Carotid angioplasty and stent-induced bradycardia and hypotension: Impact of prophylactic atropine administration and prior carotid endarterectomy

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Objective: **We compared the physiologic effect of selective atropine administration for bradycardia with routine prophylactic administration, before balloon inflation, during carotid angioplasty and stenting (CAS). We also compared the incidence of procedural bradycardia and hypotension for CAS in patients with primary stenosis vs those with prior ipsilateral carotid endarterectomy (CEA).**

Methods: **A total of 86 patients were treated with CAS at 3 institutions. Complete periprocedural information was available for 75 of these patients. The median degree of stenosis was 90% (range, 60%-99%). Indications for CAS were severe comorbidities (n = 49), prior CEA (n = 21), and prior neck radiation (n = 5). Twenty patients with primary lesions were treated selectively with atropine only if symptomatic bradycardia occurred (nonprophylactic group). Thirty-four patients with primary lesions received routine prophylactic atropine administration before balloon inflation or stent deployment (prophylactic group). The 21 patients with prior CEA received selective atropine treatment only if symptomatic bradycardia occurred (prior CEA group) and were analyzed separately. Mean age and cardiac comorbidities did not vary significantly either between the prophylactic and nonprophylactic atropine groups or between the primary and prior CEA patient groups. Outcome measures included bradycardia (decrease in heart rate >50% or absolute heart rate <40 bpm), hypotension (systolic blood pressure <90 mm Hg or mean blood pressure <50 mm Hg), requirement for vasopressors, and cardiac morbidity (myocardial infarction or congestive heart failure).**

Results: **The overall incidence of hypotension and bradycardia in patients treated with CAS was 25 (33%) of 75. A decreased incidence of intraoperative bradycardia (9% vs 50%; $P < .001$) and perioperative cardiac morbidity (0% vs 15%; $P < .05$) was observed in patients with primary stenosis who received prophylactic atropine as compared with patients who did not receive prophylactic atropine. CAS after prior CEA was associated with a significantly lower incidence of perioperative bradycardia (10% vs 33%; $P < .05$), hypotension (5% vs 32%; $P < .05$), and vasopressor requirement (5% vs 30%; $P < .05$), with a trend toward a lower incidence of cardiac morbidity (0% vs 6%; not significant) as compared with patients treated with CAS for primary carotid lesions. There were no significant predictive demographic factors for bradycardia and hypotension after CAS.**

Conclusions: **The administration of prophylactic atropine before balloon inflation during CAS decreases the incidence of intraoperative bradycardia and cardiac morbidity in primary CAS patients. Periprocedural bradycardia, hypotension, and the need for vasopressors occur more frequently with primary CAS than with redo CAS procedures. On the basis of our data, we recommend that prophylactic atropine administration be considered in patients with primary carotid lesions undergoing CAS. (J Vasc Surg 2005;41:956-61.)**

Carotid angioplasty and stenting (CAS) has been performed with increasing frequency for the treatment of carotid occlusive disease. The safety of CAS has been supported by the recent SAPPHIRE trial,[1] which revealed a lower incidence of stroke, death, and myocardial infarction (MI) with CAS compared with carotid endarterectomy (CEA) in high-risk patients.

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Bradycardia and hypotension have been observed after CAS with a reported incidence of 7% to 76%.[2-8] The causative factor is believed to be stimulation of the carotid sinus baroreceptors by the angioplasty balloon and intravascular stent. Stimulation of baroreceptors located in the carotid bifurcation causes an increase in afferent signals via the carotid sinus and glossopharyngeal nerves to the caudal medulla. The medulla then regulates a temporary increase in parasympathetic impulses and a decrease in sympathetic vascular tone, with resultant bradycardia and hypotension.[9] These hemodynamic changes induced by CAS may place the patient at risk for perioperative cardiac events. Atropine can block the parasympathetic impulses and has been used in an attempt to prevent or treat the resultant bradycardia and hypotension.

This study evaluated the physiologic effect of routine prophylactic atropine administration before balloon inflation or stent deployment during CAS. A comparison of the inci-

dence of procedural bradycardia and hypotension for CAS in patients with primary lesions vs those with prior CEA was also performed.

