

Endovascular Management of Vascular Trauma

Khanjan H Nagarsheth^{1*}

¹Division of Vascular Surgery, Robert Wood Johnson Medical School, Rutgers University, USA

***Corresponding author:** Khanjan H Nagarsheth, Division of Vascular Surgery, Robert Wood Johnson Medical School, Rutgers University, One Robert Wood Johnson Place – MEB 548, New Brunswick, NJ, USA, Email: khanjan.nagarsheth@gmail.com

Published Date: February 10, 2016

ABSTRACT

Vascular trauma accounts for 1.6 million injuries to vascular structures every year at trauma centers in the United States. Over the last five decades, the diagnosis and treatment of these injuries has been evolving. Endovascular therapies used for atherosclerotic disease have been adopted and used in the treatment of vascular injuries. This modality has revolutionized the way we think about and treat vascular trauma. Certain injuries, such as blunt thoracic aortic injuries, are now almost exclusively treated via endovascular methods. Improvements in delivery systems and imaging modalities have resulted in a significant interest in the application of endovascular techniques to vascular injuries throughout the body. Herein we will discuss the beginnings of endovascular techniques, its application and complications.

INTRODUCTION

Rogerius Salernitabus, an Italian surgeon, first described the treatment of a jugular venous injury created by a sword. In his medical text *Practica Chirurgiae* he repaired the vessel with a needle and thread to achieve hemostasis without ligating the vessel. Since that time in the late tenth century, vascular trauma surgery has matured beyond ligations and simple arterial and venous repairs.

There are approximately 40 million emergency room visits made every year for the evaluation of traumatic injuries [1]. Most of the injuries sustained after trauma occur after a blunt trauma from motor vehicle collision, falls and or penetrating trauma secondary to firearms or piercing weapons. Vascular trauma is estimated to account for 0.2% to 4% of all traumatic injuries. More than 80% of vascular trauma is confined to the peripheral vascular system. Most of these injuries are to the lower extremities; 75% are secondary to firearms, 15% secondary to stabbings, and 10% secondary to blunt force trauma [2].

Recently, the use of endovascular technology has become more frequently reported for management of vascular trauma.. Although its use is becoming more frequent in emergency scenarios, there is still no generalized consensus for the therapeutic role of endovascular techniques in the setting of trauma. Herein, we will go over a brief history of endovascular techniques and its use with respect to specific injury patterns.

DEVELOPMENT OF ENDOVASCULAR TECHNIQUES

Endovascular techniques have changed the landscape for management of vascular injuries. Historically, open surgical repair or ligation and medical therapies were the only options for treatment. With the evolution of endovascular therapies, treatment options have significantly expanded to help in dealing with these injuries. The evolution of endovascular management of vascular injuries required three major developments to occur in the field of catheter-based therapeutics. These were the development of percutaneous transluminal balloon angioplasty, the deployment of endovascular stent grafts and the development of distal protective devices to prevent embolism of atherothrombotic materials.

Catheter based therapy was initially described for the treatment of atherosclerotic disease. Dotter and Judkins initially described the transluminal treatment of atherosclerotic lesions by placing sequentially larger dilators to relieve arterial stenosis and then Dotter described the use of transluminal placement of coilspring arterial tube grafts to restore patency to blood vessels [3,4]. The method of balloon angioplasty was shown to be a viable option for stenosed blood vessels by Gruntzig some ten years later. In this method he developed a technique to pass a polyvinyl chloride balloon, percutaneously through an atherosclerotic lesion to break up the plaque found in the artery and stretches the adventitia and media of the vessel, thereby increasing the vessels diameter and relieving the stenosis [5].

Dotter was the first to report percutaneous placement of an endoluminal tube graft to restore arterial patency. This event ushered in an age of rapid growth in the development of endoluminal stent grafts. In the early 1980s the use of nitinol stents and wires was becoming more prevalent and this lead to the development of balloon expandable stents which were found to give better vascular patency rates than balloon angioplasty alone [6-9]. There are a variety of endoluminal stent grafts available and they are roughly divided into balloon expandable and self-expanding stent grafts. Balloon expandable stents conform to the arterial wall and are kept in place due

to friction between the metal of the stent and the intima of the artery and by subsequent endothelialization. These stents are most useful in protected regions of the body and for lesions at the ostia of vessels that are calcified due to blood flow characteristics. They are usually quite malleable because they are made of stainless steel alloys and have a high radial strength. Another advantage of balloon expandable stents is the ability to place them with great precision and accuracy, making them invaluable in delicate areas [10].

