Peripheral Arterial Injuries:

A Reassessment

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Ninety-four patients with peripheral arterial injuries were subjected to acute repair, negative exploration, or late repair of the complications of the arterial injury (false aneurysm, A-V fistula, and/or limb ischemia). The causes of failure after acute injury include extensive local soft tissue and bony damage, severe concomitant head, chest or abdominal wounding, stubborn reliance on negative arteriograms in patients with probable arterial injury, failure to repair simultaneous venous injuries, or harvesting of a vein graft from a severely damaged extremity. There is a positive correlation between non-operative expectant treatment and the incidence of late vascular complications requiring late arterial repair. Delayed complications of arterial injuries occurred most frequently in wounds below the elbow and knee.

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DIRECT REPAIR of human arterial injury with end-to-end union of a femoral artery was performed by Murphy in 1897.[7] Limb salvage after major arterial injury posed a problem through World War II. DeBakey and Simeone reported an amputation rate of 40% in 2,471 battle injuries involving arteries.[8] Direct arterial repair was initiated and extensively used during the Korean conflict, and the amputation rate dropped to 13%.[15] The failure rate of arterial injury repair has remained virtually static since the Korean experience.[9,12,17,24,26,33,38] As in any area of endeavor, periodic reassessment of results may identify the factors which prevent improvement. Accordingly, we have undertaken this review of our recent experience with arterial injury in order to delineate these factors. We have felt that a critical analysis of patients with failed repairs would be particularly enlightening.

There have been several excellent reviews of the diagnosis and management of acute arterial injuries.[9,12,17,20,24,30,36] However, late complications of missed arterial injuries (false aneurysm and arteriovenous fistula) indicate that the acute management of arterial injuries is not yet optimal.

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Clinical Material

All patients undergoing surgery for suspected or proven arterial injuries or their complications between 1972–1975 were reviewed. The patients were seen by the General Surgery Service at the University of Arkansas for Medical Sciences either acutely or in consultation with the Orthopedic Surgery Service. The group was comprised of 94 patients who were divided into one of the following three categories: 1) Those undergoing acute repair, 2) those undergoing negative exploration, 3) those undergoing late reconstruction for the complications of a missed arterial injury. Patients were analyzed with regard to mechanism of injury, site of injury, extent of soft tissue damage, presence of associated fractures, presence of associated major venous injury and its repair and the harvest site of autogenous vein graft. Patients undergoing negative explorations were analyzed relative to the site of injury and complications directly related to the exploration. Patients with complications of missed arterial injury were reviewed with regard to method of initial management and length of delay before definitive repair.

There were 70 males (75%) and 24 females (25%) in the

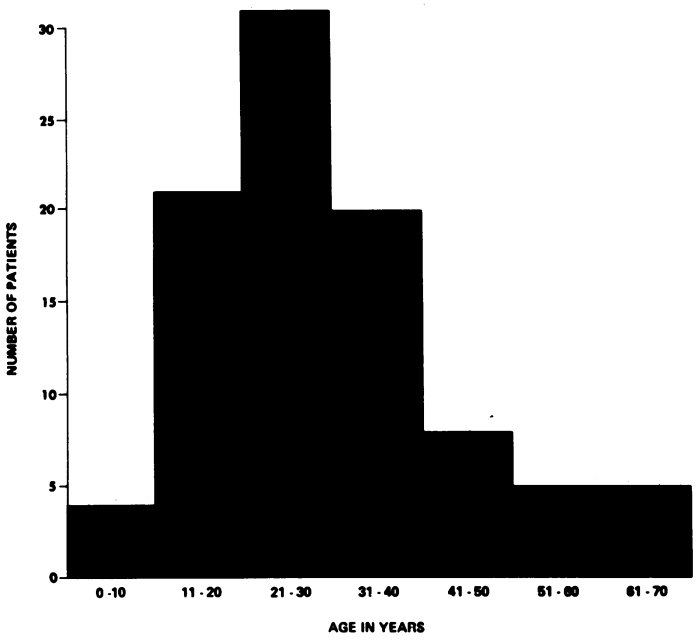

FIG. 1. Age distribution of patients.

group. The mean age was 30 years with a range of 4–68 years. The age distribution is seen in Fig. 1.

Acute reconstruction accounted for 49% of the operations performed, followed in order by negative exploration and later arterial reconstruction (Table 1).

