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Natural History of Blunt Cerebrovascular Injury::

Experience Over a 10-year Period at a Level I Trauma Center

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Abstract

Background: Blunt cerebrovascular injury (BCVI) is associated with increased stroke and mortality risk. However, the most appropriate follow-up strategy remains uncertain.

Purpose: To better understand the natural history of BCVI and help define the most optimal timing and length of follow-up imaging.

Materials and Methods: In this retrospective HIPAA-compliant study, data from all patients treated for BCVI at a level I trauma center between April 1, 2005, and June 30, 2015, were reviewed. For patients with at least one follow-up study, time-to-event analysis was performed to assess the trend in injury evolution. Association of injury grade and injury evolution was also assessed. The Fisher exact test and multivariable logistic regression were used to evaluate association of the number of injured vessels, vessel grade, and vessel type (internal carotid artery, vertebral artery) with BCVI-associated stroke.

Results: A total of 1204 patients (800 men; mean age $\pm$ standard deviation, 45 years $\pm$ 22) with 1604 vessel injuries were evaluated. High-grade (grades 3–5) injuries were less likely to resolve (hazard ratio [HR], 0.2; $P < .001$) than low-grade injuries. High-grade injuries were more likely to progress than low-grade injuries (HR, 3.3; $P = .005$). Of the injuries that improved or resolved (343 of 419 [81.9%]), 76% (259 of 343) changed within 30 days after the initial injury, and the remaining 24% (84 of 343) changed between 30 and 90 days. Of the injuries that progressed (46 of 419 [11.0%]), 87% (40 of 46) changed within 90 days. Beyond 90 days, no improvement or resolution occurred, and only 1.4% (six of 419) of injuries progressed. Higher injury grade

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(adjusted odds ratio, 2.0 per one-grade increase [95% confidence interval {CI}: 1.6, 2.4]; $P < .001$), carotid injuries versus vertebral artery injuries (49 of 420 [11.7%] vs 35 of 667 [5.2%]; $P < .001$), and increasing number of vessels injured per patient (adjusted odds ratio, 1.6 per one-vessel increase [95% CI: 1.3, 2.2]; $P < .001$) were associated with increased risk for BCVI-related stroke.

Conclusion: Most blunt cerebrovascular injury–related changes occurred within 30 days; changes rarely occurred beyond 90 days. Follow-up imaging is therefore unlikely to be helpful beyond 90 days.

Summary

For blunt cerebrovascular injuries, follow-up imaging affects treatment decisions if performed between 30 and 90 days after injury and is unlikely to be helpful beyond 90 days.

Blunt cerebrovascular injury (BCVI) occurs in approximately 0.2%–1% of patients sustaining blunt trauma (1–5). The risk for stroke attributable to BCVI ranges from 1% to 10% overall; the risk can be as high as 33% in patients with higher-grade injuries (6–10), with mortality rates of 15%–59% (1,3,11). Since the 1990s, BCVI has gained increasing attention from trauma specialists given the adverse clinical outcomes associated with untreated BCVI. Despite this, the optimal treatment and imaging follow-up are uncertain (3,9,12–14). Most commonly accepted treatment algorithms rely on empirical antithrombotic therapies (antiplatelet medications, anticoagulation medications, or both) to treat these injuries, with endovascular intervention reserved for an exceedingly small minority of cases (3,15–17). There is no consensus, however, regarding the most appropriate follow-up imaging interval, with some authors even advocating no follow-up imaging for higher-grade injuries (18). Given these uncertainties, we conducted a retrospective review of patients with blunt trauma at a level I trauma center during a 10-year period to better understand the natural history of BCVI, with the goal of providing new insights into the most appropriate management and follow-up imaging strategies.