METHODS

We retrospectively reviewed prospectively collected data from a registry of 86 consecutive patients who underwent elective CAS at 3 institutions between September 2002 and April 2004. Complete data were available for 75 of these patients. All cases were performed under local anesthesia by vascular surgeons either in the angiography suite or in the operating room. Indications for intervention were greater than 50% stenosis in symptomatic patients (32%) and greater than 80% stenosis in asymptomatic patients. The degree of stenosis was determined by angiography by using North American Symptomatic Carotid Endarterectomy Trial measurement criteria.[10] The median degree of stenosis for our patient population was 90% (range, 60%-99%). Indications for CAS over CEA were severe comorbidities (n = 49), prior ipsilateral CEA (n = 21), and prior neck radiation (n = 5).

All patients were monitored and received routine fluid administration throughout the procedure. Bradycardia and hypotension during the procedure were initially treated at the discretion of the surgeon or anesthesiologist with atropine (0.5- to 1-mg doses) and additional fluid administration immediately after the event. After observing significant bradycardia and hypotension during balloon inflation in 20 initial patients with primary lesions, we began using atropine prophylactically, just before balloon inflation, during the procedure.

Twenty initial patients with primary lesions were treated selectively with atropine only after symptomatic bradycardia developed (nonprophylactic group). A later group of 34 patients with primary lesions received routine prophylactic atropine administration (0.5-1 mg) before balloon inflation or stent deployment (prophylactic group). Twenty-one patients with prior ipsilateral CEA received selective atropine treatment only if symptomatic bradycardia developed (prior CEA group), again at the discretion of the surgeon or anesthesiologist. This group of patients was noted by the authors to have a substantially lower incidence of CAS-induced bradycardia and hypotension and therefore were analyzed separately from those with primary lesions.

Vasopressors or inotropes were used as necessary if additional atropine and fluid administration were not sufficient. Dopamine was usually used in increasing doses as a first-line drug, and phenylephrine or norepinephrine was added as needed.

All patients were given oral aspirin (325 mg daily for 2 days) and clopidogrel (300 mg the day prior or 75 mg twice daily for 2 days) or ticlopidine (250 mg twice daily for 2 days) before surgery. Aspirin was continued indefinitely, and clopidogrel or ticlopidine was continued for at least 4 to 6 weeks after surgery.

Cerebral protection devices (Percusurge [Medtronic, Santa Rosa, Calif], Filterwire [Boston Scientific, Natick, Mass], or Accunet [Guidant, Santa Clara, Calif]) were used in all patients with primary lesions and in 18 of 21 patients with prior ipsilateral CEA. All stents used in this study were self-expanding stents: Wall (Boston Scientific), Precise (Cordis, Miami, Fla), or Acculink (Guidant).

For the carotid stent procedure, cases were performed under local anesthesia. A 5F sheath was placed in the common femoral artery by the Seldinger technique. An angiogram was performed of the aortic arch and great vessels via a 5F pigtail catheter. Heparin was given intravenously (100 U/kg) to achieve an activated clotting time more than 250 seconds. The affected carotid artery was selectively catheterized with a Vitek (Cook Inc, Bloomington, Ind) or angled glide catheter (Boston Scientific). A selective carotid cervical and cerebral angiogram was performed in at least two views. After confirmation of the lesion, the catheter was advanced over a 0.035-inch glide wire (Boston Scientific) into the external carotid artery, and a stiff 0.035-inch Glidewire or Amlpatz (Boston Scientific) wire was used to exchange the 5F sheath to a 90-cm 6F or 7F Shuttle (Cook Inc). The sheath was advanced into the common carotid artery proximal to the carotid bifurcation. The lesion was crossed with the protection device, and the filter was deployed or the protection balloon was inflated in a straight portion of internal carotid artery at least 2 cm distal to the lesion. If necessary, predilation was performed with a 3- or 4-mm balloon. A self-expanding stent was delivered and deployed across the lesion. Postdilation was performed with a 4- to 6-mm balloon. Care was taken not to overdilate to a diameter larger than the distal internal carotid artery. When a filter was used, a completion angiogram was performed, and the filter was recaptured. When a protection balloon was used, the column of stagnant blood in the internal carotid artery was aspirated, and the protection balloon was deflated before the completion angiogram.