Mechanisms and Sites of Arterial Injuries

The mechanism of injury in the 94 patients reviewed is seen in Table 2. Penetrating gunshot wounds were divided at a muzzle velocity of 1800 feet/second because of the recognized blast injury effect of missiles at higher muzzle velocities.[1,2,38] The greater blast effect can inflict additional soft tissue injury locally and can result in arterial contusion for some distance from the local injury site. Soft tissue damage and arterial contusion may influence the success of arterial repair. The preponderance of small arms injuries (44%) reflects the ubiquitous nature of the inexpensive, low velocity handguns available (Table 2).

The mechanism of non-gunshot wounds-penetrating injuries includes stab wounds and major lacerations. In addition, there were two projectile injuries resulting from metal missiles propelled from a lawnmower and a chain saw.

TABLE 1. *Categories of Patients*

Category	No. Patients	Per cent
Acute Repair	46	49
Negative Exploration	35	37
Late Repair	13	14
Total	94	100

Blunt trauma and shotgun injuries were included together as both result in significant and often extensive soft tissue damage. These were further subdivided with regard to presence or absence of associated fractures.

Iatrogenic injuries included only those which required reconstruction in the operating room. There were four injuries resulting from cardiac catheterization and two successfully managed tracheo-innominate artery fistulas.[37] Excluded from cardiac catheterization injuries were those instances where a surgeon was called to perform thrombectomy or to revise lateral arteriorrhaphy in the catheterization laboratory. These four arterial catheterization injuries were the only ones seen during a period in which approximately 900 such procedures were performed. We have seen no acute injuries or late complications of arteriography during this period in which 800 vascular radiographic procedures have been performed.

The site of injury or the major vessel suspected of injury is depicted in Table 3. Each vessel is further subdivided in Figs. 2 and 3 as to the percent of injuries which were subjected to acute repair, negative exploration, or late reconstruction.

Brachial and femoral arteries were injured more frequently than were vessels proximal or distal to these sites.

Acute arterial repair was performed in all proximal vessel injuries (innominate, subclavian, axillary, and iliac). The carotid vessels had the highest incidence of negative explorations (83%), and this reflects our reluctance to manage penetrating wounds of the neck expectantly. Trauma to the forearm and calf arteries (radial-ulnar and tibio-peroneal) was associated with the highest incidence of missed injuries. One-third of radial-ulnar and four-fifths of tibio-peroneal arterial injuries required late reconstruction. There were no negative explorations of these vessels.

Methods of Repair and Results

Group I—Acute Repair

The methods for acute repair are depicted in Table 4. Resection and end-to-end anastomosis was performed in

TABLE 2. *Mechanism of Injury in 94 Patients*

	No. Patients	Per cent
Low velocity GSW*	41	44
High velocity GSW	17	18
Penetrating-non-GSW	14	15
Blunt or shotgun–without fracture	7	7
Blunt or shotgun–with fracture	9	10
Iatrogenic	6	6
Total	94	100

* Less than 1800 feet/sec muzzle velocity

TABLE 3. *Site of Injury or Suspected Injury in 94 Patients*

Site	No. Patients	Per cent
Carotid	24	26
Innominate-axillary	7	7
Brachial	12	13
Radial-ulnar	6	6
Iliac	3	3
Femoral	29	31
Popliteal	8	9
Tibio-peroneal	5	5
Total	94	100

45%, and autogenous vein graft was used in 31%. Ligation was performed in 5 patients—two radial arteries, one innominate artery (tracheo-innominate artery fistula), one profunda femoris, and one distal tibio-peroneal. The only complication was in a young fireman with extensive soft tissue and bony injury to the foot and ankle which required amputation at a later date.

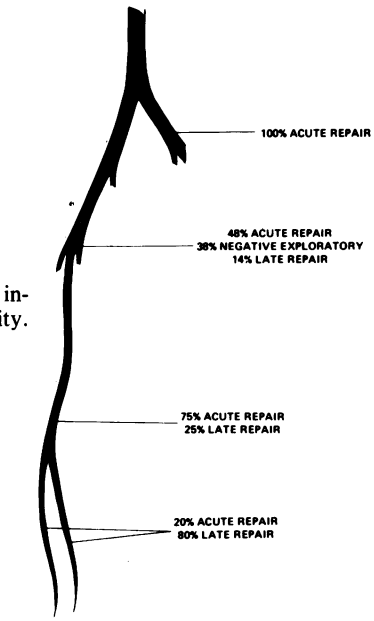

FIG. 3. Management of arterial injury of pelvis and lower extremity.

