CAROTID ARTERY TRAUMA IN MOTOR VEHICLE CRASHES: INVESTIGATION OF THE LOCAL TENSILE LOADING MECHANISM

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ABSTRACT

Blunt carotid artery injuries challenge clinicians and biomechanical researchers. Investigations leading to the mechanism of injury are limited because of the occult nature of the injury and lack of detailed medical data in most common automotive-related databases. Detailed studies by Crash Injury Research Engineering Network Centers provide a unique dataset to fully evaluate injuries and injury mechanisms. The aim of this study is to obtain carotid artery injury data from CIREN and NASS databases and delineate the injury mechanics using biomechanical experiments. Four CIREN and nine NASS cases were identified. Biomechanical experiments demonstrated that the intima is the weakest layer of the artery that responds to stretch leading to tear in a traumatic situation, and the tear occurs secondary to local tensile loading of the vessel. Stress and strain corresponding to the intimal tear are 0.84 MPa and 32%. The artery has adequate reserve strength, i.e., residual deformation and load (32% and 46%), following the initiation of yielding of the intima. The quantification of the initial intimal disruption leading to subsequent neurological deficits offers a better understanding of the injury mechanics, hitherto not reported in literature.

KEY WORDS

Carotid artery, biomechanics, injury severity, stretch mechanics, neck

THE TRUE INCIDENCE of extracranial carotid artery injury (CAI) from blunt trauma is unknown since most victims are not screened for this injury. Large series of blunt trauma patients report an incidence of 0.5 to 1.1% (DiPerna, 2002; Kerwin, 2001; Miller, 2001; Miller, 2002). These injuries are found usually after becoming symptomatic or if the severity of the injury leads to a high suspicion for CAI. The majority of traumatic extracranial CAIs is due to penetrating trauma with blunt injuries comprising from 2 to 48% of all CAIs (Fabian, 1990; Fry, 1980; Nanda, 2003). Complicating the diagnosis is the fact that many of these patients become symptomatic with an ischemic stroke many hours, and sometimes days or weeks, after the initial trauma (Fakhry, 1988; Pretre, 1994). Despite its low incidence,

mortality may be as high as 28% with long-term neurological morbidity of 26 to 45% (Hellner, 1993; Miller, 2001; Miller, 2002; Nanda, 2003; Parikh, 1997; Perry, 1980).

The mechanism of CAI has not been well characterized. The majority of injuries occur from a motor vehicle accident; however, details concerning the actual source of the injury during the accident are generally not known (Alimi, 1998; Cornacchia, 1999; Hellner, 1993; Parikh, 1997). Direct contact with the seat belt is often suggested as the mechanism of arterial damage (Mattes, 1983; Rozycki, 2002). Reddy et al. (1990) documented a fatally injured patient with traumatic internal carotid artery dissection and found that the shoulder belt pressed on the anterior cervical region. The authors suggested that the safety belt might be a cause of important traumatic carotid artery dissection. Henderson (2001) stated that cervical artery dissection is a significant cause of stroke in patients younger than 40 years; any artery in the neck may be affected, but carotid artery dissection is most common. Morbidity varies from transient to death. Henderson also noted that wearing a shoulder belt in a vehicle crash can be a mechanism of carotid artery dissection. In contrast, others have not observed this association (DiPerna, 2002). Additional reported risk factors described in patients with blunt CAI include traumatic brain injury, cervical spine fracture, aortic injury, and facial fractures (Davis, 1990; Hellner, 1993; Prall, 1998). Because evidence of direct, blunt trauma to the neck is not always present, other authors have hypothesized that hyperextension of the neck is an important etiological factor for blunt CAI (Fabian, 1990; Martin, 1991), although other authors have suggested a combination of pinching and distraction. Carr et al. (1996) summarized that injury to the carotid artery can occur by direct blow to the neck or hyperextension. After the injury, the symptoms may be mild. If the injury is not treated, the outcome can be catastrophic. They defined type 1 injury as a stretch and type 2 as a compression of the internal carotid artery. Approximately 10% have immediate symptoms, 55% have symptoms within 24 hours, and 35% have symptoms in excess of 24 hours. Brasel et al. (2002) hypothesized that the adventitia, the outer vessel layer of the carotid, has a high tolerance to stretch, and the intima, the inner layer of the vessel layer of the carotid, is least resistant to stretch. Disruption of the intima causes thrombosis (Figure 1). The postulated mechanisms of injury included a direct blow (pinching) or distraction (tension) of the artery.

