PAPER

# Carotid Artery Stents for Blunt Cerebrovascular Injury

## *Risks Exceed Benefits*

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**Background:** Carotid stenting has been advocated in patients with grade III blunt carotid artery injuries (hereafter referred to as "blunt CAIs") because of the persistence of the pseudoaneurysm and concern for subsequent embolization or rupture.

**Hypothesis:** Carotid stenting is safe and effective for blunt CAIs.

**Design:** Analysis of a prospective database of all patients with blunt CAIs.

**Setting:** A state-designated, level I, urban trauma center.

**Patients and Methods:** In January 1, 1996, we initiated comprehensive screening for blunt CAIs with angiography based on injury patterns. Patients without contraindications receive anticoagulation therapy immediately for documented lesions. Patients with persistent pseudoaneurysms on a second angiography at 7 to 10 days after injury are candidates for stent placement.

**Results:** During the study period (January 1, 1996, to May 1, 2004), 46 patients sustained blunt carotid pseudoaneurysms; 23 (50%) underwent carotid stent placement. There were 4 complications in patients undergoing carotid stent placement: 3 strokes and 1 subclavian dissection. Follow-up angiography was performed in 38 patients (18 patients with stents who received antithrombotic agents, 20 patients who received antithrombotic agents alone); 8 patients had poststent carotid occlusion despite having received concurrent anticoagulation therapy. Carotid occlusion rates were significantly different (45% in patients with stents vs 5% in those who received antithrombotic agents alone). In the patients not undergoing stent placement, the only complication was a middle cerebral artery stroke in a patient not treated with antithrombotic therapy.

**Conclusions:** Patients who have carotid stents placed for blunt carotid pseudoaneurysms have a 21% complication rate and a documented occlusion rate of 45%. In contrast, patients treated with antithrombotic agents alone had an occlusion rate of 5%; no asymptomatic patient treated with antithrombotic agents for their injury had a stroke. Antithrombotic therapy remains the recommended therapy for blunt CAIs, but the role of intraluminal stents remains to be defined.

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CAROTID STENTING HAS been used in recent years for the treatment of carotid atherosclerotic disease.[1-4] The ability to place these stents, using endovascular techniques, has permitted extrapolation of their use to other patient populations. In patients with blunt carotid artery injuries (hereafter referred to as "blunt CAIs"), particularly grade III injuries (pseudoaneurysms), stents may be warranted in addition to anticoagulation therapy.

Carotid stents have been used in patients who have blunt CAIs with persistent pseudoaneurysms because of the concern for subsequent embolization or rupture.[5] In theory, the uncovered carotid stent acts as a filter to trap any thrombus within the pseudoaneurysm, thereby preventing subsequent embolization and stroke. The stent may also decrease flow into the pseudoaneurysm by increasing laminar flow within the stented portion of the carotid lumen itself.[6] Decreasing flow into the aneurysmal sac may then reduce any egress of blood from the sac that, in turn, may cause turbulence within the lumen. In the months following carotid stent placement, endothelial cells grow into the interstices of the stent; this may help to exclude the pseudoaneurysm from the carotid lumen and eliminate the embolic potential. The purpose of this study was to determine if carotid stenting was safe



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and effective in patients with traumatic pseudoaneurysms.

## METHODS

In January 1, 1996, we initiated comprehensive screening for blunt CAIs based on injury patterns.[7] These patients have subsequently been followed up prospectively. Four-vessel cerebrovascular angiography remains our standard screening test, and patients without contraindications are administered antithrombotic agents immediately for documented lesions. Antithrombotic medications include either anticoagulation with intravenous heparin sulfate (partial thromboplastin time goal, 40-50 seconds) or antiplatelet agents, 325 mg/d, and clopidogrel bisulfate, 75 mg/d. If no contraindications exist, patients with grade I to III injuries are enrolled in a prospective, randomized study comparing antiplatelet with anticoagulation therapy. If the patient sustained intracranial injury, solid organ injury, or complex pelvic fracture, discussion with the consulting surgeons determines the choice of antithrombotic medication. If the risk of therapy is determined to be too great because of associated injuries, patients may receive no anticoagulation therapy or antiplatelet agents during the initial days following injury. If treatment with heparin is started, the patient's therapy is transitioned to warfarin sodium during the hospital stay with an international normalized ratio goal of 2.0 to 2.5. If the patient initially receives antiplatelet agents, these are continued throughout their hospital course. At discharge from the hospital, patients are instructed to continue their warfarin or antiplatelet agent therapy for 6 months.

