Neurologic outcome after penetrating extracranial arterial trauma

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Purpose: **We undertook this study to determine factors that adversely affect outcome in patients with penetrating injury to the extracranial cerebral vasculature.**

Patients and Methods: **Medical records were reviewed for all patients who had undergone surgical intervention to treat penetrating injury to the extracranial cerebral arteries between January 1989 and December 1999. Forensic autopsy findings were also reviewed for all patients who died as a result of their injury.**

Results: **One hundred fifty-one patients with injury to the brachiocephalic artery (n = 21), common carotid artery (n = 98), or internal carotid artery (n = 32) were identified. Overall mortality was 21.2%, and stroke rate in surviving patients was 15.1%. Twenty-five of 32 deaths (78.1%) were stroke-related. Brachiocephalic artery injury was associated with the highest mortality (38.1%), and survivor stroke rate was highest in patients with internal carotid injuries (22.7%). Hemodynamic instability at presentation led to both higher mortality (30.7%) and stroke rate (19.2%). Preoperative angiography did not influence mortality or stroke rate in hemodynamically stable patients. Procedural mortality associated with arterial ligation was 45% (9 of 20 patients), and no surviving patient experienced a change in pre-ligation neurologic state. Nine patients remained neurologically intact after ligation, and 2 patients with preoperative localized neurologic deficit were unchanged postoperatively. In 131 patients, mortality after arterial repair was 17.6%, and in 5 surviving patients (5.4%) an ischemic neurologic deficit developed. Twelve of 15 surviving patients (80%) with preoperative neurologic deficit who underwent arterial repair had improved neurologic status. Cerebral infarcts were confirmed at autopsy in 23 patients; 18 infarcts were ischemic (10, repair; 8, ligation), and 5 infarcts were hemorrhagic (all, repair). No factor was identified that was predictive of ischemic versus hemorrhagic infarction in patients undergoing repair.**

Conclusions: **The presence of hypovolemic shock, internal carotid artery injury, complete vessel transection, and arterial ligation are associated with unfavorable outcome. Penetrating injury to the brachiocephalic, common carotid, or internal carotid artery should be repaired rather than ligated when technically possible. Subsequent ischemic or hemorrhagic cerebral infarction is unpredictable, but overall outcome is superior to that with ligation of the injured artery. (J Vasc Surg 2003;38:257-62.)**

Central neurologic deficit resulting primarily or secondarily from surgery is a major concern in patients with extracranial arterial trauma,[1,2] eg, injury to the brachiocephalic, carotid, or vertebral arteries. Associated combined stroke and mortality rates vary between 5% and 50%.[3-7] The usual underlying pathologic lesion is ischemic cerebral infarction caused by hypoperfusion or thromboembolism.[8-11] Arterial repair may prevent or reverse cerebral ischemia in some patients, but the issue is confounded by potential conversion of ischemic cerebral infarction to hemorrhagic infarction, with resultant neurologic deterioration or death, in other patients.[12-17]

This study was undertaken to examine an institutional experience with penetrating extracranial arterial trauma, with a view to identifying factors associated with unfavorable neurologic outcome. In patients who died of their injury, autopsy findings were reviewed to determine the incidence and possible significance of ischemic and hemorrhagic cerebral infarcts, respectively.

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PATIENTS AND METHODS

The records of all patients undergoing surgical intervention to treat penetrating trauma to the brachiocephalic, carotid, or vertebral arteries at Tygerberg Hospital between January 1989 and December 1999 were retrospectively reviewed. Injuries were classified according to anatomic location (artery injured), zone of penetration, mechanism of injury, pathologic findings (complete transection, partial transection, false aneurysm, arteriovenous fistula), and patency versus occlusion of the injured vessel. Hemodynamic instability at admission (systolic blood pressure <90 mm Hg), airway compromise (intrinsic laryngotracheal injury or extrinsic compression requiring intubation or tracheostomy), and associated injuries were recorded as present or absent. Use of preoperative angiography and type of surgical intervention were noted. Duplex ultrasound scanning was not used consistently as a preoperative investigation during the study.