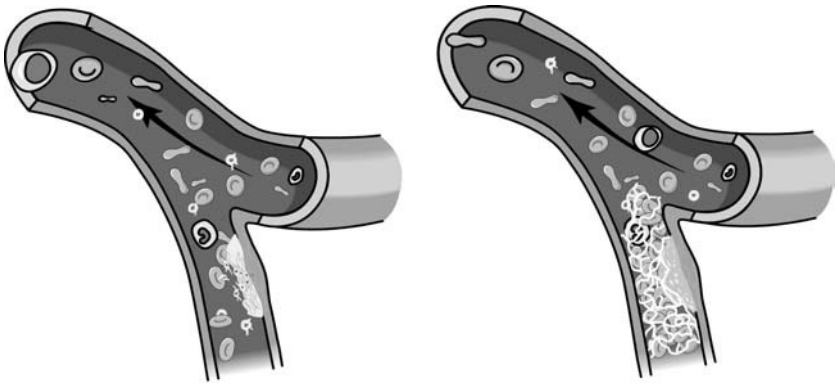

Figure 1: Disruption of intima causes thrombosis. Media disruption adds to injury.

The more detailed accident and injury specifics available to CIREN researchers provide a dataset to better evaluate injury mechanisms and to explore experimental modeling of blunt CAI. Consequently, this study was undertaken to delineate factors associated with

carotid artery trauma. The National Automotive Sampling System (NASS) database was queried for carotid artery trauma. Biomechanical experiments were conducted to quantify and identify the mechanism of injury.

METHODS

The CIREN database from 1996 to 2002 was analyzed for carotid artery trauma. This database contains detailed medical data including angiograms, CT scans, x-rays, and follow-up information in addition to impact profiles and occupant characteristics. Medical information was correlated with occupant injuries. Photographs of the vehicle, scene, crash investigation analyses, and injury interpretation by the reporting center were used for comparison with NASS data. In addition, associated injuries were used in the analyses.

In the next phase, the NASS database from 1996 to 2001 was analyzed using the following criteria: front seat outboard occupants with and without restraints including airbags. Raw data was used in the analysis. Variables extracted included occupant age, gender, height, weight, restraint use, type of injury to the carotid artery, AIS level of severity (according to the 1990 version), principal direction of force (PDoF), and vehicle factors such as body type and change of velocity. These data parallel information obtained from CIREN analyses to facilitate comparison.

For biomechanical analyses, four unembalmed PMHS isolated artery specimens were obtained by isolating the vessel at the aortic and bifurcation levels during autopsy. The specimens were prepared by opening the vessel at its mid-diameter level and fixing at the distal and proximal ends using a custom-designed gripping device. They were distracted under axial tension using a custom-designed electro-hydraulic testing device. A uniaxial load cell was used to record the applied force, and a linear variable differential transformer was used to record the displacement. Data were gathered using a digital acquisition system (DTS Inc., Seal Beach, CA, USA). The loading event was photographed using a digital video camera. The thickness of the specimen was measured by formalin fixation and staining of a cross-section. A microscope was used to facilitate measurements. Using the initial geometry (length and thickness), the stress-strain characteristics were derived from which ultimate stress and strain data were obtained. In addition, stress and strain parameters corresponding to the intimal tear were obtained by correlating digital images acquired during the loading sequence with the sensor signals. Synchronized times between the video and sensor data acquisition were used for correlation. Engineering strain and stress corresponding to the intimal tear and ultimate failure are reported.

RESULTS

Ten cases of carotid artery injury were identified in the NASS. However, one injury appeared to have double coding and, hence, was removed from the summary shown in table 1. There were five females and four male occupants. Two were right front seat passengers, and the remaining seven were drivers. With the exception of the 84-year-old female driver, occupant age ranged from 23 to 55 years. Weights ranged from 59 to 108 kg. With regard to the change in velocity, three cases were coded 59 km/h, one was coded 25 km/h, and velocity was unknown in the remaining four cases. Majority of impacts were frontal (Table 2). In only one case were both the internal and external arteries involved. Intimal tears were most common, representing five (55%) of the nine cases. However, no gross disruptions were reported. Other details of injuries and AIS coding are included in table 2.