When placed, self-expanding stents convert the potential energy stored in their closed state to kinetic energy in the form of outward force. The opposition of this kind of stent to the vessel wall is a function of this outward force and the elasticity of the vessel wall. The outward force on the blood vessel and the natural tendency of the arterial wall to recoil inward create equilibrium of balanced forces, thereby keeping the stent stationary against the intimal surface. Many self-expanding stents are made of nitinol, which is a metal alloy that is made of equal parts titanium and nickel. Nitinol affords a greater degree of flexibility than balloon expandable stents, therefore they are better suited for tortuous vessels. However, these stents exert less radial force on the blood vessel and have a less precise deployment. The decreased accuracy in deploying these stent grafts can lead to inadequate coverage of lesions and result in distal scattering of embolic material.

Endovascular interventions can result in distal embolization of atherothrombotic debris that are sometimes clinically silent, but can have devastating consequences such as cerebrovascular accidents and ischemia. Experimental models have shown that atherothrombotic debris are embolized distally during carotid artery angioplasty [11]. This work spurred the development of distal protection devices. These devices are either occlusion balloons, filter devices and flow reversal devices. Although placement of these devices requires traversing the lesion or injury and adds to the complexity and length of the procedure, their proper use can prevent adverse outcomes. In trauma situations, distal filter devices provide protection from emboli and allows for passage of contrast past the injured area, so the distal vasculature can be evaluated. Distal occlusion balloons are not usually utilized in these situations as they completely occlude blood flow and thus filling distally is heavily dependent on collateral circulation. Additionally, distal occlusion balloons may gradually deflate over the course of the procedure and allow antegrade blood flow to occur; facilitating embolization of debris and thrombus. Over-distention of the balloon can cause arterial dissection and vasospasm that can also have adverse outcomes for the patient. Although these devices are more flexible and may cross injuries and lesions easier than filter devices, there have been reports of up to 5.2% adverse neurologic outcomes when using distal occlusion balloons [12].

As mentioned previously, distal filter devices have the advantage of allowing blood and contrast to flow antegrade past the device as facilitated by pores in the filter. These devices must, however, also be maneuvered past the lesion in question to be adequately positioned and are associated with subsequent distal embolization risk. A problem inherent in the design of these filters is that though the pores are made to prevent large emboli, small debris and fibrin can still get through;

resulting in microemboli to the distal vasculature. Another concern is that these devices can also develop what is known as filter thrombosis from debris plugging the pores, which makes their removal more difficult and obstructs distal blood flow [13].

THORACIC AORTIC INJURIES

Thoracic Endovascular Aortic Repair (**TEVAR**) has been touted as the new standard of care for many traumatic aortic injuries that were traditionally treated by open surgical means [14-18]. The American Association for the Surgery of Trauma (**AAST**) Thoracic Aortic Injury Study Group has conducted the largest prospective observational trial of blunt thoracic aortic injuries [19,20]. The investigators found that 65% of the 193 patients with Thoracic Aortic Injury (**TAI**) underwent **TEVAR**. The remaining 35% of patients had a standard open repair performed. The **TEVAR** group was transfused significantly fewer blood products and had a significantly lower mortality than open surgical counterparts (adjusted odds ratio: 8.42; 95% CI: [2.76-25.69]; adjusted p value < 0.001). They also found that 20% of **TEVAR** patients developed some form of device-related complications. These complications were fewer in centers that were deemed high volume **TEVAR** centers.

The **AAST** group on thoracic aortic injuries reported on the trends in utilization of **TEVAR** between two phases of study over the course of ten years (**AAST1**, completed in 1997 and spanning 30 months and **AAST2**, completed in 2007 and lasting 26 months). The group noted a trend favoring the use of **TEVAR** when comparing these two time periods. During the **AAST1** study all **TAI** were repaired utilizing open means. In contrast, by the time of the 2007 study conclusion, only 35.2% were managed with an open surgical procedure. The majority of patients underwent a **TEVAR**. There was also a significant decrease in **TAI**-associated mortality (22% vs. 13%, p = 0.02) and incidence of procedure-related paraplegia (8.7% vs. 1.6%, p = 0.001).