Table I. Artery injured, mechanism of injury, pathologic findings, and patency, of injured vessel related to outcome

	No. of patients	*Death*		*Stroke*	
		n	*%*	*n*	*%*
Artery injured					
Brachiocephalic	21	8	38.1	1	7.7
Common carotid	98	14	14.3	12	14.3
Internal carotid	32	10	31.3	5	22.7
Mechanism of injury					
Stabbing	132	28	21.2	17	16.3
Gunshot	18	4	22.2	1	7.1
Iatrogenic	1	0		0	
Pathologic findings					
Complete transection	31	14	45.2	4	23.5
Partial transection	94	17	18.1	10	13
False aneurysm	13	1	7.7	3	25
Arteriovenous fistula	13	0		1	7.7
Injured vessel patent					
Yes	141	30	21.3	17	15.3
No	10	2	20	1	12.5

Restoration of flow through the injured vessel, whether by primary repair or interposition graft, was defined as repair, as opposed to ligation, defined as deliberate surgical interruption of flow through the injured vessel. The patients' immediate preoperative neurologic status, after any initial resuscitation required, was documented according to the classification proposed by Ledgerwood et al[15]: neurologically intact; localizing neurologic signs; coma; or shock, precluding determination of neurologic status.

Adverse outcome measures correlated with these four parameters were death, from cerebral ischemia or other cause, and stroke, with persistent neurologic deficit in surviving patients. Forensic autopsy findings in patients who died of their injury were reviewed. Cerebral infarction, when identified, was classified as ischemic or hemorrhagic.

RESULTS

Two hundred thirty-one patients with surgically confirmed penetrating trauma to the extracranial cerebral arteries were identified for the 11-year period under review. Isolated injuries to the vertebral artery (n = 49) and external carotid artery (n = 31) were managed exclusively with ligation or embolization. None of these patients died or sustained neurologic morbidity, and were accordingly excluded from further analysis. The remaining 151 patients (129 male) had a mean age of 28 years (range, 16-54 years). Intraoperative or postoperative death occurred in 32 patients (21.2%). Death was clinically stroke-related in 25 patients (78.1%) and was the result of multiple organ dysfunction syndrome in 4 patients and cardiac tamponade, cardiorespiratory arrest after displacement of an endotracheal tube, and intraoperative exsanguination in 1 patient each. Stroke, with sequella varying in severity from dysphasia and monoparesis to hemiplegia, occurred in 18 of the surviving 119 patients (15.1%).

Outcome according to site, mechanism and pathologic results of injury, and patency of the injured vessel is shown in Table I. The common carotid artery was the most commonly injured vessel, reflecting a predominance of zone II injuries (84 patients, vs 41 patients with zone I injury and 26 patients with zone III injury). Injury to the brachiocephalic artery was associated with the highest mortality but the lowest stroke rate, whereas internal carotid injury resulted in death or stroke in a high proportion of patients. Similar mortality and stroke rates were encountered for both stab and gunshot wounds. Patients with complete arterial transection fared poorer than did those with partial transection. False aneurysm was associated with a high stroke rate but low mortality. Injured vessels (5 common carotid arteries, 5 internal carotid arteries) were occluded in 10 patients. Seven occlusions were diagnosed preoperatively at angiography, and 3 were documented at exploration. Arterial occlusion was associated with complete transection in 6 patients and with partial transection in 4 patients. Death and stroke rates were similar in patients with patent and occluded injured vessels.