Materials and Methods

Patient Selection

This Health Insurance Portability and Accountability–compliant retrospective cohort study was conducted after institutional review board approval and waiver of written informed consent. Patients were identified through a retrospectively maintained database at our level I trauma center. These included all adult and pediatric patients with BCVI treated at our center from April 1, 2005, through June 30, 2015. Patients whose radiology reports or original imaging results were unavailable for review or whose clinical data were incomplete were excluded from the study (Fig 1). Screening for BCVI was done by using CT angiography at our institution if patients met the screening criteria. From 2005 to 2011, we used the modified Denver criteria. Since 2012, a slightly modified version of the Western Trauma Association guidelines has been used with one additional criterion: mandibular fracture related to high mechanism of injury.

Data Collection

BCVI grade was assigned based on manual review of radiology reports according to the Denver classification system described by Biffi et al (19) (Fig 2). All CT angiography reports were generated by members of the neuroradiology faculty, which, at the time of report creation, consisted of 10 board-certified neuroradiologists each with more than 5 years of experience. For patients whose radiology reports were conflicting or discrepant with clinical reports or whose radiology reports provided insufficient information to allow definitive assignment of injury grade, images were reviewed and grades were determined by a board-certified neuroradiologist (M.M.B., 15 years of experience) blinded to the clinical data. Grade 1 and 2 injuries were categorized as low grade, and grade 3–5 injuries were considered high grade.

Demographic information (age and sex), mechanism of injury, injured vessel, injury grade, in-hospital strokes, and vascular territory of the strokes were collected. The diagnosis of stroke was based on clinical work-up by experienced inpatient teams at our comprehensive level I stroke center. All cases were confirmed with CT, MRI, or both. Stroke was considered attributable to BCVI if the stroke occurred in the same vascular territory as the injured vessel after exclusion of other alternative diagnosis, such as direct brain contusion, global hypoxia or ischemia, cardiac arrhythmia, and neurosurgical intervention. For patients with follow-up imaging, injury evolution was evaluated. For patients with grade 3 injuries, the maximum axial dimension of the pseudoaneurysm was measured by a board-certified neuroradiologist (L.W.) with 7 years of experience (Fig 3). Temporal change of grade 3 injuries was based on change in pseudoaneurysm size or change in the Denver grade described in the radiology reports. For other injury grades, injury improvement, stability, or progression was defined by change in the Denver grade described in the radiology reports.

Statistical Analysis

Time-to-event analysis was used to analyze rates of injury improvement, resolution, or progression by vessel during follow-up. To account for variable follow-up, dropout, and imaging intervals, these outcomes were treated as interval censored, with the interval being defined by time of the current imaging examination (when the outcome was first observed) and the prior examination. The time to outcome was right censored at the last examination if not observed during follow-up. Cumulative incidence curves were calculated by using the Turnbull nonparametric algorithm to account for interval censoring. Similarly, Weibull proportional hazard regression models were used to evaluate associations of the outcomes with injury grade and medications while accounting for interval censoring. Associations were summarized as unadjusted or adjusted hazard ratios (HRs). Robust sandwich variance estimators were used to calculate 95% confidence intervals (CIs) and *P* values for these HRs to account for correlation between multiple injured vessels from the same patient. The sandwich estimator was not used for the progression outcome because of the low event rate, which leads to unstable estimates.

A linear mixed model with a random intercept per vessel and follow-up time as a linear fixed effect was used to estimate the change in pseudoaneurysm size during follow-up. Pseudoaneurysm size was log-transformed to reduce right-skewness and achieve

approximate normality based on visual assessment of quantile-quantile plots. The nonparametric bootstrap with resampling by patient was used to calculate the 95% CI and *P* value. The Fisher exact test and multivariable logistic regression were used to evaluate the association of the number of injured vessels, vessel grade, and vessel type (internal carotid artery vs vertebral artery) with BCVI-associated stroke. Throughout, two-sided tests were used, with statistical significance defined as $P < .05$. All statistical calculations were conducted with the statistical computing language R, version 3.1.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Patient Information

During the study period, a total of 47 773 patients with blunt trauma received care at our center. There were 1204 patients (2.5%) confirmed to have BCVI at CT angiography. Many patients had more than one vessel injured, which resulted in 1604 individual vessel injuries. There were 800 male patients (66.4%), and the mean age was 45 years $\pm$ 22 (standard deviation) (range, 0–98 years). Motor vehicle collision was the most common mechanism of injury, accounting for 56.1% (675 of 1204) of injuries (Table 1). Injury profile, including the number of injured vessels per patient and the maximum Denver grade, is summarized in Table 1.