Table 1: Summary of NASS case demographics

ID	Gender	Age (years)	Height (cm)	Weight (kg)	Seating Position
1	Male	24	175	68	Left
2	Male	37	185	108	Left
3	Female	47	175	59	Right
4	Female	84	163	59	Left
5	Female	55	170	80	Right
6	Male	23	U	U	Left
7	Male	44	178	95	Left
8	Female	34	163	84	Left
9	Female	33	175	61	Left

Table 2: Summary of CAI from NASS data

ID	Seatbelt Use	PDoF	ΔV km/h	Vehicle Body Type	AIS Code	Carotid Artery Injury Description
1	Not specific	11	59	4-door sedan	320299.3	Common, internal, not further specified
2	None	12	U	Compact utility	320299.3	Common, internal, not further specified
3	Yes	12	U	4-door sedan	320202.3 320499.2	Common, internal, intimal tear, no disruption, External, not further specified
4	Unknown	2	U	4-door sedan	320499.2	External, not further specified
5	Shoulder	12	25	4-door sedan	320202.3	Common, internal, intimal tear, no disruption
6	None	62	59	Compact pickup	320299.3	Common, internal, not further specified
7	Shoulder	-	U	Compact pickup	320202.3	Common, internal, intimal tear, no disruption
8	None	12	59	3-door hatchback	320202.3	Common, internal, intimal tear, no disruption
9	None	12	U	4-door sedan	320202.3	Common, internal, intimal tear, no disruption

Note: Case 3 had two injuries: internal and external artery injuries with an intimal tear for the internal and not further specified status for the external artery.

The CIREN database consists of information on structured vehicle cases and medical cases: structured vehicle cases represent crash aspects, and medical cases represent occupant aspects. To include a case in the CIREN study, information is input into two separate but linked databases. A case can be initiated by creating it in CIREN application or NASSMAIN application. In the CIREN application, data on only one entity, i.e., the case occupant, is entered. Collision-specific data for a CIREN case is input into NASSMAIN portion of CIREN database. One collision can result in multiple CIREN case occupants. There may be multiple case occupants in the same vehicle or occupants from two or more vehicles may become case occupants. Consequently, many CIREN medical cases may be linked to one structured vehicle case in the NASSMAIN portion of the CIREN database.

From 1996 to April 12, 2003, 1,781 structured vehicle cases are reported in the CIREN database. Of these vehicle case counts, 1,964 medical cases were logged. However, 1,501 structured vehicle cases were coded with digital information for data retrieval and

analyses. The number of cases post quality control is 806. Four carotid artery injuries were identified in this dataset. This results in 0.5%, i.e., 4 out of 806, for the closed case ensemble. One occupant in each crash sustained a carotid artery injury. Details of the subject demographics are presented in table 3. One male and three females sustained carotid artery injuries (Table 4). All were drivers, with the exception of one female front seat passenger. Frontal airbags deployed in cases 1 and 4, and no airbag was present on the passenger side in case 2. Because the principal direction of force (PDoF) was 3 o'clock in case 3, the issue of frontal airbag deployment did not arise. The change in velocity ranged from 38 to 73 km/h, with the highest velocity accounting for the front seat passenger impact. Barrier equivalent speeds, another measure of impact severity, are not used in this paper. Following is a brief description of the details of each case.

Table 3: Summary of CIREN case demographics

ID	Gender	Age (years)	Height (cm)	Weight (kg)	Seating Position
1	Male	38	180	80	Driver
2	Female	14	152	61	Right
3	Female	43	170	55	Driver
4	Female	34	165	75	Driver

Table 4: Summary of CAI from CIREN data

ID	Seatbelt Use	PDoF	ΔV km/h	Vehicle Body Type	AIS	Carotid Artery Injury Description
1	2-point belt worn, lap belt not worn	11	38	Compact 4-door sedan	4	Right internal carotid artery thrombosis with neurological deficit
2	2-point automatic shoulder belt, w/o 2-point lap belt	1	73	Compact 4-door sedan	4	Left carotid dissection with middle cerebral artery infarct and right hemiparesis aphasia
3	3-point belt	7	58	Compact 3-door coupe	4	Bilateral carotid artery dissections with left hemiparesis
4	3-point belt	80	Severe	Compact 4-door sedan	3	Left internal carotid artery dissection