A second angiography is performed at 7 to 10 days after injury; patients with persistent pseudoaneurysms are considered candidates for carotid stent placement. Carotid stent placement was performed at the second diagnostic angiogram by dedicated interventional radiology attending physicians. The Magic Wallstent (Boston Scientific Scimed, Maple Grove, Minn), a self-expanding coronary stent measuring 5 to 6 mm in diameter and 31 to 47 mm in length, was used in most cases. Poststent deployment of balloon angioplasty was performed if the stent did not fully extend to its preconfigured diameter. Patients were scheduled for a third angiography 3 to 6 months following discharge from the hospital.

All patients with grade III blunt CAIs over an 8½-year period (January 1, 1996, to May 1, 2004) were evaluated. Specific variables analyzed included patient demographics, injury mechanism, Injury Severity Score, type of anticoagulation therapy, stent intervention, complications of therapy, and follow-up radiographic imaging results. Statistical analysis was performed using SAS for Windows (SAS Institute, Cary, NC). The Colorado Multi-Institutional Review Board approved this study.

## RESULTS

### DEMOGRAPHICS

During the 8½-year study period, 140 patients suffered blunt cerebrovascular injury (BCVI), of whom 46 (33%) sustained grade III (pseudoaneurysm) blunt CAIs. Most patients were men (65%) with a mean±SD age of 32±2 years and mean±SD Injury Severity Score of 27±2. Patients had the following multiple injuries: 67% with head injuries, 50% with thoracic injuries, 43% with abdominal injuries, 43% with extremity fractures, 28% with spinal fractures, and 13% with pelvic fractures. Mechanism of injury included motor vehicle collisions, motorcycle collisions, falls, and automobile-pedestrian, assault, and skiing injuries. Eight patients (17%) had associated vertebral artery injuries.

**Table 1. Treatment Stratification and Complications of Patients With Blunt Grade III Carotid Artery Injuries**

| Treatment | Patient Status Before Angiography | Type of Anticoagulation Therapy Received | Periprocedural and Postprocedural Complications |
|---|---|---|---|
| Stent (n = 23) | Asymptomatic (n = 19) | Heparin sulfate (n = 14), antiplatelet agents (n = 2), and none (n = 3) | CVA (n = 3) and subclavian dissection (n = 1) |
| | Symptomatic (n = 4) | Heparin (n = 4) | None |
| No stent (n = 23) | Asymptomatic (n = 19) | Heparin (n = 12), none (n = 4), antiplatelet agents (n = 2), and low-molecular-weight heparin (n = 1) | CVA (n = 1) (none who received anticoagulation therapy) |
| | Symptomatic (n = 4) | Heparin (n = 2), low-molecular-weight heparin (n = 1), and none (n = 1) | None |

Abbreviation: CVA, cerebrovascular accident.

### PATIENTS WITH A CAROTID STENT

Carotid stents were placed in 23 patients (50%). Treatment strategies and outcome are summarized in **Table 1**. Four patients were symptomatic at the time of blunt CAI diagnosis; the remaining 19 patients were diagnosed as having a blunt CAI after undergoing angiographic screening. Carotid stents were placed a median of 6 days (range, 2-70 days) after injury. There were 4 complications of carotid stent placement including 3 strokes and 1 subclavian dissection in asymptomatic patients.

The first patient who had a stroke underwent bilateral carotid stent placement. The patient returned to the hospital with right-sided hemiparesis 6 days after the procedure. A second diagnostic imaging showed bilateral carotid occlusion and cerebrovascular accidents. This patient who had left the hospital against medical advice was subtherapeutic while receiving warfarin therapy (international normalization ratio, 1.7); the patient was immediately started on a heparin regimen, and the neurologic deficit subsequently improved. The second patient who suffered a cerebrovascular accident underwent carotid stent placement for a right-sided internal carotid artery pseudoaneurysm. The patient suffered a right-sided cerebrovascular accident either during or immediately after the procedure. This was presumed to be embolic as a third angiogram revealed a patent carotid artery. The patient received heparin therapy following diagnosis and was discharged to inpatient rehabilitation while receiving warfarin therapy. During rehabilitation the patient had some improvement in neurologic status, but some upper extremity motor deficits persisted. The third patient who sustained a cerebrovascular accident underwent right-sided carotid stent placement on postinjury