There were 664 of 1604 (41.3%) vessels with grade 1 injuries, 464 of 1604 (28.9%) vessels with grade 2 injuries, 188 of 1604 (11.7%) vessels with grade 3 injuries, 271 of 1604 (16.9%) vessels with grade 4 injuries, and 19 of 1604 (1.2%) vessels with grade 5 injuries. There were 145 patients with 201 injured vessels whose CT angiography results were reviewed by one author (M.M.B.) to determine injury grade because of insufficient radiology reports. Of the 201 injured vessels, 62 (30.8%) were grade 1, 87 (43.3%) grade 2, 20 (10.0%) grade 3, 30 (14.9%) grade 4, and two (1.0%) grade 5 injuries. The proportions of low-grade and high-grade injuries were similar to those among the remainder of the cohort (149 of 201 [74.1%] vs 977 of 1403 [69.6%] and 52 of 201 [25.9%] vs 426 of 1403 [30.3%], respectively; $P = .22$).

Forty-seven of 145 patients (32.4%) did not receive antiplatelet or anticoagulation treatment, 58.6% (85 of 145) received antiplatelet treatment alone, 2.8% (four of 145) received anticoagulation treatment alone, and 6.2% (nine of 145) received both treatments. These percentages are also similar to those among the remainder of the cohort, in which 37.9% (401 of 1059) received no treatment, 55.2% (585 of 1059) received antiplatelet treatment only, 2.1% (22 of 1059) received anticoagulation treatment only, and 4.8% (51 of 1059) received both treatments ($P = .47$). Follow-up CT angiography was performed for 27.4% (55 of 201) of vessel injuries. Of these injuries, 54.5% (30 of 55) remained stable, 30.9% (17 of 55) resolved, 10.9% (six of 55) improved, and 3.6% (two of 55) progressed.

BCVI Evolution

There were 419 injured vessels for which at least one follow-up CT angiographic examination was performed. The vessels with follow-up available were more likely to be

high grade than the vessels without follow-up (71 of 173 [41%] vs 64 of 246 [26%], $P < .001$). Figure 4 summarizes injury evolution across all vessels and injury grades, and Figure 5 summarizes injury evolution by grade. High-grade injuries (173 vessels) were 61% less likely to improve (unadjusted HR: 0.4 [95% CI: 0.3, 0.5]; $P < .001$) and 77% less likely to resolve (unadjusted HR, 0.2 [95% CI: 0.1, 0.4]; $P < .001$) than were low-grade injuries (246 vessels). High-grade injuries were also significantly more likely to progress than low-grade injuries (unadjusted HR, 3.3 [95% CI: 1.4, 7.6]; $P = .005$). Most injury improvement or resolution occurred within 30 days after the initial injury (Fig 5). For example, 39% (163 of 419) of injuries improved and 23% (96 of 419) resolved within 30 days, whereas an additional 9% (38 of 419) improved and 11% (46 of 419) resolved by 90 days, with no additional changes thereafter. Low-grade injuries had only a 5% (13 of 246) progression rate compared with a progression rate of 19% (33 of 173) in high-grade injuries by 180 days (Fig 5). Only 1.2% (three of 246) of low-grade injuries and 1.7% (three of 173) of high-grade injuries showed progression beyond 90 days. The three cases of high-grade injuries that progressed after 90 days were all carotid pseudoaneurysms demonstrating only minimal size increase of 1 mm. There were 81 vessels in 69 patients with baseline pseudoaneurysms who had at least one follow-up study. The median size at baseline was 3 mm (interquartile range, 2–5 mm). On average, the pseudoaneurysm decreased in size by 5.5% per year (95% CI: –40.3%, 0.6%; $P = .10$).