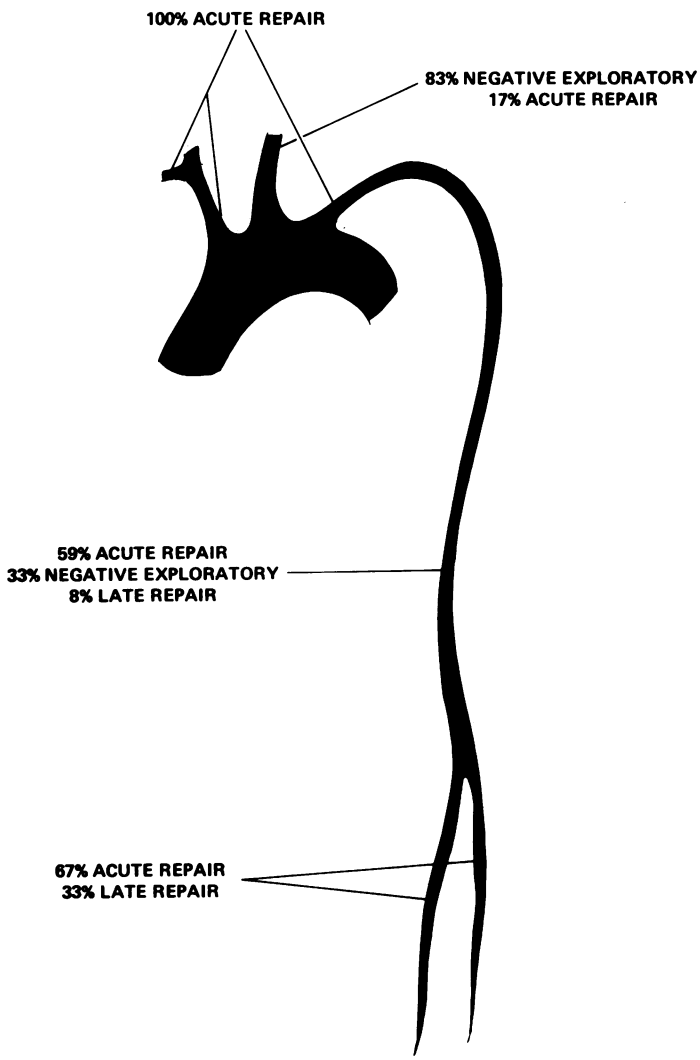

FIG. 2. Management of arterial-injury of neck and upper extremity.

Table 5 shows the relationship between the harvest site of autogenous donor vein and the results of arterial repair. Results are graded as good for patients with an asymptomatic viable limb without edema, and poor in patients with postoperative edema or in whom amputation was the ultimate outcome. Although the donor vein was taken from the injured extremity in only 5 cases, two resulted in amputation and one in persistent edema. We obtained better results in those patients in whom the donor vein was obtained from a non-injured extremity.

Thirteen major vein injuries were seen in association with the 46 acute arterial injuries (Table 6). There were no amputations and no edema in the group in which the venous injuries were repaired. However, there was a 38% incidence of poor results in the group in which the injured veins were ligated.

There were two deaths in the group of 46 patients undergoing acute repair of arterial injuries, an incidence of 4%. These were the only deaths in this series. One patient with extensive neurological damage on admission died a few hours after control of hemorrhage and repair of a common carotid artery transection. The other death occurred 19 days following repair of a penetrating injury of the superficial femoral artery in a patient with extensive remote injuries. This patient had sustained multiple gunshot wounds to the chest and abdomen with extensive liver injury. The cause of death was endotracheal exsanguination and suffocation from an innominate artery-tracheal fistula.

The failures (amputations) in the acutely reconstructed group are detailed in Table 7. Seven of 46 patients (15%) went on to amputation following acute repair. All 7 patients had either fractures and/or extensive soft tissue damage in addition to the arterial injury.

TABLE 4. *Acute Reconstructions Method of Repair*

Method of Repair	No. Patients	Per cent
Lateral suture	5	10
End–end anastamosis	22	45
Autogenous vein graft	15	31
Ligation	5	10
Evacuation hematoma@ fracture	1	2
Release from compressing bone fragments	1	2
Total	49*	100

* Includes three reoperations.

Group II—Negative Explorations

There were 35 negative explorations for suspected arterial injuries in the 4-year period covered by this review. Suspected carotid injuries accounted for the highest number of negative explorations, followed in order by the femoral and brachial vessels (Figs. 2 and 3).