There are still some unanswered questions about the use of **TEVAR** to repair blunt aortic injury. Further research is required to better define the natural history and problems that can arise over the course of a patient's lifetime after having a **TEVAR** performed for aortic injury. Although more sophisticated endovascular technologies and improved management algorithms have attempted to reduce the risks associated with graft deployment, graft failure, migration and inadvertent coverage of arch great vessels, further research is required. The relative younger age of many trauma patients and their smaller, non-aneurysmal thoracic aortas may increase the potential for stent malfunction, migration or other complications over time.

BLUNT CEREBROVASCULAR INJURIES

Blunt Cerebrovascular Injury (**BCVI**) has been proposed as being due to any combination of the following four mechanisms: a direct blow to the neck, hyperextension injury with contralateral rotation of the head, intra-oral injury and arterial injury from adjacent bony fractures in the neck or at the base of the skull [21]. The reported incidence of **BCVI** was in the realm of 0.1% but as

Miller and Fabian found, the incidence was actually 1-2% [22]. This was likely not due to a real increase in the incidence of this injury pattern, but more likely due to a more rigorous screening process implemented in many high volume institutions and the higher depth of detail afforded by modern day multidetector computed tomographic (**MDCT**) scanners [23].

The Western Trauma Association (**WTA**) has proposed diagnostic and treatment algorithms for **BCVI** [24]. This work identifies which patients are deemed high risk for a **BCVI** and which ones should be screened to identify an injury. The following patients should be felt to have a **BCVI** until definitely ruled out: trauma patients who have arterial hemorrhage from the neck, mouth, nose; a large cervical hematoma; a cervical bruit in younger patients; focal or lateralizing neurologic deficits; cerebral infarction on advanced imaging; or a patient who has neurologic deficits not explained by advanced imaging. The group recommends that prompt diagnostics and treatment should be pursued to avoid potentially catastrophic consequences of **BCVI**.

The gold standard for diagnosis of **BCVI** has long been the four vessel biplanar cerebral angiogram. This imaging modality has a very high sensitivity for **BCVI**, but is invasive and carries certain risks. Catheter related complications, such as hematoma, retroperitoneal bleeding and arterial pseudoaneurysm formation is reported to be 1-2%. Renal dysfunction and acute kidney injury from contrast material occurs in 1-2% of cases. Development of a stroke from this procedure occurs in less than 1% of patients [25].

The most commonly utilized grading scale for **BCVI** was devised by Biffel and colleagues [26]. A comprehensive grading scale is important because most treatment recommendations are tailored to the grade and severity of the injury. Grade I is said to be intimal irregularity with < 25% luminal narrowing. Grade II injuries involve a dissection or intramural hematoma with >25% luminal narrowing. Grade III injuries have a pseudoaneurysm that has developed, and grade IV injuries have resulted in complete occlusion of the vessel. Grade V injuries are defined as arterial transection and extravasation.

As alluded to by this grading system, there are **BCVI** that do undergo operative intervention based on signs of hemorrhage or expanding hematoma. Intimal flaps, dissections and pseudoaneurysms (Grade I, II, and III injuries) do not routinely undergo operative intervention. Although the natural history of these injuries is not well known, the current treatment recommendations for them include anticoagulation and observation. It has been reported, however, that up to 40% of these patients may require endovascular intervention for their injuries [27]. Based on the endovascular experience with angioplasty and stents for cerebrovascular disease, experience with carotid stenting for some high extracranial internal carotid injuries has grown. Endovascular approaches are useful in regions that are anatomically inaccessible and unsuited for surgical exploration and intervention. Endovascular techniques may be helpful in the treatment of grade III or higher **BCVI**. The potential of these less invasive modalities is important, as surgical exploration and treatment of these pseudoaneurysms may be associated with high rates of mortality and cerebral complications.