Seventy-five patients (49.7%) were hemodynamically unstable at admission. Twenty-three of these patients (30.7%) died, as opposed to 9 deaths (11.8%) in initially stable patients. Stroke occurred in 10 (19.2%) and 8 (11.9%) hemodynamically unstable and stable patients, respectively. Airway compromise was present in 29 patients (19.2%). Mortality was marginally higher in these patients (8 deaths [27.6%]) than in those with an intact airway (24 deaths [19.7%]), but stroke occurred with similar frequency in both groups (3 patients [14.3%] vs 15 patients [15.3%]).

One or more additional structures were injured in 106 patients (70.2%), including venous trauma (n = 77), hemothorax or pneumothorax (n = 40), peripheral nerve injury (n = 35), upper aerodigestive tract injury (n = 15), injury to other arteries (n = 12), and injury to the thoracic duct (n = 3). The only associated injury that negatively influenced outcome was injury to the upper aerodigestive tract. Seven of these 15 patients (46.7%) died. Six deaths were stroke-related; the seventh patient died after displacement of the endotracheal tube maintaining the airway.

Preoperative angiography was performed in 34 of the 76 patients who were hemodynamically stable at admission. The number of deaths (4 patients undergoing angiography vs 5 in whom angiography was not performed) and strokes (3 vs 5 patients) was similar. Of the 42 stable patients who did not undergo preoperative angiography, 25 had zone II injuries, as opposed to only 10 of 34 patients who did undergo angiography. No other factor differentiating patients undergoing and not undergoing angiography was identified.

Preoperative neurologic status and type of surgical intervention performed are shown in Table II. All but 4 of 75 patients who were hemodynamically unstable at admission could be resuscitated sufficiently that neurologic evaluation could be performed. Of 108 patients who were neurologically intact preoperatively, the injured vessel was occluded in 8. Four patients underwent primary repair

Table II. Preoperative neurologic status and nature of surgical intervention related to outcome

Preoperative neurologic status	*No. of patients*	*Surgical intervention*	*No. of patients*	*Death*		*Stroke-related death*		*Stroke*	
				n	*%*	*n*	*%*	*n*	*%*
Intact	108	Repair	95	3	3.2	3	100	5	5.4
		Ligation	13	4	30.8	3	75		0
Localizing signs	13	Repair	9	1	11.1	1	100	6*	75
		Ligation	4	2	50	2	100	2	100
Coma	26	Repair	23	16	69.9	13	81.3	4†	57.1
		Ligation	3	3	100	2	66.7	—	—
Unable to assess (shock)	4	Repair	4	3	75	1	33.3	1	100
Total	151		151	32		25	78.1	18	15.1

*Three patients improved in comparison to preoperative status.
†All improved in comparison to preoperative status.

Table III. Type of surgical intervention related to outcome

Intervention	*No. of patients*	*Death*		*Stroke*	
		n	*%*	*n*	*%*
Primary repair	99	16	16.2	11	13.3
Interposition graft (ePTFE)	30	6	20	5	20.8
Autogenous vein	2	1	50		0
Ligation	20	9	45	2	18.2

ePTFE, Expanded polytetrafluoroethylene.

Table IV. Surgical intervention related to mortality and autopsy findings

Surgical intervention	*No. of deaths*		*No. of autopsy-confirmed infarcts*	
	Total	*Clinically stroke-related*	*Ischemic*	*Hemorrhagic*
Repair	23	17	10	5
Ligation	9	8	8	0

without complication. Four underwent ligation, with 1 patient dying as a result of perioperative stroke. Of the remaining 2 patients with occluded injured vessels, 1 had localizing signs, unchanged after arterial ligation, and the other had coma and died after arterial ligation. Surgical interventions performed are detailed in Table III. Reasons for ligation were given as an emergency measure to achieve hemostasis (n = 6), distal occlusion demonstrated at angiography (n = 6), inability to obtain distal control in high internal carotid injuries (n = 4), and established neurologic deficit (n = 4). Of 20 patients undergoing arterial ligation, repair would have been technically feasible in 16 patients. Number of deaths, number of stroke-related deaths, and autopsy findings per surgical intervention are shown in Table IV. Overall, 131 patients underwent arterial repair and 20 underwent arterial ligation. Twenty-three patients (17.6%) undergoing repair died, as opposed to 9 of 20 patients (45%) who underwent arterial ligation. Stroke occurred in 5 surviving patients (5.4%) who were neurologically intact preoperatively and who underwent repair (5.4%). Neurologic deficits were all apparent immediately postoperatively. Arteries repaired (3, primary repair; 2, expanded polytetrafluoroethylene [ePTFE] grafting) were patent on duplex scans in these patients, and associated cerebral infarcts appeared ischemic on computed tomography scans in all instances. Four of the 5 patients were hemodynamically unstable at admission, but there were no other consistently associated factors.