Case 1: The case vehicle was traveling southbound in the outside lane of a four-lane divided highway. It was daylight and snowing. Vehicle two was in the inside northbound lane. The driver of vehicle two lost control, entered a counterclockwise rotation departing the northbound roadway, crossed the grassy median, entered the southbound roadway, and struck the case vehicle in an offset frontal mode. The case vehicle rotated clockwise, departing the roadway off the west shoulder. After approximately 140 degrees of rotation, the vehicle came to rest on the roadside facing north-northwest. The driver side airbag deployed. The driver was wearing the two-point automatic belt (not manual lap belt), indicated by a diagonal linear bruise across the left shoulder and left-superior chest. During the frontal impact, the driver moved forward toward the shoulder belt and airbag striking the left knee with the intruding knee bolster.

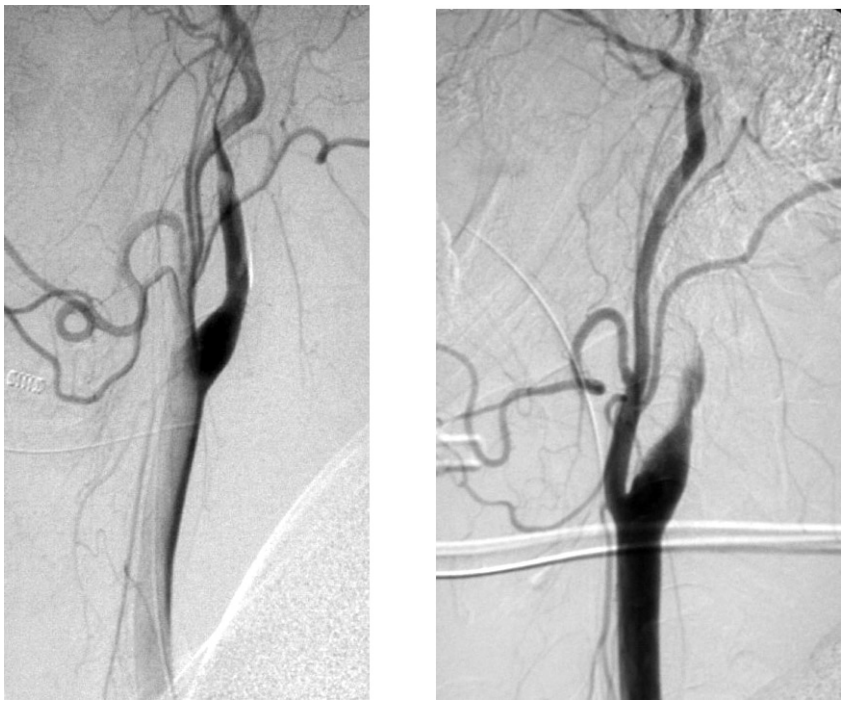

Figure 2: Lateral left common carotid artery angiogram demonstrates normal filling of the external carotid and significant narrowing of the internal carotid just above the bifurcation. Lateral right common carotid artery view also shows normal filling of the external carotid and narrowing to occlusion of the internal branch.

Knee to knee bolster contact prevented the occupant from submarining and acted as a restraint to ramp raise the torso. An abrasion to the left superior forehead occurred from contact with the headliner. The thrombosis of both internal carotid arteries (Figure 2) resulting in a neurological defect occurred secondary to hyperextension of the neck stemming from head to headliner contact. Hyperextension of the neck induces flexion to the dorsal region and concomitantly applies tension the ventral region resulting in local tension in the anterior most components including the carotid arteries. A 7-cm dent in the headliner was present behind the sun visor. The airbag resulted in a chin abrasion. Seatbelt loading induced contusions to the left shoulder, left chest, abrasions to the superior central and inferior right abdomen including right lung contusion, eighth right rib fracture, liver contusion, and right kidney contusion (AIS<3). Instrument panel contact resulted in contusion and superficial abrasion and laceration to the left wrist (AIS=1). The knee bolster contact with the patella resulted in a comminuted left patella and left femoral neck fractures (AIS=3), abrasion and laceration of the right knee, laceration to the lower left leg, and abrasion to the right lower leg (AIS=1). The left first through third metatarsal fractures occurred due to contact with the clutch pedal (AIS=2 each).