Blood pressure and heart rate (HR) were monitored continuously throughout the procedure. The patients were placed in a monitored setting (intensive care unit or step-down unit) with continuous monitoring for at least 24 hours. Any intraoperative or postoperative (up to 24 hours) bradycardia (HR <40 bpm or decrease $\leq 50\%$) or hypotension (systolic blood pressure <90 mm Hg or mean arterial pressure <50 mm Hg) was recorded. Instances of tachycardia (HR >100 bpm or increase $\geq 50\%$) were also recorded. In addition, evidence of cardiac morbidity, defined as MI on the basis of cardiac enzymes or electrocardiogram or clinical evidence of congestive heart failure (CHF), was noted. Cardiac enzymes (troponin) and electrocardiogram were checked at a minimum immediately after the procedure and the morning after the procedure. Outcomes included the incidence of bradycardia, hypotension, cardiac morbidity, and the use of vasopressors to maintain adequate blood pressure. Intraoperative, postoperative (24 hours after the conclusion of the operation), and perioperative (intraoperative plus postoperative) time periods were evaluated.

Data were analyzed by using standard statistical methods. Categorical variables were compared by using the Fisher exact test, and continuous variables were evaluated by using analysis of variance and the Student t test. All continuous values are represented as mean $\pm$ SD; correla-

Table I. Demographics: comparison of all patients with primary lesions, the nonprophylactic group, and the prophylactic group

Variable	*All primary (n = 54)*	*Nonprophylactic (n = 20)*	*Prophylactic (n = 34)*
Age, y (mean ± SD)	74.1 ± 11	75.7 ± 11	73.2 ± 10
Male	74%	65%	79%
Female	26%	35%	21%
Hypertension	80%	90%	74%
Hypercholesterolemia	41%	45%	37%
CAD*	57%	70%	50%
Prior MI	32%	35%	29%
CHF	24%	25%	24%
Diabetes	32%	40%	27%
CRI†	11%	5%	15%
Smoking	59%	55%	62%
Prior stroke	19%	25%	15%
COPD‡	15%	25%	9%
Neck radiation	9%	10%	9%
Symptomatic§	26%	35%	21%
β-Blockers	56%	55%	56%
% Stenosis (mean ± SD)	86.1% ± 10%	85.4% ± 12%	86.5% ± 9%

MI, Myocardial infarction; *CHF,* congestive heart failure.
*CAD (coronary artery disease): history of myocardial infarction, angina, or electrocardiographic evidence of myocardial ischemia.
†CRI (chronic renal insufficiency): preoperative creatinine >1.5 mg/dL.
‡COPD (chronic obstructive pulmonary disease): forced expiratory volume in 1 second <1 L or forced expiratory volume in 1 second/forced vital capacity <75.
§History of transient ischemic event or stroke.

Table II. Demographics: comparison of patients with primary lesions and patients with prior carotid endarterectomy (CEA)

Variable	*All (n = 75)*	*Primary (n = 54)*	*Prior CEA (n = 21)*
Age, y (mean ± SD)	73.4 ± 10	74.1 ± 11	71.3 ± 8
Male	65%	74%	38%
Female	35%	26%	62%
Hypertension	81%	80%	83%
Hypercholesterolemia	40%	41%	39%
CAD*	57%	57%	56%
Prior MI	28%	32%	17%
CHF	19%	24%	6%
Diabetes	26%	32%	11%
CRI†	13%	11%	17%
Smoker	61%	59%	67%
Prior stroke	22%	19%	33%
COPD‡	14%	15%	11%
Neck radiation	8%	9%	6%
Symptomatic§	32%	26%	50%
β-Blockers	56%	56%	59%
% stenosis (mean ± SD)	85.6% ± 11%	86.1% ± 10%	84.5% ± 13%

MI, Myocardial infarction; *CHF,* congestive heart failure.
*CAD (coronary artery disease): history of myocardial infarction, angina, or electrocardiographic evidence of myocardial ischemia.
†CRI (chronic renal insufficiency): preoperative creatinine >1.5.
‡COPD (chronic obstructive pulmonary disease): forced expiratory volume in 1 second <1 L or forced expiratory volume in 1 second/forced vital capacity <75.
§History of transient ischemic event or stroke.

tions were considered significant at the .05 level. Calculations were performed with SPSS (version 10.0) for Windows (SPSS Inc, Chicago, Ill).

RESULTS

Mean ages and comorbidities did not differ significantly either between the prophylactic and nonprophylactic atropine administration groups (Table I) or between the primary lesion and prior CEA groups (Table II). The mean age overall was 73 ± 10 years (range, 45-93 years). Overall, there were significantly ($P < .05$) more men (65%) than women (35%). The smaller subgroup of patients with prior CEA had significantly more women (62%) compared with those who had primary lesions ($P < .01$).