Treatment and Treatment-related Responses

Of the 419 injured vessels with at least one follow-up examination, 273 (65%) were treated with aspirin, 17 (4%) were treated with clopidogrel, and 39 (9%) were treated with an anticoagulant as the highest-tier medication used. Treatment exposure stratified by injury grade is summarized in Table 2. After adjustment for injury grade, sex, and age, the use of any antiplatelet agent or anticoagulant was significantly associated with a lower risk for injury progression (adjusted HR, 0.4 [95% CI: 0.2, 0.9]; $P = .020$), although not with improvement (adjusted HR: 0.8 [95% CI: 0.5, 1.4]; $P = .43$) or resolution (adjusted HR: 1.0 [95% CI: 0.5, 1.8]; $P = .91$) of the injury (Table 3). The number of higher-tier medications (clopidogrel or an anticoagulant) was too small to permit reliable assessment of their effect separately. Only four vessels (0.9%) received interventions (stents for two, clipping for one, and embolization for one).

BCVI-related Stroke Events and Mortality

Overall, 102 BCVI-attributable strokes occurred, corresponding to a stroke rate of 8.5% (102 of 1204). The median time to stroke was 2.0 days (range, 0–12 days). The number of vessels affected and the maximum Denver grade were significantly associated with stroke for both internal carotid artery and vertebral artery injuries ($P < .001$ for each) (Table 4). In a multivariable analysis of the 1204 patients, with adjustments for sex and age, the total number of vessels (adjusted odds ratio, 1.6 per one-vessel increase [95% CI: 1.3, 2.2]; $P < .001$) and maximum Denver grade (adjusted odds ratio, 2.0 per one-grade increase [95% CI: 1.6, 2.4]; $P < .001$) were independently associated with stroke. On the basis of this model, the stroke risk from one grade 3 injury was similar to that from two vessels with grade 2 injuries (8.5% vs 7.6%), whereas the stroke risk from one grade 4 injury was similar to the risk from three or four vessels with grade 2 injuries (15% vs 12%–19%). Those with

only internal carotid artery injuries had a higher risk for stroke than did those with vertebral artery injuries only (11.7% vs 5.2%; $P < .001$). Stroke rates did not significantly differ between patients who received treatment (antiplatelet treatment, anticoagulation treatment, or both) and those who were not treated after adjustment for injury grade (adjusted HR, 0.5 [95% CI: 0.2, 1.3]; $P = .17$). A total of 472 of 1204 patients (39.2%) experienced intracranial hemorrhage, and 94 of 1204 (7.8%) patients had extracranial hemorrhage. A total of 175 of 1204 patients (14.5%) died during their hospital stay.

Discussion

The current study evaluated the natural history of blunt cerebrovascular injury (BCVI) at a single institution during a 10-year period, representing the largest BCVI cohort to our knowledge. Of the injuries that improved or resolved (343 of 419), 76% (259 of 343) occurred within 30 days after the initial injury, and the remaining 24% (84 of 343) occurred by 90 days. Of the injuries that progressed (46 of 419), 87% (40 of 46) happened within 90 days. Beyond 90 days, no additional improvement or resolution occurred, no high-grade vertebral artery injury showed progression, and only three high-grade carotid artery injuries (all pseudoaneurysms) showed only minimal (1-mm) size change beyond 90 days.

Our study results suggest that follow-up CT angiography has the highest diagnostic yield in detecting injury change within the first 30 days after the initial injury and that the yield significantly diminishes beyond 90 days to less than 1%. Thus, follow-up CT angiography has the greatest effect on treatment of BCVI when performed within 30 days of injury and has intermediate effect between 30 and 90 days; it is unlikely to change management when performed beyond 90 days, particularly in high-grade injuries.