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**Table 2. Documented Carotid Artery Patency Rates on Follow-up Angiography**

| Treatment (No. of Patients) | Type of Anticoagulation Therapy Received (No. of Patients) | Documented Carotid Artery Patency, No. of Patients* | | | Documented Patency Rate, % |
|---|---|---|---|---|---|
| | | Yes | No | Unknown | |
| Stent (n = 23) | Heparin sulfate (n = 18), antiplatelet agents (n = 2), and none (n = 3) | 10 | 8 | 5 | 55 |
| No stent (n = 23) | Heparin (n = 14), antiplatelet agents (n = 2), low-molecular-weight heparin (n = 2), and none (n = 5) | 19 | 1 | 3 | 95* |

*$P<.05$.

day 7; left-sided hemiplegia developed in the patient following the procedure, and the patient was treated with heparin. The patient's neurologic condition improved, and the patient was discharged to inpatient rehabilitation. One patient suffered a subclavian dissection during attempted carotid stent placement. The patient underwent successful stenting of the subclavian and carotid arteries.

Treatment with antithrombotic agents was started in 20 patients (87%) following the initial diagnosis of blunt CAI. All 4 symptomatic patients were treated with heparin. Of the asymptomatic patients, 14 were treated with heparin and 2 with antiplatelet agents; 3 patients received no anticoagulation therapy because of intracranial hemorrhage (Table 1).

## PATIENTS WHO RECEIVED ANTITHROMBOTIC AGENTS ALONE

Antithrombotic therapy was the sole treatment in 23 patients (50%). Four patients were symptomatic at the time of CAI diagnosis; 19 patients were diagnosed as having a blunt CAI after undergoing angiographic screening. Treatment with antithrombotic agents was started in 18 patients (78%) following diagnosis of blunt CAI (Table 1). Of the symptomatic patients, 2 were treated with heparinization—1 with low-molecular-weight heparin, and 1 received no anticoagulation therapy. Of the 19 asymptomatic patients, 12 were treated with heparin, 2 with antiplatelet agents, and 1 with low-molecular-weight heparin; 4 patients received no anticoagulation therapy because of intracranial hemorrhage.

No patient treated with antithrombotic therapy for their injury had a stroke. The only complication was a middle cerebral artery stroke in a patient not treated with anticoagulation therapy.

## CAROTID OCCLUSIONS

Angiography was performed a second time in 38 patients—18 with carotid stents and 20 without stents. In patients with carotid stents, angiography revealed 10 vessels were patent, and 8 (45%) had occluded arteries (**Table 2**). All patients with occluded arteries received anticoagulation therapy with heparin before and after carotid stent placement.

In patients treated solely with antithrombotic agents, only 1 (5%) of 20 had occlusion of this artery on second angiography. In the patient with carotid occlusion, the occlusion occurred within the first 3 days after injury despite heparinization. The occlusion rates between patients with carotid stents and those without stents were statistically significantly different using the Fisher exact test ($P=.03$).

## LONG-TERM FOLLOW-UP

Follow-up evaluation was inconsistent in this patient population. Angiography performed a second time during the acute hospitalization was achieved in almost all of the patients (mean 6 days after injury). Of the 23 patients who underwent carotid stent placement, 18 had follow-up angiography a mean±SD of 72±18 days after injury (range, 2-210 days). However, no patient receiving anticoagulation therapy alone returned for clinical follow-up or for a scheduled angiography at 3 to 6 months after injury. Ten (43%) of these patients were seen in other surgical subspecialty clinics such as orthopedics, plastic and reconstructive surgery, or neurosurgery. Patients were evaluated by subspecialty physicians between 2 and 35 months after hospital discharge; none of the 10 patients had new neurologic findings suggestive of stroke.

Patients with carotid stent placement underwent repeated angiography within 2 weeks of admission (mean, 9 days). Eighteen (78%) of these patients returned for angiography following hospital discharge at a mean of 72 days after injury.