Table III. Comparison of intraoperative hemodynamic changes between the nonprophylactic and prophylactic patient groups

Variable	*Bradycardia*	*Hypotension*	*Vasopressor use*
Nonprophylactic atropine	10/20 (50%)	7/20 (35%)	4/20 (20%)
Prophylactic atropine	3/34 (9%)	7/34 (20%)	6/34 (18%)
P value	<.001	NS	NS

NS, Not significant.

Table IV. Comparison of perioperative hemodynamic changes between nonprophylactic and prophylactic patient groups

Variable	*Bradycardia*	*Hypotension*	*Vasopressor use*	*Cardiac morbidity*
Nonprophylactic atropine	11/20 (55%)	9/20 (45%)	7/20 (35%)	3/20 (15%)
Prophylactic atropine	7/34 (21%)	8/34 (24%)	9/34 (27%)	0/34 (0%)
P value	<.05	NS	NS	<.05

NS, Not significant.

Table V. Comparison of intraoperative hemodynamic changes between patients with primary lesions and patients with prior ipsilateral CEA

Variable	*Bradycardia*	*Hypotension*	*Vasopressor use*
Primary	13/54 (24%)	14/54 (26%)	10/54 (19%)
Prior CEA	1/21 (5%)	1/21 (5%)	0/21 (0%)
P value	NS	.05	.05

CEA, Carotid endarterectomy.

The overall incidence of hypotension and/or bradycardia was 25 (33%) of 75. Twenty-eight percent of patients had periprocedural bradycardia, and 24% had periprocedural hypotension. Intraoperative hypotension was associated with an increased incidence of postoperative hypotension ($P < .001$). The overall incidence of periprocedural tachycardia was 10%, and this was similar in the prophylactic (9%) and nonprophylactic (10%) groups. Thirty-five percent of the nonprophylactic group required atropine for bradycardia during CAS.

A decreased incidence of intraoperative bradycardia (9% vs 50%; $P < .001$) and perioperative cardiac morbidity (0% vs 15%; $P < .05$) was observed in the prophylactic group as compared with the nonprophylactic atropine group (Tables III and IV). The mean starting HR for all patients with primary lesions was 71 bpm. There was no significant difference in mean starting HRs in the nonprophylactic group (71 ± 19 bpm) and the prophylactic group (71 ± 10 bpm). The use of perioperative β-blockade was similar in the nonprophylactic group (53%) and the prophylactic group (56%; not significant). The mean minimum intraoperative HR was lower for the nonprophylactic group than for the prophylactic group (52 ± 14 bpm vs 64 ± 17 bpm; $P < .05$). A decreased incidence of perioperative bradycardia (21% vs 55%; $P < .05$) was observed in the prophylactic group as compared with the nonprophylactic group (Table IV). In addition, there was a trend toward a lower incidence of perioperative hypotension (24% vs 45%) and vasopressor requirement to maintain adequate HR or blood pressure (27% vs 35%) in the prophylactic group compared with the non-prophylactic group (not significant; Table IV).

Perioperative cardiac events were significantly lower in the prophylactic group compared with the nonprophylactic group (0% vs 15%; $P < .05$). Two of the three patients with cardiac events in the nonprophylactic group experienced an MI diagnosed by electrocardiogram and cardiac enzymes. The remaining patient had an episode of CHF that required a prolonged stay in the intensive care unit. All three patients experienced intraoperative bradycardia, and two of the three experienced hypotension that necessitated vasopressor support. The one patient with an episode of CHF was receiving preoperative β-blocker medication, whereas the two MI patients were not.

CAS after prior CEA was associated a decreased incidence of intraoperative hypotension (5% vs 26%; $P = .05$) and vasopressor requirement (0% vs 19%; $P = .05$) as compared with patients treated by CAS for primary carotid lesions (Table V). Patients with prior CEA also had a significantly reduced incidence of perioperative bradycardia (10% vs 33%; $P < .05$), hypotension (5% vs 32%; $P < .05$), and vasopressor requirement (5% vs 30%; $P < .05$), with a trend toward overall reduced cardiac morbidity (0% vs 6%; not significant; Table VI).