Of 15 surviving patients with defined preoperative neurologic deficit undergoing repair, 12 (80%) improved, with return to normal neurologic function in 5 patients. Only 2 patients with preoperative neurologic deficit survived ligation, and neither demonstrated improvement in neurologic status. Autopsy findings were available for review in 27 of 32 patients who died postoperatively, and 23 examinations confirmed the presence of cerebral infarction. All infarcts in patients undergoing ligation were of the ischemic type, whereas ischemic infarction was twice as common as hemorrhagic infarction in patients undergoing arterial repair. Hemorrhagic infarction was identified at autopsy in 1 patient without preoperative neurologic deficit, in 3 patients with preoperative coma, and in 1 patient with preoperative shock. None of these 5 patients received anticoagulation therapy. Four patients had undergone primary repair, and 1 underwent ePTFE interposition grafting. Ischemic cerebral infarcts were identified in 8 patients who died after primary arterial repair. All repaired vessels were patent at autopsy in these patients. In 2 patients undergoing ePTFE grafting, death was associated with ischemic cerebral infarction and an occluded graft, respectively. Both patients were comatose preoperatively. At autopsy in the patient who died after autogenous venous interposition repair, the graft was patent and no cerebral infarction was documented. Death was ascribed to multiple organ dysfunction syndrome.

DISCUSSION

Neurologic deficit as a result of damage to the extracranial cerebral vasculature was first reported in 1552 when Ambroise Paré reported left-sided hemiplegia after ligation of a lacerated right common carotid artery in a duelist.[7]

With development of modern surgical techniques, repair, when technically feasible, became the accepted method of treatment. This approach was questioned by Cohen et al,[14] who postulated that repair might be detrimental in some patients by converting ischemic cerebral infarct to hemorrhagic cerebral infarct. Bradley[12]added support to this postulation in reporting 2 patients with neurologic deficit who died after repair and who were found to have hemorrhagic cerebral infarcts at autopsy. Since then, numerous reports have been published for surgical repair[5,7,17-20] and against surgical repair[1,12,13,15-17,21] of accessible injury in patients with fixed preoperative neurologic deficit.

We undertook this study to address the issue and in addition to identify general factors associated with adverse outcome after penetrating trauma to the extracranial cerebral vasculature. Data for 6 patients who received treatment of blunt extracranial arterial trauma during the study were excluded from analysis because of the confounding effect of multiple concomitant injuries. This provided a study population with relatively pure cervicothoracic trauma.

Stab wounds predominated. Gunshot injuries were not associated with a higher rate of stroke or mortality. These patients may represent a selected group, because patients with more serious gunshot injuries may not have survived to hospital admission. Indeed, the entire study population probably represents a selected group with respect to outcome, inasmuch as an unknown number of patients with similar injuries are likely to not have survived to hospital admission. The high mortality associated with injury to the brachiocephalic artery can be attributed to difficulty in achieving control of bleeding, whereas the lower stroke rate in this group probably reflects collateral flow via the external carotid and subclavian arteries, affording cerebral protection during repair. The high stroke and mortality rates associated with internal carotid artery injuries were probably related to absence of collateral flow when this vessel is damaged or clamped, especially in patients with an incomplete circle of Willis,[22,23] as well as inaccessible bleeding in distal injuries.