Case 2: The driver lost control of the vehicle, crossed the centerline, impacting an oncoming vehicle head-on. The right front seat passenger in the case vehicle was wearing the automatic 2-point shoulder belt (not manual 2-point lap belt) and was pinned under the dash. Although the driver airbag deployed, there was no passenger side airbag. On day 20, magnetic resonance images indicated a left carotid artery dissection with left middle cerebral artery infarct resulting in right hemiparesis and aphasia (AIS=4). Initial injuries included hangman's fracture with no cord involvement (AIS=3), larynx contusion (AIS=1), and abrasions and laceration to the throat and neck (AIS=1), due to contact with the shoulder belt.

Contact with the instrument panel contributed to the seven bilateral rib fractures (AIS=4), bilateral lung contusions (AIS=4), a right clavicle fracture (AIS=2), femur fracture (AIS=3), and left patella and right metacarpal fractures (AIS=2 each). The instrument panel and its inferior components caused multiple lacerations and contusions on both legs (AIS=1). Facial abrasions (AIS=1) occurred due to submarining or contact with the instrument panel. Contact with the intruding toe pan resulted in the three left metatarsal fractures (AIS=2). Although direct evidence of tensile loading is not clear, local stretch of the artery may have occurred secondary to the shoulder belt acting as a causal agent. Shoulder belt may have locally pinched the carotid region of the neck anatomy resulting in its tear. This is because the intimal layer still responds to a local tension.

Case 3: The case vehicle was traveling west in the westbound lane of a two-lane roadway, and vehicle two was traveling east in the eastbound lane. It was clear daylight with snow-covered bituminous roadway. The case vehicle began to fishtail and spun-out in a clockwise direction (180 deg), crossing the centerline into the path of vehicle two, resulting in an impact of the front of vehicle two with the rear of case vehicle. Both vehicles ran off the south road edge and came to rest off the roadway. The extensive intrusion of the left side quarter panel toward the front and the center of the vehicle engaged the driver seat back. The three-point belted driver sustained bilateral carotid artery dissections (AIS=4), attributed to neck bending rearward and to the left due to loading by the intruding seat back. Traumatic brain injury with loss of consciousness >24 hours (AIS=4) and a minor vertebral artery laceration with neurological deficit (AIS=3) were also attributed to these mechanisms. The cerebellum subarachnoid (AIS=3) brainstem (AIS=5) hemorrhages and a right frontoparietal subgaleal hematoma (AIS=2) were also attributed to inertial loading or contact with the steering wheel components. The left posterior rib fractures (AIS=4) and a spleen laceration (AIS=2) occurred due to contact with the intruding seat back. Similar to the previous case, local tension of the artery secondary to rearward bending may be responsible for the injury.

Case 4: The case vehicle was traveling southbound in the far left lane of a highway (five travel lanes in each direction). Vehicle two was traveling southbound in the far right lane. The driver of the case vehicle lost control, swerved to the left, traveled off the left side of the roadway, and the left front end of the vehicle struck a concrete barrier. It was daylight, and the asphalt roadway was wet. The case vehicle rotated clockwise crossing all five lanes, entered the path of the second vehicle, with the second vehicle striking the case vehicle on the right. The driver was wearing a three-point restraint, and the airbag deployed. Multiple large contusions to both sides of the cerebrum (AIS=4), cerebrum intraventricular (AIS=4) and subarachnoid (AIS=3) hemorrhages, and left carotid artery dissection (AIS=3) were identified. The artery dissection was possibly due to a rapid and extreme right bending of the neck. Various facial abrasions (AIS=1) and upper extremity trauma (AIS=1) occurred from unknown injury sources.

Biomechanical tests conducted with four carotid artery specimens had spans ranging from 32 to 42 mm. The shorter span was due to the subclavian artery above the aorta on the right side. Video analyses revealed intimal tear occurrence before ultimate failure of the artery. Mean stress and strain data corresponding to the intimal pathology were 0.84 MPa and 0.32 (Table 5). These values were 54% and 68% of the ultimate failure parameters, indicating the ductility of the vessel to axial distractive forces. This reserve strength and extensibility present following intimal tear is responsible for the artery to carry additional loads and absorb energy.