Our results also showed that high-grade injuries were 3.3 times more likely to worsen compared with low-grade injuries. Stroke rates progressively increased with higher injury grade (odds ratio, 2.0 per one-grade increase), and carotid injuries were associated with higher likelihood of stroke compared with vertebral artery injuries (49 of 420 [11.7%] vs 35 of 667 [5.2%]; $P < .001$), which may warrant more aggressive management. In addition, there was a 1.6-fold increase in stroke risk per additional vessel injury, which has not been reported before. Stroke rates did not significantly differ between patients who received treatment and those who did not (adjusted HR, 0.5 [95% CI: 0.2, 1.3]; $P = .17$).

Previously, Wagenaar et al (18) argued that follow-up CT angiography for high-grade injuries may not be necessary at all given their low rate of resolution (7% by 11 days), which is similar to the 5% in our study. Our study builds on their findings by demonstrating that CT angiographic follow-up may be unnecessary for low- and high-grade injuries if performed beyond 90 days after injury because no injuries improved, resolved, or significantly progressed. Greater tendency for high-grade injuries to progress was similarly reported by Scott et al (7,8). However, another study by Laser et al (12) showed contradictory results. Nearly all patients in their study population with grade 3 and 4 injuries remained stable or resolved, whereas almost 20% of grade 2 injuries showed progression in their population compared with 11% in ours. The discrepancy in these injury trends may be related to differences in monitoring and treatment strategies and may also relate to their

smaller sample size (1204 patients in the current study vs 379 patients in the study by Laser et al). Furthermore, there is variability in interobserver reproducibility in confirmation of BCVI grades with use of CT angiography compared with MR vessel wall imaging techniques, particularly in lower-grade injuries; this can contribute to diagnostic differences in determination of injury grade (20).

Finally, 19% of pseudoaneurysms increased in size, which is higher than the 6% found in a study by Burlew et al (17). This variability may also reflect differences in management strategies. Our incidence of BCVI-related strokes (8.5%) is similar to previously reported rates of 1%–10% (6–9). Higher risk for stroke associated with higher-grade injuries and carotid injuries (as opposed to vertebral artery injuries) is also concordant with prior studies (6–9). Our median time to stroke was 2.0 days, with a range of 0–12 days. This is similar to prior studies by Burlew et al (21) and Cothren et al (15), in which median times to stroke were 2.0 days (range, 0–46 days) and 2.1 days (range, 0–10 days), respectively.

Our study had some limitations. The retrospective nature of the study made it difficult to control for potential confounding factors, such as sex, mechanism of injury, selection for follow-up, and duration of follow-up intervals. In addition, we could not control for effect of treatment exposure. Although we did not find a significant difference in stroke rates between patients who were treated and untreated, the potential effect of antithrombotic treatment on stroke prophylaxis is beyond the scope of the current study, given these limitations and lack of clinical insights into reasons that many patients did not receive treatment. Furthermore, there is potentially some bias from the physician-driven decisions on which patients were followed up with CT angiography (higher-grade injuries were more likely to be followed up), frequency of follow-up, and duration of follow-up. Although the time-to-event analysis methods we used accommodate variable follow-up, they do not account for biases from physicians ordering follow-up in a manner correlated with outcome (informative censoring). The current study looked at only BCVI at a single institution, but population characteristics may differ between institutions. A large multicenter trial could better account for institutional and regional variations, as well as therapeutic variations. A randomized controlled trial focused on various treatment approaches for different BCVI injury grades could also better represent the relationship between injury grade, treatment, and outcomes. Finally, our scanning protocol and screening criteria for BCVI continually evolved over the study period, reflecting developments in the trauma literature regarding BCVI screening and management, as well as improvements in CT technology that likely have affected the rate of BCVI detection.