DuBose and colleagues performed an extensive review of the literature from 1994 to 2008 and identified 113 patients undergoing carotid stenting for trauma [28]. The injury types treated by stenting included pseudoaneurysm (60.2%), arteriovenous fistula (16.8%) dissection (14.2%), partial transection (4.4%), occlusion (2.7%), intimal flap (0.9%) and aneurysm (0.9%). Over radiographic and clinical follow-up periods ranging from 2 weeks to 2 years, a follow-up patency of 79.6% was documented. New neurologic deficits after stent placement occurred in only 3.5% of patients. This was better than was historically reported and accepted after open surgical intervention. There is still no large prospective study looking at endovascular versus open treatment of **BCVI**. More research is warranted in this area before recommendations can definitively be made.

SUBCLAVIAN AND AXILLARY ARTERY INJURIES

Subclavian and axillary artery injuries are associated with high rates of morbidity and mortality. Demetriades and colleagues examined penetrating subclavian and axillary injuries, identifying 79 patients with these injuries over approximately 4 years [29]. The overall mortality was 34.2%. Mortality was still 14.8% when patients arriving in extremis and those needing resuscitative thoracotomy were selected out. The anatomy of the thoracic inlet is such that there is a large number of vital organs and structures in a very small and difficult to access area of the body. This contributes to the difficulty open surgical treatment of axillo-subclavian trauma. For these reasons, endovascular therapy has been a very attractive alternative to open techniques for these challenging injuries.

In the largest review of this topic, investigators identified 32 published reports of endovascular treatment of subclavian or axillary artery injuries with sufficient information for review [30]. They found 160 patients (150 subclavian; 10 axillary), the majority of which (56.3%) were due to penetrating injuries. Lesions treated included pseudoaneurysm, arteriovenous fistula, perforation, occlusion, partial or complete transection and dissection. They identified six complications associated with the endovascular treatment and one death in the endovascular suite after the stent was deployed. Overall, radiographic and clinical follow-up periods ranging from hospital discharge to 70 months were reported. There were no reports of device-related infections, migrations or acute limb threatening ischemic events reported among the 160 identified cases. An asymptomatic patency rate of 84.4% was identified on follow-up. This report highlights that, while early results of endovascular treatment for selected subclavian and axillary injuries are promising, further research is needed to identify which patients and which injury patterns are ideally suited to this form of management.

Hybrid Procedures: Combining Open and Endovascular Techniques for Hemorrhage Control:

There has been a realization in recent years that endovascular and open techniques may be able to coexist and work in conjunction to obtain hemorrhage control and facilitate repair.

So called “hybrid approaches” may prove to be very helpful in the treatment of vascular injury. Endovascular balloons have been effectively utilized for proximal occlusion, dilation and stent deployment in the treatment of atherosclerotic disease. Increasingly, their use in hybrid vascular trauma applications has also been examined.

The use of endovascular occlusion for trauma indications was described by Scalea and Scalafani from Kings County Hospital of Brooklyn New York in 1991 [31]. This concept has been evaluated in numerous animal models looking at aortic occlusion to control hemorrhage [32-34]. This approach appears to be a natural progression in the realm of hemorrhage control for trauma. There have been reports of success with aortic cross clamping for exsanguinating hemorrhage associated with abdominal injuries [35]; and endovascular aortic balloon occlusion has been used in the area of aortic aneurysm rupture to control hemorrhage for some time [36]. The use of proximal endovascular balloon occlusion use in traumatic hemorrhage has, however, revealed mixed results. For this reason, the utilization of endovascular balloons in trauma and the ideal manner in which to employ them remains a matter of active investigation.

Avaro and colleagues have reported the feasibility of placing aortic balloons without radiographic guidance. In their animal model, they found that aortic balloon occlusion significantly improved survival in hemorrhagic shock. Mean arterial blood pressures were increased, blood loss was decreased and fluid resuscitation needed to restore normal physiology was decreased. As expected, they noted that metabolic acidosis worsened with increased duration of aortic balloon occlusion [37]. The limitations of this study and others like it include inherent differences in the methods of hemorrhage control. Aortic cross clamping, hemostatic agents and endovascular aortic balloon occlusion are all very different methods of control and all have different effects on a subject’s physiology. Additionally, the hemorrhagic shock and coagulopathy created in these animal studies is artificial and may not directly mimic that which is found in the trauma victim. The trauma victim may experience a more profound coagulopathy and metabolic derangement than is created in these models. That being said, these are important building blocks to bring this technique for hemorrhage control into effective trauma practice. Future study on these types of hybrid approaches is likely to demonstrate the complementary nature of endovascular and open surgical interventions for vascular trauma.