## COMMENT

Over the past decade, there has been an explosion in the use of percutaneous transluminal arterial interventions for both traumatic injuries and atherosclerotic lesions. Placement of carotid stents was initially used for atherosclerotic occlusive disease, specifically in high-risk populations.[4,8,9] Originally thought to have too high an incidence of periprocedural complications,[10] there has been an increased enthusiasm for carotid stenting in recent years.[1-4] Most recent studies report a 95% to 98% success rate of carotid stent placement, equivalent or fewer 30-day adverse events compared with carotid endarterectomy, and restenosis rates of 2.6% to 21%.[2,3,11-15]



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The indication for percutaneous intervention for traumatic injuries is less well defined. Several isolated case reports have advocated the use of percutaneous angioplasty and stenting of carotid injuries.[16-21] Not surprisingly, these case reports represent a diverse range of pathologic conditions and symptoms. The mechanisms of injury vary from blunt trauma following strangulation or motor vehicle collisions to penetrating wounds, and diagnosis ranged from the time of injury to 15 years after trauma. Patients in these reports were both asymptomatic and had symptoms of ischemia at the time of carotid stent placement. Although most appear to have patency of the stented carotid artery documented in follow-up radiographic evaluation with angiography, computed tomographic angiography, or duplex ultrasonography, several cases of carotid artery occlusion following stent placement have been reported.[17,19] To our knowledge, no large series of carotid stents following blunt CAI, nor comparison to nonoperative-nonprocedural management, has been reported.

Our study focuses on a group of patients with blunt carotid pseudoaneurysms, of whom half were treated with early percutaneous stent intervention with antithrombotic agents while the remainder received treatment with antithrombotic agents alone. In the group undergoing carotid stent placement, 3 patients had a stroke and 1 patient sustained a subclavian artery dissection. In patients treated solely with antithrombotic agents, including heparin or antiplatelet agents, there were no ischemic neurologic events. One patient, who was not receiving antithrombotic agents because of an intracranial hemorrhage, had a middle cerebral artery stroke. The cause of strokes in the group receiving carotid stents seems to be a combination of embolic and occlusive artery disease as 2 patients had carotid artery occlusion and 1 patient had a patent carotid artery.

There was a surprisingly high incidence of carotid artery occlusion in the group undergoing stent placement. Forty-five percent of patients undergoing repeated angiography had documented occlusion of their carotid artery compared with only 5% of patients receiving antithrombotic agents alone. With reported occlusion rates in the literature confined to experience with atherosclerotic disease, aside from a few trauma-related case reports, one can only postulate the cause of this.

Several theories of responsible mechanisms could explain the alarming carotid artery occlusion rate. Injured patients have a relative hypercoagulable state after mechanical trauma[22,23] with early activation of the coagulation system; this hypercoagulability may persist for up to a month after injury.[24] Additionally, neointimal hyperplasia following carotid stent placement in acutely traumatized vessels may be increased compared with atherosclerotic disease. Some animal studies suggest the amount of restenosis and myointimal hyperplasia relates to the depth of vascular injury with damage to the internal elastic lamina.[25] The response to carotid injury and foreign body placement may be more vigorous in the younger population having traumatic injury. Hypotheses also include the idea that placement of stents across the internal and common carotid juncture for atherosclerosis increases flow through the stented area vs stents confined to the internal carotid artery for blunt injuries that may have decreased flow. There have been reports of subacute carotid thrombosis related to use of drug- eluting coronary stents.[26] However, since adverse event reporting is voluntary and the actual numbers of devices implanted questionable, the rate of such events is largely unknown.

Another possible cause for such high occlusion rates is the choice of antithrombotic agents in these patients. Initial therapy for BCVI was based on anecdotal reports of neurologic improvement with heparinization in patients suffering stroke related to BCVI.[27-29] Subsequently, intravenous heparin was thought to be the treatment of choice for those asymptomatic patients with blunt injuries.[30,31] Results from our institution suggest that treatment with antiplatelet agents have similar stroke prevention as anticoagulation with heparin.[32] To determine the ideal therapeutic regimen for BCVI, we are enrolling patients in a prospective randomized study comparing antiplatelet agents with intravenous heparin therapy in patients with grades I to III blunt CAIs. Most of the carotid stents we insert, however, were placed from 1996 to 2001, during the timeframe when heparin was accepted as the gold standard treatment. Concurrent with our decreased placement of carotid stents in the last 3 years has been the emergence of antiplatelet agent therapeutic regimens for stents. Although there is controversy regarding the standard antithrombotic therapy for arterial disease, that is, anticoagulation vs antiplatelet agents.[33-35] Recent studies advocate lifelong aspirin and at least 2 weeks of clopidogrel therapy after a carotid stent placement.[4] This evolution of antithrombotic regimens may explain the high occlusion rates found in our study.