Before 1994 a policy of mandatory neck exploration for all wounds penetrating the platysma muscle was adhered to at our institution[24]; thereafter a policy of selective exploration, as suggested by others,[1,3,5,11,25-28] was followed. The subsequent more liberal use of angiography did not, however, lead to decreased mortality or stroke rate compared with not using angiography. This experience differs from that of others,[5,19,26] and appears to be due to a predominance of the more prognostically favorable zone II(common carotid artery) injuries in patients not undergoing arteriography. Since the study, selective neck exploration continues to be the policy of this unit, based on findings at arteriography in qualifying patients with zone I and III injuries and at duplex ultrasound scanning in patients with zone II injuries. Patients who qualify for arterial imaging have one “hard” sign or more than one “soft” sign of arterial injury. This limits the number of unnecessary neck explorations and facilitates planning of operative access.

Synthetic repair with ePTFE grafting performs well in this setting.[6,21] In this study, slight increases in mortality and stroke rate were associated with ePTFE repair, possibly reflecting that synthetic prostheses were generally used in the setting of more extensive arterial damage. Two thrombosed ePTFE grafts, as demonstrated at autopsy, could have led to ischemic cerebral infarction, but both patients were comatose at presentation and stroke could have occurred preoperatively.

Patients undergoing repair generally benefitted, compared with patients who underwent ligation. Neurologic improvement was most pronounced in the group with localizing neurologic deficit, but even in the group with coma, some patients improved after revascularization. The pathologic basis for this improvement centers on the principle of reperfusion of marginally perfused cerebral tissue. Better neurologic outcome is the result of recruitment of this ischemic penumbra to normally functioning neurologic tissue.[8-10]

Revascularization appears to be of overall benefit, but whether this is true for all patients is uncertain. To answer this question it is necessary to examine the group of patients who experienced neurologic deterioration after repair and those patients who underwent repair but died. Surviving patients with postoperative neurologic deterioration all had normal neurologic function before repair. Search for a common denominator in this group revealed only hemodynamic instability at admission in 4 of 5 patients. Intraoperative events appear to have been responsible for stroke in these patients. Hypoperfusion during clamping and repair in patients with an incomplete circle of Willis or a thromboembolic event during surgical exposure or control could have resulted in ischemic infarction. Hemorrhagic infarcts[12,14,15,29] do not appear to have had a role in the postoperative neurologic deterioration in this group of patients. Policy in this unit is now aimed at limiting intraoperative stroke by adhering to a basic operative strategy. Access is gained, as needed, with sternotomy or standard anterior sternocleidomastoid incision. High internal carotid artery injuries require division of the posterior belly of the digastric muscle and the mouth-open position,[30] enlarging the space behind the mandible. In stable patients the lesion is approached with meticulous dissection, early wide potential control, and heparinization before clamping. The key to preventing thromboembolism is to arrest prograde flow as early as possible. In unstable patients this may not always be possible, with active bleeding necessitating local pressure to control bleeding, thereby increasing risk for clot dislodgment. In these patients stable local pressure should be maintained at the site of bleeding while expeditious proximal and distal control is attempted. If the extent of associated injuries or active bleeding precludes heparinization before clamping, systemic heparin should still be administered after hemostasis has been achieved. Shunting[3,27,31] may be considered in patients with shock, internal carotid artery injury, and stable patients with low stump pressure, but we generally reserve shunting, when technically possible, for patients with preoperative neuro-

logic deficit in whom extensive repair is foreseen. Although shunts may decrease duration of ischemia, they may inherently increase thromboembolic events. All visible clot should be gently removed with a vascular forceps, together with proximal and distal flushing. Injuries should be debrided to healthy intima, and repair should be flawless, with no exposed thrombogenic surfaces remaining. Patients should be routinely prepared and draped for saphenous vein harvest, although synthetic interposition grafting is often mandated by size discrepancy in common carotid artery and brachiocephalic artery injuries. Quality control with duplex Doppler scanning or completion angiography can be considered,[25,32] but for logistical reasons these methods are still not routinely used in this unit.