Six patients with primary lesions (three prophylactic and three nonprophylactic) experienced perioperative transient neurologic deficits. Five occurred during balloon inflation of either the Percusurge balloon or the angioplasty

Table VI. Comparison of perioperative hemodynamic changes between patients with primary lesions and patients with prior ipsilateral CEA

Variable	*Bradycardia*	*Hypotension*	*Vasopressor use*	*Cardiac morbidity*
Primary	18/54 (33%)	17/54 (32%)	16/54 (30%)	3/54 (6%)
Prior CEA	2/21 (10%)	1/21 (5%)	1/21 (5%)	0/21 (0%)
P value (95% CI)	<.05	<.05	<.05	NS

CEA, Carotid endarterectomy; *CI,* confidence interval; *NS,* not significant.

balloon and rapidly resolved with deflation. Only one of these five patients had both bradycardia and hypotension, two had associated hypotension, and two had neither bradycardia nor hypotension. One transient neurologic deficit occurred when the protection device crossed the lesion. This patient's symptoms resolved by the end of the case. There was no clinical evidence of perioperative stroke in this patient series. There was no 30-day mortality.

Predictors of either bradycardia or hypotension were analyzed by univariate analysis. Cardiac comorbidities including hypertension, hypercholesterolemia, coronary artery disease, and a history of MI or coronary artery bypass grafting were not associated with an increased incidence of bradycardia or hypotension. Other comorbidities—including diabetes mellitus, chronic renal insufficiency, and smoking history—were also not predictive of hypotension or bradycardia. The only demographic predictor of bradycardia and hypotension that approximated statistical significance in this cohort of patients was symptomatic carotid disease ($P = .054$).

DISCUSSION

Our data suggest that prophylactic atropine may decrease the incidence of bradycardia, hypotension, and cardiac morbidity in patients with primary lesions undergoing CAS. Because most of our patients were asymptomatic, these findings may be more valid in the subgroup of patients with primary lesions. We observed a decreased incidence of intraoperative bradycardia and of bradycardia in the perioperative period when atropine was given prophylactically before balloon inflation during CAS. We also observed a decreased incidence of cardiac morbidity when prophylactic atropine was given in these patients with primary lesions.

Patients with prior CEA in our series had a lower incidence of intraoperative hypotension and need for vasopressors. In the perioperative period, we observed a lower incidence of bradycardia, hypotension, and vasopressor use, with a trend toward a lower incidence of cardiac morbidity in patients with prior CEA. Normally, carotid baroreceptor impulses reach the medulla via the carotid sinus nerve and the glossopharyngeal nerve. During CEA, there can be manipulation and injury to the carotid sinus nerve that lies in the carotid bifurcation. The injury and subsequent scarring may interrupt a portion of the afferent nerve fibers, thus blunting the baroreceptor reflex to stretch from the angioplasty balloon or stent. Mehta et al[11] found a higher incidence of hypertension after eversion CEA. They attributed the higher blood pressure in these patients to a loss of the baroreceptor reflex secondary to nerve injury or transection during the procedure. They also found a decreased need for procedural vasopressors in the patients who had their carotid sinus nerve transected.

Bradycardia and hypotension after CAS have been previously reported with an incidence of 7% to 76%.[2-8] Our overall incidence of hypotension and/or bradycardia was 33%. The incidence was 41% for primary lesions and 10% for recurrent lesions. The highest incidence (55%) was in the subgroup of patients with primary lesions who received selective atropine (nonprophylactic group). We did not find any significant demographic predictors for perioperative bradycardia and hypotension after CAS. It is interesting to note that although most of our patients were asymptomatic, the only factor that approached statistical significance in our series was a history of symptomatic carotid disease ($P = .054$). Other authors have found demographic factors associated with bradycardia and hypotension after CAS. Mlekusch et al,[2] in a review of 471 patients undergoing CAS, concluded that increased age and coronary artery disease predicted perioperative hypotension and bradycardia. Qureshi et al[5] reviewed 51 CAS patients and found increased age and previous MI to be predictive of CAS-induced bradycardia and hypotension. In addition, in contrast to what our data suggest, they concluded that symptomatic carotid disease was protective against, and not predictive of, postoperative hypotension.