With the unacceptable carotid stent occlusion rate we observed, the natural question becomes what is the long-term history of these lesions? In our earlier reports we demonstrated grade III blunt CAI persist in 91% of patients on angiography performed a second time 7 to 10 days after injury.[31] However, comprehensive long-term follow-up has not been accomplished as in most trauma populations. Therefore, whether these injuries heal or persist at 3 to 6 months is unknown as no patient treated with antithrombotic agents alone underwent a second angiogram after hospital discharge. There are anecdotal reports of carotid pseudoaneurysm rupture, particularly in the petrous portion of the canal leading to epistaxis.[36-41] However, aside from these limited cases located intracranially, few other reports are evident in the literature.[42,43] With so little long-term data, it is difficult to ascertain the true risk of rupture or embolic stroke.

In the past 2 decades, expectant management of contained arterial injuries has been advocated. Stain et al[44] reported on the success of selective management of 61 nonocclusive extremity arterial injuries, including 26 pseudoaneurysms. In 1991, Frykberg et al[45] demonstrated the safety of nonoperative observation in 50 patients with clinically occult injuries of major extremity arteries. Patients in this and subsequent studies[46-49] who ultimately required operative intervention presented with clinical findings within 3 months. Specific criteria for nonoperative management in this group of patients with penetrating trauma included (1) low-velocity injury, (2) minimal (<5-mm) ar-



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terial wall disruption, (3) intact distal circulation, and (4) no active hemorrhage; this approach was successful in 98% of patients without associated morbidity or mortality. In each of these studies, a proportion of those patients observed sustained postinjury pseudoaneurysms. While success with select extremity arterial injuries has been established, the cerebrovascular circulation may be different because of the sensitivity to embolic events.

Location of the patient's arterial injury clearly plays a role in the management decision tree. In these previously reported series,[44,45] most injuries caused by penetrating extremity wounds were surgically accessible. Surgically accessible extracranial carotid artery aneurysms, although rare, have usually been repaired.[50] In patients with blunt CAIs, however, the lesion is typically located in the distal internal carotid artery precluding surgical intervention.[7] For this reason, anticoagulation therapy was selected as the optimal treatment.[28,29] Subsequent studies have shown a decrease in the rate of ischemic neurologic events in patients diagnosed as having asymptomatic BCVI as well as neurologic improvement in patients suffering stroke related to a BCVI.[30,32,51]

In patients with postinjury carotid pseudoaneurysms, there is a concern that the aneurysmal sac may act as a nidus for platelet aggregation and subsequent embolization, as well as a possible source for future rupture. For this reason, the application of carotid stent technology to surgically inaccessible pseudoaneurysms appears attractive. However, to our knowledge, long-term follow-up in patients with traumatic pseudoaneurysms who were treated with anticoagulation alone has not been evaluated. Although relatively small numbers and lack of randomization limit our study, the placement of stents for these injuries was associated with an alarming carotid occlusion rate and periprocedural complications. Further understanding and evaluation of the role of appropriate concurrent antithrombotic therapy, as well as evolving stent technology including smaller delivery systems and covered stents, may improve the outcome for postinjury intraluminal carotid stents. In the interim, however, our experience to date suggests carotid stenting should be performed in selective cases and antithrombotic agent therapy remains the cornerstone of treatment for posttraumatic pseudoaneurysms.

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## DISCUSSION

**Bruce Gewertz, MD, Chicago, Ill:** The authors are to be commended for sharing less than ideal outcomes of a new therapy which, in today's tech-driven medicine, is too infrequently done. They advance 2 principal conclusions concerning the treatment of these serious carotid injuries:

1. Stenting of these lesions has a high immediate complication rate, much higher than that currently associated with dilation and stenting for atherosclerotic lesions.

2. The long-term patency of the stented arteries was remarkably only 55% and was much worse than the 95% patency of those treated with antithrombotic agents alone.