The complication rate directly related to exploration of the vessel was 8.6%. The complications were all associated with neck explorations and consisted of one wound infection, one unilateral vocal cord paralysis, and one hypertrophic scar which required revision.

Group III—Late Complications of Missed Injuries

The salient features of 13 patients who underwent surgery for late complication of a missed arterial injury are depicted in Table 8. Six patients had an initial investigation which overlooked their arterial injury. The length of delay from injury to definitive reconstruction ranged from one week to 43 years. False aneurysm was the most common late complication (62%) followed by arteriovenous fistula (31%) and claudication (15%). The most striking feature in this group is the finding that 46% of these late reconstructions involved the radial-ulnar or tibio-peroneal vessels.

Although there were no deaths and no limb loss in this group, the results are worse than for acute arterial repair. Four patients (31%) are felt to have results which are less than satisfactory. Two patients have undergone reoperation for persistent A-V fistulae, and one of these still has arteriovenous communication. In addition, two patients had claudication following arterial repair of false aneurysm.

Repair of false aneurysm has been accomplished by either lateral suture, endoaneurysmorrhaphy or interposed vein graft. A-V fistulae have been repaired by either ligation or resection and vein graft. Claudication has been caused by arterial stenosis (Cases 1 and 9, Table 8) and was corrected by resection and vein graft.[23]

TABLE 5. *Results of Autogenous Vein Grafts*

Site of Donor Vein	Performed	Poor Results	Per cent
Ipsilateral	5	3	60
Contralateral	10	3	30
Total	15	6	40

TABLE 6. *Associated Major Venous Injuries*

Management	No. Patients	Poor Results	Per cent
Vein Repaired	5	0	0
Vein Ligated	8	3	38
Total	13	3	23

Discussion

In this review, we have attempted a critical analysis of three questions: 1) What are the factors contributing to the failure rate of acute arterial repair? 2) What are the implications of a negative exploration of suspected arterial injury, and is a significant percentage of negative exploration justified? 3) Why do large numbers of post-traumatic false aneurysms and A-V fistulae occur as late complications of arterial injury?

An in-hospital amputation rate of 7–15% following elective reconstructive surgery for arteriosclerotic occlusive disease is unacceptable to most vascular surgeons. Why then, should one be complacent with this failure rate following repair of normal vessels in younger patients? The basic tenets for successful arterial repair include: minimal delay in recognition and treatment, generous use of preoperative and intraoperative arteriography, proximal and distal thrombectomy with a balloon tipped catheter, judicious use of heparin, and use of autogenous vein rather than cloth grafts. These are well recognized and have contributed to the improvement of results since World War II. Other important factors are concomitant venous repair, harvest site of autogenous donor graft and the type of treatment for associated fractures and soft tissue injuries.

Experimental work has been reported concerning the role of venous repair in arterial injuries. Barcia and Wright demonstrated a fall in femoral artery flow following venous occlusion, and return of femoral artery flow following release of venous occlusion.[3,40] Hobson, in subsequent studies, showed that if venous occlusion persisted, femoral artery flow would not return to normal before 72 hours.[14] This low femoral artery flow was associated with an increase in peripheral resistance and venous pressure distal to the femoral vein occlusion. Alpha adrenergic blockade and sympathectomy will decrease the fall in femoral artery flow in this experimental setting by decreasing peripheral vascular resistance.[41,42] This work has been reproduced in the primate, whose vascular anatomy in the extremity more closely resembles man.[43]

The mechanism of increased resistance is unclear in these studies. Following venous occlusion, a transitory

TABLE 7. *Failures of Acute Arterial Reconstruction*

Case	Mechanism of Injury	Site of Injury	Delay >6 hr	Ipsilateral Donor Vein	Associated Venous Inj.	Venous Repair	Fasciotomy
1	Blunt + fracture	Popliteal	+	–	–	–	–
2	Blunt + fracture	Popliteal	–	–	–	–	–
3	Blunt + fracture	Common Femoral	–	+	–	–	–
4	Shotgun + fracture	Superficial Femoral	–	+	+	–	Delayed
5	Blunt + fracture	Popliteal	+	–	–	–	Simultaneous
6	Wringer	Brachial	+	–	–	–	Simultaneous
7	Blunt + fracture	Tibio-Pero	–	–	+	–	–
	Per cent		43	29	29	0	43

increase in venous pressure may recruit and distend existing venous channels. However, previous work from this institution indicates that decreased femoral artery flow can follow arterial injury without concomitant venous occlusion.[39] The decreased femoral artery flow and increased peripheral resistance can be reversed by sympathectomy. We have not routinely employed sympathectomy or alpha adrenergic blockade in the management of peripheral arterial injuries. We have, however, used subarachnoid and supraclavicular brachial plexus block when the injury was suitable. In a few instances we have noted subjective improvement in limb warmth and color.

