynamic impairment, but the ability of these findings to identify individual patients with hemodynamic impairment has been poor.^{8,37} Thus, in patients without characteristic limb shaking or orthostatic TIAs, which can be associated with hemodynamic impairment,²² we relied on additional CT perfusion scanning data to determine the need for intervention. Treatment was reserved for unstable, symptomatic patients with chronic intracranial near occlusion or complete occlusion with steal phenomena when challenged with acetazolamide.

We present, to the best of our knowledge, the first case series on the direct revascularization of intracranial nonacute near and complete occlusions treated with angioplasty and stent therapy. The characteristics of patients amenable to such treatment include symptomatic patients with clinical or radiological evidence of hemodynamic failure and a focal segment of occlusion. This procedure is feasible with current technology. The intraprocedural complication rate is not insignificant and the complications tend to be severe. One patient experienced a perforator stroke presumably from the snow-plowing effect of the balloon angioplasty, and one patient suffered a fatal periprocedural intracerebral hemorrhage. After 30 days, there were no ipsilateral strokes at 1 year. In nearly all the patients with follow-up imaging, restenosis was demonstrated (4 of 7). All patients with restenosis were asymptomatic. Two patients underwent retreatment. The restenosis rate appears to be higher than that in other published series of intracranial angioplasty and stent placement for patients with the traditional 50–99% stenosis.^{36,40} This can be partially explained by the longer mean follow-up period of 399 days reported in this case series. In addition, a treated near occlusion or complete occlusion may have a higher restenosis rate secondary to the pathological changes in the vessel wall. The direct revascularization achieved through angioplasty and stenting in our series dramatically improved cerebral perfusion and eliminated patients' symptoms in the short term. Prudent follow-up imaging is still warranted.

Conclusions

Although conclusions drawn from this report are limited by the small number of patients and the inherent flaws of a nonindependently adjudicated retrospective case series, it does represent the largest published patient series concerning the direct revascularization with angioplasty and stenting of chronic intracranial occlusions. This review also demonstrates the technical feasibility of directly revascularizing selected, nonacute intracranial occlusions with angioplasty and stenting well beyond the arbitrary time limits. It also helps define a subgroup of patients with near occlusions or complete occlusions with hemodynamic failure who may be amenable to some type of revascularization treatment. Whether this represents a definitive solution or substantial risk reduction compared with the best medical therapy with antiplatelet agents or statins has yet to be determined by randomized prospective studies.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Manuscript submitted November 11, 2008

Accepted December 22, 2008.

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