COMPLICATIONS OF ENDOVASCULAR TECHNIQUES

Endovascular techniques may prove to be very useful in the control of hemorrhage secondary to vascular injuries, but it is important to recognize potential complication of this mode of management as it becomes more prevalent in trauma care. Complications are divided into local and systemic complications. Systemic complications usually involve derangements in physiology affecting the cardio-pulmonary and renal systems. In this section we will discuss the local complications associated with endovascular therapies.

Most data present on access site injury is based on data from Percutaneous Coronary Interventions (**PCIs**) and ranges between 2.1% and 6.6% [38-40]. These complications are those that require either medical, surgical or transfusion related interventions. Complications after endovascular interventions include bleeding, hematoma formation, pseudoaneurysm, arteriovenous fistulas, arterial dissections, distal embolism of atherosclerotic debris or air and arterial thrombosis. Risk factors for access site injury include the use of larger access sheaths, the site of arteriotomy, previous catheterization at that site, smaller body mass index, coagulopathy, female gender, anti-platelet therapy and uncontrolled hypertension [41-43].

Hematomas are the most common complication noted after endovascular therapies. They are divided into access site hematomas and retroperitoneal hematomas. They are often noted when multiple punctures or attempts have been made in gaining access. The developing hematoma can obscure the pulsation and make access even more challenging. In the aging population, calcified arterial plaques are more of a problem. These plaques make access not only difficult but result in an irregular puncture site that can extravasate around the sheath and not completely seal even with appropriate pressure. Retroperitoneal hematomas occur most often because of a high puncture in the groin. The danger here is that these hematomas are not as obvious as groin or access site hematomas and the retroperitoneum is a large compartment that can accommodate a massive volume of blood before clinical signs are present. The diagnostic test of choice for this complication is a **CT** scan of the abdomen and pelvis. Most of these hematomas can be observed and monitored with serial hematocrit levels and correction of coagulopathy. Occasionally surgical intervention is needed in which case the site of arterial puncture on the vessel should be interrogated.

In the groin, Arteriovenous Fistula (**AVF**) formation occurs when an iatrogenic communication between the femoral artery and vein is created by a puncture to the bifurcation of the common femoral artery or profunda femoral artery. In most cases **AVFs** are identified on physical exam when a palpable thrill or audible bruit is found. Duplex ultrasound is the imaging of choice to characterize this complication. The incidence of AVF is typically felt to be less than 1%, but ranges from 0.05% to 2.8% in the literature [44,45]. The management of these fistulas has been an area of controversy. Historically they were treated with ultrasound guided compression with rather poor results. Some works have found that most of these **AVFs** will resolve spontaneously between four months and one year, and these patients are very unlikely to have any adverse physiologic events [46]. There has also been interest in the use of covered stents to exclude the **AVF** and prevent any further flow into the low pressure venous system, but this is not yet a recommended treatment in asymptomatic patients.

Pseudoaneurysms (**PSA**) occur when the arteriotomy does not seal completely when the sheath is removed after a puncture. Blood spills out of the vessel and the surrounding soft tissue contains this blood. Occasionally a fibrinous capsule will form around the **PSA**. This entity has arterial blood flow within it, which is why it is not considered a hematoma. There are several

risk factors that can predispose a patient to having a **PSA** after an endovascular procedure. These include obesity, large arterial sheath, inadequate compression after removal of the sheath, hypertension, calcified vessels and low arterial puncture, defined as below the bifurcation of the common femoral artery. They present as a pulsatile mass one to two days after the endovascular procedure. Duplex ultrasound is again the test of choice to characterize the **PSA** and its neck. Treatment of these injuries can be with simple observation in small **PSA**, ultrasound guided compression, ultrasound guided thrombin injection if there is a long well defined neck, and surgical intervention with repair of the arteriotomy in patients with rupture of the **PSA**. Currently ultrasound guided thrombin injection is the therapy of choice.