Why does acute repair of arterial injury sometimes fail? The extent and severity of local injury to soft tissue and bone and the coexistence of extensive remote injury in the chest and abdomen may negate or compromise the success of peripheral arterial repair. In patients with multiple injuries including fracture and/or dislocation, limb ischemia may be thought to be due to vascular spasm. The diagnosis of arterial spasm is a frequent trap which is attractive to weary surgeons awakened to care for a trauma victim. Limb ischemia due to arterial spasm is rare. Arterial damage and/or thrombosis must be considered as the causative agent for ischemia, especially in patients with bony or extensive soft tissue injury. Patients with successful resuscitation after thoracoabdominal injuries are vulnerable to delayed complications (false aneurysm, arteriovenous fistula, or limb ischemia) after undiagnosed and/or untreated arterial injuries of the extremities.

Several authors have recommended repair of venous injuries, both isolated and in association with arterial injuries.[21,27,32,35] Our data (Table 6) corroborate that repair of venous injuries associated with arterial injuries results in less postoperative edema and contributes to fewer amputations. In patients with extensive soft tissue, bony and vascular damage, all available veins should be preserved and/or repaired to augment limb salvage. In addition to the theoretical enhancement of arterial flow,[3,14,40–43] maintenance of venous outflow will lessen edema and tissue pressure in closed fascial compartments and improve arterial flow. If autogenous tissues are used in the venous repair, the patency rate is acceptable. If clotting does occur, recanalization may re-establish patency.[32] Because venous outflow is important in the success of arterial repair, it is essential to preserve venous collaterals. In this study (Table 5), use of a donor vein from the injured limb was associated with a greater frequency of edema and amputation than was the use of a

TABLE 8. *Late Arterial Repairs*

Case	Mechanism of Injury	Site of Injury	Time of Delay	Presentation	Initial Invest.*	Results
1	Cardiac cath	Brachial	1 mo	Claudication	None	Good
2	High velocity	Popliteal	2 mo	False Aneur.	None	Good
3	Shotgun (no fract)	Rad/ulnar	1 mo	A-V Fistula	Arteriogram	Fair
4	Low velocity	Tibio-pero	20 yr	False Aneur.	Explored	Good
5	High velocity	Tibio-pero	9 mo	Aneur./Fistula	Explored	Fair
6	Stab-penetration	Radial	1 mo	False Aneur.	Explored	Good
7	Cardiac cath	Femoral	2 wk	False Aneur.	None	Good
8	Low velocity	Tibio-pero	2 mo	A-V Fistula	None	Good
9	Blunt/dislocation	Popliteal	1 wk	Claudication	Arteriogram	Good
10	Low velocity	Femoral	9 wk	False Aneur.	None	Fair
11	Low velocity	Femoral	43 yr	False Aneur.	None	Fair
12	High velocity	Tibio-pero	3 wk	A-V Fistula	Arteriogram	Good
13	Low velocity	Femoral	8 mos	False Aneur.	None	Good

* Exploration or arteriography.

vein from the uninjured limb. The use of an ipsilateral vein graft is especially hazardous in patients with extensive soft tissue, bony, and vascular injury.

We have had no experience with the use of porcine skin grafts to cover exposed vein grafts in patients with extensive skin and soft tissue loss.[19]

When edema impairs limb blood flow, fasciotomy should be considered. Fibulectomy-fasciotomy has salvaged limbs with extreme edema and impaired blood flow.[10,20] In the upper extremity we have used long fasciotomies and have avoided short fasciotomies through limited skin incisions.