Occlusion of an injured carotid vessel was not associated with inferior survival or neurologic outcome in this study. Repair, even in this context, appears to be beneficial. After achieving potential control proximally and distally, together with heparinization, visible thrombus should be carefully removed. If backbleeding from the distal injured vessel is observed, repair is indicated. In the absence of backbleeding, distal thromboembolectomy should not be attempted, and the procedure is best terminated with proximal and distal ligation of the injured carotid vessel. This policy can be used regardless of patient preoperative neurologic status or the mechanism of injury, blunt trauma included.

Autopsy in the 23 patients who died after repair revealed 10 ischemic infarcts and 5 hemorrhagic infarcts as the cause of death. The small group of 5 patients who died as a result of hemorrhagic infarct raises the question as to whether repair in some patients can cause progression of neurologic damage by converting ischemic infarct to hemorrhagic infarct. The proposed pathologic mechanism is based on the phenomenon of vascular discontinuity, especially of venules, caused by ischemic anoxia.[8-10] Reperfusion of these areas leads to macroscopic hemorrhagic infarction. The extent of injury depends on the extent and duration of ischemia of the tissue before reperfusion takes place. In elective surgery, cerebral reperfusion injury is well described after carotid endarterectomy.[33,34] In the small number of patients with hemorrhagic infarct after repair, no common denominator could be found that was predictive of this complication. The evolution of hemorrhagic infarct after repair probably is multifactorial. Factors that may contribute include those that influence extent and duration of preoperative ischemia, eg, completeness of the circle of Willis; delayed presentation; presence, duration, and degree of preoperative shock; and duration of clamping. Use of anticoagulation and the presence of coagulopathy associated with major blood loss and transfusion may also have an effect. The favorable results associated with repair in all categories of patients in this study suggest that repair should be attempted whenever possible, regardless of the threat of hemorrhagic infarction, because this occurs rarely and unpredictably. This should be done as expeditously as possible to limit duration of cerebral ischemia. Use of agents commonly administered to prevent or minimize reperfusion injury elsewhere, eg, mannitol and calcium channel blockers, may also be considered.[34,35]

Toward the end of the study we selectively treated cervicothoracic arterial injury in some patients (not included in the study) with endovascular stent grafts.[36] Five proximal common carotid artery injury and one distal internal carotid artery injury were successfully treated by this means. No early or late neurologic complications occurred over mean follow-up of 9 months. This method shows early promise, especially in patients in whom surgical access is a source of considerable morbidity. Stent-graft treatment is now considered in selected patients with a proximal lesion (requiring sternotomy) or distal internal carotid artery injury. We regard active bleeding, intraluminal clot visible on angiograms, compression symptoms caused by hematoma, concomittent injuries needing exploration, and infected wounds as relative contraindications. Stents, together with expertise for placement, ideally should be available at diagnostic angiography. This therapeutic method may in future be more extensively applied in selected patients, especially with newly developed adjunctive neurologic protection devices.[37,38]

We conclude that, in management of extracranial cerebrovascular injury, ischemic cerebral infarct occurs more commonly in the presence of hypovolemic shock, internal carotid artery injury, complete vessel transection, and arterial ligation. The mainstay of treatment should be early and aggressive resuscitation and revascularization. Attention should be paid to technical aspects of repair as well as cerebral protection to avert unnecessary ischemic infarction. The sporadic occurrence of hemorrhagic infarction after repair cannot be predicted and should not deter the surgeon from adopting this approach as the treatment of choice. Arterial ligation should be considered only for distal arterial occlusion (absence of backbleeding), unattainable distal control, and in the patient with multiple trauma in whom instability precludes extensive surgery.

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