Dissection of the intima and media of the vessel wall can occur with percutaneous endovascular procedures. Angioplasty causes stretching of the blood vessel and if there is an atherosclerotic plaque present, it will cause disruption of the intima and the underlying plaque. If pulsatile flow gets into this disruption a dissection can occur and propagate distally. These complications are usually noted at the time of intervention. Most times sustained inflation of a low pressure balloon at the site of dissection will seal the dissection, but if this does not work or if the dissection limits flow distally and vascular stent can be used to collapse the dissection and restore blood flow. Subintimal dissection planes can be created by inappropriate passage of guidewires or catheters. Under fluoroscopic guidance the wire will tend to coil and advancing will be difficult. At this point a contrast angiogram will confirm a dissection plane, and if flow limiting a stent can be placed to resolve the dissection and prevent propagation.

Distal embolization is a well documented phenomenon in endovascular therapy of all vessels. The reported incidence is in the realm of 3% to 5 % but often times clinically silent [47]. The most feared complications of distal embolization include cerebrovascular accident and ischemia during aortic or carotid stent graft placement and critical limb ischemia in control peripheral arterial hemorrhage. Prevention of this complication can be achieved by adequate heparinization of the patient prior to vessel occlusion, though this is not often possible in the trauma patient, and using distal protection devices that were discussed earlier.

Thrombosis of an artery after endovascular therapy is uncommon and typically preventable. The factors that increase the risk of thrombosis include overly large sheath placed in a smaller artery, such as the brachial artery and excessive compression of the artery after sheath removal. These patients will present with signs of distal ischemia and prompt intervention is needed to remove the thrombus or restore distal flow.

CONCLUSION

Endovascular management of vascular injuries and hemorrhage control is an ever evolving field. As noted earlier these techniques are helpful either on their own or when combined with open surgical procedures. Further research into the appropriate use of this technology is needed to create lucid treatment algorithms. Additionally long term follow up data is needed to help define the optimal use of endovascular techniques in the setting of trauma.