Table 5: Summary of biomechanical data

Parameter (unit)	Intimal tear	Ultimate load
Engineering stress (MPa)	0.84 ± 0.23	1.55 ± 0.23
Engineering strain (percent)	0.32 ±0.03	0.48 ± 0.06
Absorbed energy (N-m)	0.28 ±0.10	0.89 ± 0.11
Stress ratio (intimal to peak)	0.54 ± 0.12	-
Stress ratio (intimal to peak)	0.68 ± 0.08	-
Energy ratio (intimal to peak)	0.30 ± 0.09	-

DISCUSSION

The NASS and CIREN databases gather information from vehicle crashes. The latter provides significant clinical and biomechanical measures to the epidemiological process. The structure of the two databases is, therefore, different from this perspective. Furthermore, the CIREN database includes outcomes as a part of the longitudinal data collection process, a feature absent in the other database. Coding of injuries is, however, identical in the two databases as they follow the 1990 version of the AIS scheme. CIREN is a collection of self-selected specific case studies; in contrast, NASS is a randomly sampled dataset. The CIREN cases require consent from occupant(s) of one or more of the vehicles involved in a collision. The CIREN 'database' primarily consists of two linked databases. The medical data are stored in CIREN table structure, which is accessed via CIREN application. The collision data are stored in a version of NASSMAIN application. This application uses almost the same table structure and user interface of the NASS database.

Although the incidence of carotid artery injuries is small, their occult and serious nature with potentially devastating neurological consequences compels a thorough investigation to ameliorate trauma. Miller et al. (2002) reported 24 patients with blunt CAI. Thirty-three percent of these patients suffered a stroke and 25% (six) died. Similarly, high mortalities have been reported in other large series (Hellner, 1993; Perry, 1980). Due to the high morbidity and mortality with the injury, Miller et al. (2002) initiated an aggressive screening policy to identify the injury early. All victims of blunt trauma with cervical spine fractures, LeFort II or III facial fractures, Horner's syndrome, skull base fractures involving the foramen lacerum, neck soft tissue injury, or neurological abnormalities unexplained by intracranial injuries underwent cerebral angiography. In this select population, the incidence of either CAI or vertebral artery injury was 29%, suggesting these criteria are useful. Prall et al. (1998) performed angiography in all victims of blunt trauma requiring aortic angiography to evaluate possible injury to the great vessels. The incidence of CAI was 2.5%, which was ten times the incidence in their entire population of blunt trauma patients. The 1-3% risk of stroke associated with performing routine cerebral angiography precludes its widespread use as a screening tool in blunt trauma victims.

The NASS database did not identify many severe injuries although all AIS 32xxxx codes were included in the analysis. Because the injury does not manifest immediately following impact, and in some cases may remain asymptomatic, it is likely to be underreported in routine databases. In contrast, the CIREN database adds medical and engineering data to better delineate injury biomechanics. It should be noted that the weighted NASS data corresponds to 276 cases of carotid artery injury, emphasizing the need to further analyze the injury and associated characteristics. However, this estimate of the population

number may not be fully reliable owing to the extrapolation method used in the NASS study coupled with the late diagnosis and occult nature of the injury. Cases 3 and 4 both had associated traumatic brain injury that others have also found to be associated with CAI (Kerwin, 2001; Rozycki, 2002). Case 2 suffered a cervical spine fracture, also often associated with CAI (Cornacchia, 1991). Case 1 is of interest since most of the associated injuries were to the chest and abdomen; therefore, this patient would not have been considered high risk for CAI. However, the detailed information from the accident scene demonstrates a 7-cm dent in the headliner where the patient's head presumably struck as it was forced into hyperextension.

Direct blunt trauma to the neck has been postulated to be a mechanism of CAI (An, 1989; Mattes, 1983; Rozycki, 2002). We believe that localized stretching of the artery secondary to neck extension is the most plausible mechanism of CAI in motor vehicle crashes. Indeed, Martin et al. (1991) and Fabian et al. (1990) documented hyperextension in the majority of their patients with blunt CAI. The damage to the intima of the carotid artery after vessel stretching seen in our modeling study is also consistent with the type of vessel injury seen clinically. The reserve strength, i.e., residual deformation and load (32% and 46%, Table 5), following pathology to the intima present in the artery indicates that the vessel has considerable ability to accommodate subsequent external loads.

CONCLUSIONS

1. Four cases of CAI were identified in the CIREN database.
2. Nine cases were identified in the NASS database (raw data). This corresponds to 276 weighted cases.
3. Biomechanical experiments revealed that the intimal stretch/tear occurs prior to the ultimate failure of the artery, and the artery has adequate reserve strength following the initiation of yielding of the intima.
4. Axial local tensile loading of the intima is a probable mechanism of CAI.

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