In a single-institution retrospective study of blunt cerebrovascular injury (BCVI) during a 10-year period, most injury improvement, resolution, and progression occurred within 30 days of the initial injury and rarely occurred beyond 90 days. Follow-up CT angiography is therefore most likely to affect treatment decisions if performed between 30 and 90 days after injury and is unlikely to be helpful if performed beyond 90 days. Higher injury grade, injury of the carotid arteries versus the vertebral arteries, and increasing number of vessels injured per patient are all associated with increased risk for BCVI-related strokes.

Abbreviations

BCVI	blunt cerebrovascular injury
CI	confidence interval
HR	hazard ratio

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Key Results

- Higher Denver grade blunt cerebrovascular injury (odds ratio, 2.0 per one-grade increase; $P < .001$), carotid injuries versus vertebral artery (11.7% vs 5.2%; $P < .001$), and more vessels injured per patient (adjusted odds ratio, 1.6 per one-vessel increase; $P < .001$) were associated with increased risk for stroke related to blunt cerebrovascular injury.
- Of all injuries that improved or resolved (343 of 419 [81.9%]), 76% (259 of 343) occurred within 30 days after the initial injury.
- Of all injuries that progressed (46 of 419 [11.0%]), 87% (40 of 46) occurred within 90 days.

Figure 1:
Patient flowchart. BCVI = blunt cerebrovascular injury.

Figure 2:

Examples of each injury grade by Denver classification (arrows). **(a)** Grade 1 (sagittal CT angiography in 20-year-old man): luminal irregularity with less than 25% narrowing in the left cervical internal carotid artery (ICA). **(b)** Grade 2 (sagittal oblique maximum-intensity projection from CT angiography in 18-year-old man): intimal flap in the left cavernous ICA. **(c)** Grade 3 (axial CT angiography in 23-year-old man): pseudoaneurysm of the left cervical ICA. **(d)** Grade 4 (sagittal CT angiography in 65-year-old man): complete occlusion of the right cervical ICA. **(e)** Grade 5 (sagittal CT angiography in 20-year-old man): transection with extravasation of the left vertebral artery.

Figure 3:

Axial CT angiographic images demonstrate a pseudoaneurysm (arrows) of the left cervical internal carotid artery in a 23-year-old man at presentation (**a**), which is increased in size at 2-month follow-up (**b**). The maximum size of the pseudoaneurysm in the axial plane was measured (white line) in **b**. Measurement at initial CT (**a**) is purposely not shown to avoid obscuration of the small pseudoaneurysm.

Figure 4:

Graph shows estimated rates of injury improvement and progression during follow-up. The shaded regions are 95% pointwise confidence bands. The number at risk starts at the original n of 419 and decreases over time as patients are censored or an event occurs. Overall, 175 vessel injuries improved, 114 resolved, and 33 progressed. CTA = CT angiography.

Figure 5: Graphs show rates of improvement and progression by grade ($n = 419$ with at least one follow-up CT angiography [CTA]). The shaded regions are 95% pointwise confidence bands. Treatment exposure stratified by injury grade is summarized in Table 2. The number at risk starts at 419 and decreases over time as patients are censored or an event occurs.

Table 1:**Patient and Injury Characteristics**

Variable	Value (<i>n</i> = 1204)
Male sex	800 (66.4)
Mean age $\pm$ SD (y)	45 $\pm$ 22
Mechanism of injury	
MVC or other mode of transportation	675 (56.1)
Fall	338 (28.1)
Pedestrian	75 (6.2)
Assault	31 (2.6)
Other	85 (7.0)
No. of injured vessels per patient *	
1	870 (72.2)
2	285 (23.7)
3	31 (2.6)
4	18 (1.5)
Maximum Denver grade per patient	
1	448 (37.2)
2	339 (28.2)
3	154 (12.8)
4	247 (20.5)
5	16 (1.3)

Note.— Unless otherwise noted, values are numbers of patients, with percentages in parentheses. MVC = motor vehicle collision, SD = standard deviation.