References

1. Bonnie RJ, Fulco CE, Liverman CT. Magnitude and Costs. Reducing the Burden of Injury. Advancing Prevention and Treatment. Washington, DC, National Academy Press. 1999; 41-59.
2. Gupta R, Rao S, Sieunarine K. An epidemiological view of vascular trauma in western Australia: a 5-year study. *ANZ J Surg.* 2001; 71: 461-466.
3. Dotter CT, Judkins MP. Transluminal Treatment of Arteriosclerotic Obstruction. Description of a New Technic and a Preliminary Report of its Application. *Circulation.* 1964; 30: 654-670.
4. Dotter CT. Transluminally-placed coilspring endarterial tube grafts. Long-term patency in canine popliteal artery. *Invest Radiol.* 1969; 4: 329-332.
5. Gruntzig A, Kumpe DA. Technique of percutaneous transluminal angioplasty with the Gruntzig balloon catheter. *Am J Roentgenol.* 1979; 132: 547-552.
6. Dotter CT, Buschmann RW, McKinney MK, Rosch J. Transluminal expandable nitinol coil stent grafting: preliminary report. *Radiology.* 1983; 147: 259-260.
7. Cragg A, Lund G, Rysavy J, Castaneda F, Castaneda W, Amplatz K. Nonsurgical placement of arterial endoprostheses: a new technique using nitinol wire. *Radiology.* 1983; 147: 261-263.
8. Palmaz JC, Sibbitt RR, Reuter SR, Tio FO, Rice WJ. Expandable intraluminal graft: a preliminary study. Work in progress. *Radiology.* 1985; 156: 73-77.
9. Fischman DL, Leon MB, Baim DS, Schatz RA, Savage MP, Penn I, et al. A randomized comparison of coronary-stent placement and balloon angioplasty in the treatment of coronary artery disease. Stent Restenosis Study Investigators. *N Engl J Med.* 1994; 331: 496-501.
10. Leung DA, Spinosa DJ, Hagspiel KD, Angle JF, Matsumoto AH. Selection of stents for treating iliac arterial occlusive disease. *J Vasc Interv Radiol.* 2003; 14: 137-152.
11. Ohki T, Marin ML, Lyon RT, Berdejo GL, Soundararajan K, Ohki M, et al. Ex vivo human carotid artery bifurcation stenting: Correlation of lesion characteristics with embolic potential. *J Vasc Surg.* 1998; 27: 463-471.
12. Tubler T, Schluter M, Dirsch O, Sievert H, Bösenberg I, Grube E, et al. Balloon-protected carotid artery stenting: relationship of periprocedural neurological complications with the size of particulate debris. *Circulation.* 2001; 104: 2791-2796.
13. Kindel M, Spiller P. Transient occlusion of an Angioguard protection system by massive embolization during angioplasty of a degenerated aortocoronary saphenous vein graft. *Catheter Cardiovasc Interv.* 2002; 55: 501-504.
14. Willis M, Neschis D, Menaker J, Lilly M, Scalea T. Stent grafting for a distal thoracic aortic injury. *Vasc Endovascular Surg.* 2011; 45: 187-190.
15. Neschis DG, Moainie S, Flinn WR, Scalea TM, Bartlett ST, Griffith BP. Endograft repair of traumatic aortic injury-a technique in evolution: a single institution's experience. *Ann Surg.* 2009; 250: 377-382.
16. Garcia-Toca M, Naughton PA, Matsumura JS, Morasch MD, Kibbe MR, Rodriguez HE, et al. Endovascular repair of blunt traumatic thoracic aortic injuries: seven-year single-center experience. *Arch Surg.* 2010; 145: 679-683.
17. Neschis DG, Scalea TM. Endovascular Repair of Traumatic Aortic Injuries. *Adv Surg.* 2010; 44: 281-292.
18. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Blunt traumatic thoracic aortic injuries: early or delayed repair-results of an American Association for the Surgery of Trauma prospective study. *J Trauma.* 2009; 66: 967-973.
19. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Diagnosis and treatment of blunt thoracic aortic injuries: changing perspectives. *J Trauma.* 2008; 64: 1415-1418.
20. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. American Association for the Surgery of Trauma Thoracic Aortic Injury Study Group. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. *J Trauma.* 2008; 64: 561-570.
21. Crissey MM, Bernstein EF. Delayed presentation of carotid intimal tear following blunt craniocervical trauma. *Surgery.* 1974; 75: 543-549.
22. Miller PR, Fabian TC, Croce MA, Cagiannos C, Williams JS, Vang M, et al. Prospective screening for blunt cerebrovascular injuries: analysis of diagnostic modalities and outcomes. *Ann Surg.* 2002; 236: 386-393.
23. Stein DM, Boswell S, Sliker CW, Liu FY, Scalea TM. Blunt cerebrovascular injuries: does treatment always matter? *J Trauma.* 2009; 66: 132-143.