The association of fractures with arterial injuries as a determinant in limb survival cannot be over emphasized. All but one amputation in our patients occurred in association with fractures (Table 7). It is doubtful that the presence of a fracture per se has a detrimental influence on the outcome of arterial repair. Usually there is an element of delay in reconstruction either preoperatively or intraoperatively while orthopedic manipulations are carried out.[4] In addition, injuries with sufficient force to cause fractures can result in associated soft tissue and venous injury which may have a negative bearing on the eventual outcome. There is no clear answer to the question of internal versus external stabilization of fractures. Internal fixation has been recommended by some,[25,34] but the Vietnam experience and subsequent studies suggest the use of external stabilization.[5,6,28,29]

The question of negative exploration for suspected injury is closely related to that of the role of arteriography in the management of arterial trauma. Diagnostic confirmatory arteriography delays acute repair of obvious arterial injuries, and is not indicated. Equivocal arterial injury is the most frequent indication for arteriography.[33] Arterial injury may be present despite a normal arteriogram.[17,18,33] We had 23% false negative arteriograms in patients with late complications (false aneurysms and A-V fistulae). We feel that direct exposure and examination of a vessel suspected of injury is more accurate than arteriography. Intraoperative arteriography can be used to determine presence or absence of other injuries, peripheral thrombus, and adequacy of repair. Precise arteriography is mandatory prior to definitive repair of traumatic A-V fistulae or false aneurysms. These lesions may be multiple and easily overlooked resulting in incomplete repair.

Negative exploration carries an acceptable morbidity rate when compared to the complications of missed arterial injury. Surgeons should not be any more reticent or apologetic about negative exploration for equivocal arterial injury than they are in performing thoracotomy for a possibly malignant pulmonary lesion.

Late complications of arterial injuries—false aneurysms, arteriovenous fistulae, and ischemia—continue to be seen by the vascular surgeon.[11,13,16,22,31] Analysis of our patients reveals that the greatest percentage of complications (46%) occurs in those individuals with radial-ulnar and tibio-peroneal injury. Arterial injury below the elbow and knee is oftentimes watched until, or unless, obvious hemorrhage or ischemia appears. Arteriography and/or early exploration of these areas may lessen these complications. Recent technical advances allow surgical reconstruction of small vessels, and suggest aggressive rather than expectant treatment in these sites. We concur with a recent report that advises exploration of these vessels with revascularization when possible, and ligation where it is not.[17] Reconstruction of traumatic aneurysms and arteriovenous fistulae is more demanding than is acute repair of arterial injury.

References

1. Amato, J. J., Billy, L. J., Gruber, R. P., et al.: Vascular Injuries: An Experimental Study of High and Low Velocity Missile Wounds. Arch. Surg., 101:167–174, 1970.
2. Amato, J. J., Rich, N. M., Billy, L. J., et al.: High-velocity Arterial Injury: A Study of the Mechanism of Injury. J. Trauma, 11(5):412–416, 1971.
3. Barcia, P. J., Nelson, T. G. and Whelan, T. J., Jr.: Importance of Venous Occlusion in Arterial Repair Failure: An Experimental Study. Ann. Surg., 175:223–227, 1972.
4. Blaisdell, F. W.: In Discussion: Rosenthal, J. J., Gaspar, M. R., Gjerdrum, T. C., et al.: Vascular Injuries Associated with Fractures of the Femur. Arch. Surg., 110:494–499, 1975.
5. Connolly, J.: Management of Fractures Associated with Arterial Injuries. Am. J. Surg., 120:331, 1970.
6. Connolly, J. F., Whittaker, D. and Williams E.: Femoral and Tibial Fractures Combined with Injuries to the Femoral or Popliteal Artery. J. Bone Joint Surg., 53-A:56–58, 1971.
7. Dale, W. A.: The Beginnings of Vascular Surgery. Surgery, 76:849–866, 1974.
8. DeBakey, M. E., and Simeone, F. A.: Battle Injuries of the Arteries in World War II. An Analysis of 2,471 Cases. Ann. Surg., 123:534–579, 1946.
9. Drapanas, T., Hewitt, R. L., Weichert, R. F. III, et al.: Civilian Vascular Injuries: A Critical Appraisal of Three Decades of Management. Ann. Surg., 172:351–360, 1970.
10. Ernst, C. B. and Kaufer, H.: Fibulectomy—Fasciotomy. An Important Adjunct in the Management of Lower Extremity Arterial Trauma. J. Trauma, 11(5):365–380, 1971.
11. Fomon, J. J. and Warren, W. D.: Late Complications of Peripheral Arterial Injuries. Arch. Surg., 91:610–616, 1965.
12. Hardy, J. D., Raju, S., Neely, W. A., et al.: Aortic and Other Arterial Injuries. Ann. Surg., 181:640–653, 1975.
13. Hewitt, R. L., Smith, A. D. and Drapanas, T.: Acute Traumatic Arteriovenous Fistulas. J. Trauma, 13:901–906, 1973.
14. Hobson, R. W., Howard, E. W., Wright, C. B., et al.: Hemodynamics of Canine Femoral Venous Ligation: Significance in Combined Arterial and Venous Injuries. Surgery, 74:824–829, 1973.
15. Hughes, C. W.: Arterial Repair During the Korean War. Ann. Surg., 147:555–561, 1958.
16. Hughes, C. W. and Jahnke, E. J., Jr.: The Surgery of Traumatic Arteriovenous Fistulas and Aneurysms. A Five-Year Follow-Up Study of 215 Lesions. Ann. Surg., 148:790–797, 1958.
17. Kelly, G. L. and Eiseman, B.: Civilian Vascular Injuries: J. Trauma, 15(6):507–514, 1975.
18. Lain, C. K. and Williams, G. R.: Arteriography in Acute