* Out of 4 (bilateral internal carotid arteries and bilateral vertebral arteries)

Table 2:

Treatment Exposure by Injury Grade

Variable	Grade 1 (n = 662)	Grade 2 (n = 464)	Grade 3 (n = 188)	Grade 4 (n = 271)	Grade 5 (n = 19)	Total (n = 1604)
No treatment	293 (44.3)	163 (35.1)	56 (29.8)	67 (24.7)	16 (84.2)	595
Antiplatelet only	331 (50.0)	264 (56.9)	120 (63.8)	178 (65.7)	3 (15.8)	896
Anticoagulation Only	10 (1.5)	13 (2.8)	2 (1.1)	5 (1.8)	0 (0.0)	30
Both	28 (4.2)	24 (5.2)	10 (5.3)	21 (7.7)	0 (0.0)	83

Note.—Values are numbers of patients, with percentages in parentheses.

Table 3:
Multivariable Associations of Initial Injury Grade and Medications with Injury Outcomes during Follow-up

Variable	No. of Patients	Improved		Resolved		Progressed	
		Hazard Ratio*	P Value	Hazard Ratio*	P Value	Hazard Ratio*	P Value
Sex							
Male	270	Reference		Reference		Reference	
Female	149	0.8 (0.5, 1.3)	.41	0.9 (0.6, 1.5)	.78	0.7 (0.3, 1.5)	.33
Age, per 10-year increase	419	0.8 (0.7, 0.9)	<.001	0.8 (0.7, 1.0)	.016	0.9 (0.7, 1.1)	.24
Injury grade							
Low (1 or 2)	246	Reference		Reference			
High (3–5)	173	0.3 (0.2, 0.5)	<.001	0.2 (0.1, 0.5)	<.001	3.8 (1.7, 8.8)	.002
Any antiplatelet or anticoagulant used							
No	89	Reference					
Yes	330	0.8 (0.5, 1.4)	.43	1.0 (0.5, 1.8)	.91	0.4 (0.2, 0.9)	.020

Note.— Each of the three models contained sex, age, grade, and medications as the predictor variables.

* Data in parentheses are 95% confidence intervals.

Table 4:**Stroke Rates**

Variable	No. of Patients with BCVI Stroke	Percentage	P Value*
No. of injured vessels			
ICA			<.001
0	35/668	5 (4, 7)	...
1	39/407	10 (7, 13)	...
2	28/129	22 (15, 30)	...
VA			<.001
0	49/419	12 (9, 15)	...
1	32/630	5 (3, 7)	...
2	21/155	14 (9, 20)	...
ICA and/or VA			<.001
1	51/870	6 (4, 8)	...
2	40/285	14 (10, 19)	...
3	6/31	19 (7, 37)	...
4	5/18	28 (10, 53)	...
Maximum Denver grade			
ICA			<.001
0 (no injury)	35/668	5 (4, 7)	...
1	5/204	2 (1, 6)	...
2	16/166	10 (6, 15)	...
3	24/124	19 (13, 27)	...
4	19/37	51 (34, 68)	...
5	3/5	60 (15, 95)	...
VA			<.001
0 (no injury)	49/419	12 (9, 15)	...
1	7/299	2 (1, 5)	...
2	15/215	7 (4, 11)	...
3	3/45	7 (1, 18)	...
4	25/215	12 (8, 17)	...
5	3/11	27 (6, 61)	...
ICA and VA			<.001
1	7/448	2 (1, 3)	...
2	25/339	7 (5, 11)	...
3	23/154	15 (10, 22)	...
4	41/247	17 (12, 22)	...
5	6/16	38 (15, 65)	...

Note.—Data in parentheses are the 95% confidence interval. BCVI = blunt cerebrovascular injury, ICA = internal carotid artery, VA = vertebral artery.

* Test for trend between increasing group variable and BCVI stroke, excluding no injury groups.