Dangas et al[4] observed that 44% of cases with hypotension after CAS were preceded by intraprocedural hypotension. Only 10.4% of their patients without post-CAS hypotension had evidence of intraprocedural hypotension. Qureshi et al[5] found intraprocedural hypotension to be the strongest predictor of postprocedural hypotension. As in these prior studies, we also found an association between intraprocedural hypotension and postoperative hypotension. Our cohort of patients had a 13% (10/75) incidence of postoperative hypotension. Seven (70%) of the 10 patients with postoperative hypotension also had intraoperative hypotension, whereas only 3 (4%) of 68 patients without post-CAS hypotension had intraprocedural hypotension ($P < .001$).

Qureshi et al[5] paradoxically found a higher risk of postprocedural bradycardia associated with the prophylactic use of atropine in patients undergoing CAS. We did not have similar results. Although this was not statistically significant, we found a lower incidence of bradycardia in the postoperative period for the prophylactic group (15%) compared with the nonprophylactic group (30%).

It should be recognized that a well-known side effect of atropine is tachycardia. With resultant tachycardia, there is an increased cardiac oxygen demand that may result in an in-

creased incidence of MI. We did not find an increase in the incidence of operative tachycardia in patients given 0.5 to 1 mg of atropine prophylactically before balloon inflation during CAS, and we found no increase in the incidence of cardiac complications in these patients. We do, however, recognize that with a relatively small sample size, the possibility of a type II error exists.

In conclusion, prophylactic atropine administration before balloon inflation in primary CAS patients reduced the incidence of bradycardia and cardiac morbidity in our series. CAS after prior ipsilateral CEA was associated with a decreased incidence of perioperative bradycardia, hypotension, and vasopressor requirement, with a trend toward a lower incidence of cardiac morbidity. On the basis of our data, we recommend consideration of routine prophylactic atropine administration in patients with primary lesions undergoing CAS. Further studies should be performed to confirm these findings.

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INVITED COMMENTARY

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Atropine as routine prophylaxis against bradycardia has been incorporated in many carotid artery stenting (CAS) protocols as evident from several published carotid stent trials. This is remarkable considering the waning popularity of atropine as an adjunct to general anaesthesia, particularly for the cardiovascularly compromised patient. Evidence for the use of atropine in CAS either on indication or prophylactically is currently lacking.

Cayne et al have addressed this question in a retrospective study. Although patients with CAS after prior carotid endarterectomy were also included in the current analysis, the main conclusion pertains to primary CAS procedures. In the initial 20 CAS procedures, the authors noted episodes of bradycardia in 10 patients and they decided to start atropine prophylactically in the subsequent patients. With only three intraoperative episodes of bradycardia in the following 34 procedures, a statistically significant difference with the historic controls was found. In their conclusion, the authors recommend that prophylactic atropine use be considered in patients undergoing primary CAS.

There are several reasons why care should be taken before changing clinical practice on the basis of this small study. Atropine administration and the resulting tachycardia may increase the risk of cardiac complications. This is exactly why atropine has lost its appeal with anesthesiologists dealing with vascular surgery patients. In fact, current guidelines recommend the perioperative use of medication with opposite effects (β-blockers).[1]

The routine use of atropine appeared to have had no impact on periprocedural cardiac morbidity, but as 50% of the patients in the prophylactic group and 70% in the non-prophylactic group had a history of coronary artery disease, this can also be interpreted as another argument against the validity of the historic control group.

Obviously, this study carries all the drawbacks of a retrospective study. Of most importance is that a selection bias with a temporal basis is in effect. Although none of the individual baseline characteristics show a statistically significant difference in the univariate analysis of the two groups, most of the risk factors are skewed towards the nonprophylactic group. This implies that the historic control group consisted of sicker patients, which may have contributed to higher complication rates.

Furthermore, the definitions of bradycardia and hypotension are subjective. Although bradycardia appeared to be more frequent in the nonprophylactic group, there is no evidence for this having resulted in worse outcomes.

Finally, this being a historic control study, the reduction of bradycardia may also be related to changes in techniques and materials over time. With smaller size postdilatation balloons, or entirely abandoning postdilation, the risks of bradycardia may have diminished irrespective of the use of prophylactic atropine.

Cayne et al have provided persuasive arguments for reconsidering prophylactic atropine medication in CAS procedures, but it is level 4 evidence at best. In view of the potential risks of tachycardia in these particular patients and the well-established benefit of β-blockade, only a randomized trial will solve this issue.

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