Peripheral Arterial Injuries: An Experimental Study. Surg. Forum, 21:179–181, 1970.
19. Ledgerwood, A. M. and Lucas, C. E.: Massive Thigh Injuries with Vascular Disruption. Role of Porcine Skin Grafting of Exposed Arterial Vein Grafts. Arch. Surg., 107:201–205, 1973.
20. Lord, R. S. and Irani, C. N.: Assessment of Arterial Injury in Limb Trauma. J. Trauma, 14(12):1042–1053, 1974.
21. McNamara, J. J., Brief, D. K., Beasley, W., et al.: Vascular Injury in Vietnam Combat Casualties: Results of Treatment at the 24th Evacuation Hospital 1 July 1967 to 12 August 1969: Ann. Surg., 178:143–147, 1973.
22. Montales, E., Yao, S. T. and Silva, Y. J.: Management of Traumatic Peripheral Arteriovenous Fistulas. J. Trauma, 13:161–165, 1973.
23. Page, C. P., Hagood, C. D., Jr. and Kemmerer, W. T.: Management of Postcatheterization: Brachial Artery Thrombosis. Surgery, 72:619–623, 1972.
24. Perry, M. O., Thal, E. R. and Shires, G. T.: Management of Arterial Injuries. Ann. Surg., 173:403–408, 1971.
25. Pradhan, D. J., Juanteguy, J. M., Wilder, R. J. and Michelson, E.: Arterial Injuries of the Extremities Associated with Fractures. Arch. Surg., 105:582–585, 1972.
26. Rich, N. M., Baugh, J. H. and Hughes, C. W.: Acute Arterial Injuries in Vietnam: 1000 Cases. J. Trauma, 10:359–369, 1970.
27. Rich, N. M., Hughes, C. W. and Baugh, J. H.: Management of Venous Injuries. Ann. Surg., 171:724–730, 1970.
28. Rich, N. M., Metz, C. W., Jr., Hutton, J. E., Jr., et al.: Internal versus External Fixation of Fractures with Concomitant Vascular Injuries in Vietnam. J. Trauma, 11:463–473, 1971.
29. Rosenthal, J. J., Gaspar, M. R., Gjerdrum, T. C., et al.: Vascular Injuries Associated with Fractures of the Femur. Arch. Surg., 110:494–499, 1975.
30. Rubio, P. A., Reul, G. J., Jr., Beall, A. C., Jr., et al.: Acute Carotid Artery Injury: 25 Years Experience. J. Trauma, 14:967, 1974.
31. Sako, Y. and Varco, R. L.: Arteriovenous Fistula: Results of Management of Congenital and Acquired Forms, Blood Flow Measurements, and Observations on Proximal Arterial Degeneration. Surgery, 67:40–61, 1970.
32. Schramek, A., Hashmonai, M., Farbstein, J., et al.: Reconstructive Surgery in Major Vein Injuries in the Extremities. J. Trauma, 15:816–822, 1975.
33. Smith, R. F., Elliott, J. P., Hageman, J. H., et al.: Acute Penetrating Arterial Injuries of the Neck and Limbs. Arch. Surg., 109:198–205, 1974.
34. Smith, R. F., Szilagyi, E. and Elliott, J. P., Jr.: Fracture of Long Bones with Arterial Injury Due to Blunt Trauma. Arch. Surg., 99:315–324, 1969.
35. Sullivan, W. G., Thornton, F. H., Baker, L. H., et al.: Early Influence of Popliteal Vein Repair in the Treatment of Popliteal Vessel Injuries. Am. J. Surg., 122:528–531, 1971.
36. Thal, E. R., Synder, W. H. III, Hays, R. J., et al.: Management of Carotid Artery Injuries. Surgery, 76(6):955–962, 1974.
37. Weiss, J. B., Ozment, K. L., Westbrook, K. C. and Williams, G. D.: Tracheal-Innominate Artery Fistula: Successful Management of Two Cases. J. South. Med. Assoc., to be published.
38. Williams, G. D.: Peripheral Vascular Trauma. Report of Ninety Cases. Am. J. Surg., 116:725–730, 1968.
39. Williams, G. D., Crumpler, J. B. and Campbell, G. S.: Effect of Sympathectomy on the Severely Traumatized Artery. Arch. Surg., 101:704–707, 1970.
40. Wright, C. B. and Swan, K. G.: Hemodynamics of Venous Occlusion in the Canine Hindlimb. Surgery, 73:141–146, 1973.
41. Wright, C. B., Swan, K. G., Reynolds, D. G., et al.: Venous Occlusion in the Canine Hindlimb: Hemodynamic Effects of Adrenergic Stimulation and Blockade. Surgery, 73:507–511, 1973.
42. Wright, C. B., Sayre, J. T., Casterline, P. I., et al.: Hemodynamic Effects of Sympathectomy on Canine Femoral Occlusion. Surgery, 74:405–411, 1973.
43. Wright, C. B. and Hobson, R. W.: Hemodynamic Effects of Femoral Venous Occlusion in the Subhuman Primate. Surgery, 75:453–460, 1974.