24. Biffi WL, Cothren CC, Moore EE, Kozar R, Cocanour C, Davis JW, et al. Western Trauma Association critical decisions in trauma: screening for and treatment of blunt cerebrovascular injuries. *J Trauma*. 2009; 67: 1150-1153.
25. Biffi WL, Ray CE, Jr., Moore EE, Franciose RJ, Aly S, Heyrosa MG, et al. Treatment-related outcomes from blunt cerebrovascular injuries: importance of routine follow-up arteriography. *Ann Surg*. 2002; 235: 699-706.
26. Biffi WL, Moore EE, Offner PJ, Brega KE, Franciose RJ, Burch JM. Blunt carotid arterial injuries: implications of a new grading scale. *J Trauma*. 1999; 47: 845-853.
27. DiCocco JM, Fabian TC, Emmett KP, Magnotti LJ, Zarzaur BL, Bate BG, et al. Optimal outcomes for patients with blunt cerebrovascular injury (BCVI): tailoring treatment to the lesion. *J Am Coll Surg*. 2011; 212: 549-557.
28. DuBose J, Recinos G, Teixeira PG, Inaba K, Demetriades D. Endovascular stenting for the treatment of traumatic internal carotid injuries: expanding experience. *J Trauma*. 2008; 65: 1561-1566.
29. Demetriades D, Chahwan S, Gomez H, Peng R, Velmahos G, Murray J, et al. Penetrating injuries to the subclavian and axillary vessels. *J Am Coll Surg* 188: 290-295.
30. DuBose JJ, Rajani R, Gilani R, Arthurs ZA, Morrison JJ, Clouse WD, et al. Endovascular management of axillo-subclavian arterial injury: a review of published experience. *Injury*. 2012; 43: 1785-1792.
31. Scalea TM, Sclafani SJ. Angiographically placed balloons for arterial control: a description of technique. *J Trauma*. 1991; 31: 1671-1677.
32. Morrison JJ, Percival TJ, Markov NP, Villamaria C, Scott DJ, Saches KA, et al. Aortic balloon occlusion is effective in controlling pelvic hemorrhage. *J Surg Res*. 2012; 177: 341-347.
33. White JM, Cannon JW, Stannard A, Markov NP, Spencer JR, Rasmussen TE. Endovascular balloon occlusion of the aorta is superior to resuscitative thoracotomy with aortic clamping in a porcine model of hemorrhagic shock. *Surgery*. 2011; 150: 400-409.
34. Stannard A, Eliason JL, Rasmussen TE. Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. *J Trauma*. 2011; 71: 1869-1872.
35. Seamon MJ, Pathak AS, Bradley KM, Fisher CA, Gaughan JA, Kulp H, et al. Emergency department thoracotomy: still useful after abdominal exsanguination? *J Trauma*. 2008; 64: 1-7.
36. Matsuda M, Tanaka Y, Hino Y, Matsukawa R, Ozaki N, Okada K, et al. Transbrachial arterial insertion of aortic occlusion balloon catheter in patients with shock from ruptured abdominal aortic aneurysm. *J Vasc Surg*. 2003; 38: 1293-1296.
37. Avaro JP, Mardelle V, Roch A, Gil C, de Biasi C, Oliver M, et al. Forty-minute endovascular aortic occlusion increases survival in an experimental model of uncontrolled hemorrhagic shock caused by abdominal trauma. *J Trauma*. 2011; 71: 720-725.
38. Omoigui NA, Califf RM, Pieper K, Keeler G, O'Hanesian MA, Berdan LG, et al. Peripheral vascular complications in the Coronary Angioplasty Versus Excisional Atherectomy Trial (CAVEAT-I). *J Am Coll Cardiol*. 1995; 26: 922-930.
39. Blankenship JC, Hellkamp AS, Aguirre FV, Demko SL, Topol EJ, Califf RM. Vascular access site complications after percutaneous coronary intervention with abciximab in the Evaluation of c7E3 for the Prevention of Ischemic Complications (EPIC) trial. *Am J Cardiol*. 1998; 81: 36-40.
40. Piper WD, Malenka DJ, Ryan TJ Jr, Shubrooks SJ Jr, O'Connor GT, Robb JF, et al. Predicting vascular complications in percutaneous coronary interventions. *Am Heart J*. 2003; 145: 1022-1029.
41. The PURSUIT Trial Investigators. Inhibition of Platelet Glycoprotein IIb/IIIa with Eptifibatid in Patients with Acute Coronary Syndromes. *N Engl J Med*. 1998; 339: 436-443.
42. Sherev DA, Shaw RE, Brent BN. Angiographic predictors of femoral access site complications: implication for planned percutaneous coronary intervention. *Catheter Cardiovasc Interv*. 2005; 65: 196-202.
43. Applegate RJ, Sacrinty MT, Kutcher MA, Baki TT, Gandhi SK, Kahl FR, et al. Vascular complications in women after catheterization and percutaneous coronary intervention 1998-2005. *J Invasive Cardiol*. 2007; 19: 369-374.
44. Kresowik TF, Khoury MD, Miller BV, Winniford MD, Shamma AR, Sharp WJ, et al. A prospective study of the incidence and natural history of femoral vascular complications after percutaneous transluminal coronary angioplasty. *J Vasc Surg*. 1991; 13: 328-333.
45. Ricci MA, Trevisani GT, Pilcher DB. Vascular complications of cardiac catheterization. *Am J Surg*. 1994; 167: 375-378.
46. Kelm M, Perings SM, Jax T, Lauer T, Schoebel FC, Heintzen MP, et al. Incidence and clinical outcome of iatrogenic femoral arteriovenous fistulas: implications for risk stratification and treatment. *J Am Coll Cardiol*. 2002; 40: 291-297.
47. Lam RC, Shah S, Faries PL, McKinsey JF, Kent KC, Morrissey NJ. Incidence and clinical significance of distal embolization during percutaneous interventions involving the superficial femoral artery. *J Vasc Surg*. 2007; 46: 1155-1159.