I very much appreciate the chance to pose a few questions to begin this discussion. Regarding patient selection: As I understand it, this was not a randomized study. How were patients and arteries selected for stent placement vs antithrombotic therapy? Was it the size of the pseudoaneurysm? Its appearance? Whether it was surgically accessible? Whether it served the dominant hemisphere? As an aside, if the patients were not randomized, how were they so fortunate to have equal numbers of patients in both treatment arms? Even in Chicago voting, this would be considered unusual. Regarding technique: The dilations and/or stent placements were done by their interventional radiologists. What was their experience in other carotid lesions? Did their periprocedural complication rate for atherosclerotic lesions match the good results reported across the country? Have their results changed as they developed increased experience? Importantly, did they routinely use so-called neuroprotective devices downstream to trap dislodged debris?

Regarding poststent placement drug treatment: It is accepted and referenced in the authors' excellent discussion that current guidelines for postcoronary stenting mandate at least 6 weeks of clopidogrel (Plavix) followed by lifelong aspirin therapy. While this regimen was not promulgated during the time of this study, do the authors feel such drug treatment may have made a difference in the 55% patency seen in this group?

In conclusion, I share the authors' frustration with the treatment of these lesions. They look bad, threaten worse, and are frequently associated with other thoracic, abdominal, and extremity injuries that complicate management and make anticoagulation therapy an adventure. At the University of Chicago, we have used a wide variety of therapies including direct repair, ligation of the carotid with EC-IC [exterior carotid-interior carotid] bypass, anticoagulation therapy alone, and stent placement. The results are generally good, but since the population is heterogeneous and the techniques are constantly evolving, it has not allowed a generation of a highly developed decision tree.

I think the authors' report of a large number of patients managed in a single institution advances our understanding. Most importantly, it introduces a needed note of caution in the rush to stent everything. With that said, I do believe that with increased experience and routine use of neuroprotective devices, lower postprocedural complications will argue for the use of such interventional techniques in selected patients. It remains our job to identify which ones.

**Dr Burch:** There were several factors including the size of the aneurysm and enthusiasm of both the surgeons and the interventional radiologists considered when deciding to stent a patient. None of these lesions were surgically accessible or we would have operated on them; the choice of hemisphere, non-dominant vs dominant, was



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not considered since a stroke on either side is catastrophic. As mentioned, this is not a randomized study, it was purely a coincidence that exactly half of the patients were treated with stents and the other half were not. Most of the experience of our interventional radiologists has been with the iliac and renal arteries. We have not yet used neuroprotective devices in the trauma patients. Two of the 3 cerebrovascular accidents occurred well after the angiography. The high incidence of complications with placement and a poor performance of the stent in general have caused us to shy away from this treatment in recent years. A potential issue in the study was the selection of Coumadin (warfarin) as opposed to aspirin and clopidogrel for antithrombotic treatment, recognizing now that antiplatelet therapy may be more effective in the arterial side of the vascular tree. This choice was based on class III evidence from our institution and several others that Dr Cothren demonstrated in her slides. We have embarked on a prospective randomized study comparing antiplatelet therapy to anticoagulation. It may turn out that blunt CAIs are inherently different from atherosclerotic lesions since the exposed subendothelial intima is among the most thrombotic substances in the body.

**Scott Petersen, MD, Phoenix, Ariz:** This was a nice paper and it continues to bring out that blunt CAIs are becoming more and more recognized as we aggressively screen these patients. Your group has been one of the pioneers in suggesting aggressive screening, specifically with conventional angiography. Would you want to comment on what screening may look like in, say 5 years, with the use of new multisection computed tomography scanners? Will new technology make a difference with regard to how we screen and diagnose these injuries?

**Dr Burch:** Well, we have been criticized for the aggressive use of angiography, but comparative studies to date have shown that angiography is the gold standard. I think multidetector instruments may prove effective in identifying most of those injuries, although not all of them.

**Fiemu Nwariaku, MD, Dallas, Tex:** I wonder if you noticed any difference in the prevalence of atherosclerotic disease or small artery size between the 2 patient groups. Prior studies suggest that patients who had preexisting atherosclerosis had a high incidence of angiography-related complications. So, I wonder if there are any surrogate markers for atherosclerotic disease such as advanced age or hyperlipidemia.

**Dr Burch:** No, not in our study. The patients did not stratify according to that. I think it is difficult to compare some of the recent publications, particularly in the *New England Journal of Medicine* with our work here.



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