Discussion

Dr. David M. Conkle (Nashville, Tennessee): I thoroughly enjoyed Dr. Campbell's presentation, and I agree with his conclusions regarding the correlation of the type of injury and the success of salvage, and the need for concomitant venous repair, whenever possible.

(Slide) Since January 1, 1959, we have seen 34 patients with popliteal artery injuries. All 34 patients underwent operative exploration following arteriography. There were no operative or hospital deaths.

(Slide) In breaking down our experience, we also found our salvage rate to be related to the type of injury incurred. In the 13 patients with penetrating injuries, successful reconstruction was achieved in 85% of the patients. In the 21 patients who experienced blunt trauma, our salvage rate fell to 52%. The overall success rate was 62%.

(Slide) In correlating the salvage rate with associated injuries, primarily in those who experienced blunt trauma, our data again backs Dr. Campbell's, in that it points out the negative influence that massive trauma has on successful salvage.

Our mode of treatment has been primarily with reversed saphenous vein segments taken from the noninvolved leg. If sufficient length is present to permit the anastomosis to be made without tension, end-to-end anastomosis is employed.

I'd likewise agree that all vessels should be explored and looked at very carefully, because minor procedures such as thrombectomy ordinarily lead to failure because a small intimal tear is overlooked.

We likewise employ fasciotomy in the postoperative patient who demonstrates evidence of decreased perfusion and compression in the compartment distal to the repair.

In closing, I would like to ask Dr. Campbell if fasciotomy was employed in any of his patients, and if he felt that it affected his results.

Dr. Frank C. Spencer (New York, New York): Regarding diagnosis, as the authors quite properly emphasize, a liberal index of suspicion is especially important. Any wound in proximity to a major artery should be regarded as a potential arterial injury, regardless of the absence of clinical signs of arterial injury. An arterial injury can be excluded with certainty only by an arteriogram or operative exploration.

The long-term goal with arterial injuries, of course, is to decrease the amputation rate. In this series, like other recent reports, the overall amputation rate was 10–12%. Future improvement will probably come in two areas. First is the more extensive teaching of principles which have been enunciated several times. The time interval between arterial injury and restoration of flow following arterial repair is crucial. If the objective of restoring blood flow within six to eight hours after injury is considered of paramount importance, arranging priorities accordingly, decisions such as preoperative angiography, priority of treatment of multiple injuries and similar considerations can be made accordingly. A retrospective analysis of many amputations finds that the basic problem was simply that arterial repair was not accomplished within six to eight hours for a variety of reasons, partly because extreme emphasis was not placed on this point.

Another future improvement in therapy can probably be achieved with the frequent use of operative angiography. The two most common failures of arterial repair are either an inadequate vascular repair or inadequate removal of thrombus from the distal arterial tree with a Fogarty catheter. Both mishaps can be promptly detected with operative arteriography and treated accordingly.