Blunt Aortic Injury occurs in less than 1% of motor vehicle crashes but is responsible for 16% of the deaths. This injury is second only to head injury as the leading cause of death after vehicular crashes. Up to 80% of patients die before their arrival at a hospital. Of those who survive the initial injury, a majority will die without definitive treatment.

Blunt aortic injury most often occurs after sudden deceleration, usually in automobile crashes. Other causes include crashes of motorcycles and aircraft, auto–pedestrian collisions, falls, and crush injury. Mechanism of Injury

Blunt aortic injury is most often due to a tear at the junction between the fixed and mobile portions of the aorta, usually near the isthmus. Injury may also occur to the ascending aorta, the distal descending thoracic aorta, or the abdominal aorta. Despite the increased use of restraint systems, the overall incidence of blunt aortic injury has remained the same during the past 12 years. The factors that appear to have a strong correlation with thoracic aortic injury are a change in velocity of 20 mph or more, impact on the patient’s side of the car, and the intrusion of the vehicular wall into the passenger compartment of 15 inches or more. Restraint devices, such as seat belts and front airbags, provide little protection in side-impact crashes. In one study of severe car crashes in which the majority of patients with thoracic aortic injury did not survive, 85% of patients with thoracic aortic injury had been involved in a crash in which the primary impact was against the side of the vehicle. It is possible that the theory regarding the increased risk of front impact is based on a relatively small subgroup of survivors and may not be the predominant mechanism in the total number of patients with blunt aortic injury to the extent previously believed. In a recent autopsy study, 42% of fatalities involving blunt aortic injury were due to side-impact crashes.

There are a number of theories regarding the pathophysiological features of blunt aortic injury in addition to the stretching effect from sudden deceleration (Fig. 1). Aortic rupture during a sudden increase in intraabdominal pressure may explain the association between blunt aortic injury and diaphragmatic rupture. A “water-hammer” effect, which involves simultaneous occlusion of the aorta and a sudden eleva-
tion in blood pressure, and the “osseous pinch” effect from entrapment of the aorta between the anterior chest wall and the vertebral column have also been theorized.\textsuperscript{10,11} Most injuries probably involve a combination of forces.

The theoretical sequence of injury involves rupture of the intimal and medial layers. After a period of unpredictable duration, rupture of the external, adventitial aortic wall occurs.\textsuperscript{3,12} In an in vitro study of porcine aortic injury, an intima–media tear occurred before complete disruption of the entire vessel in 93\% of specimens. This partial disruption occurred at a mean of 74\% of the physical stress required for complete rupture.\textsuperscript{13} These findings suggest that sufficient residual strength exists after an intima–media injury before complete rupture to allow timely diagnosis and treatment.

\textbf{DIAGNOSIS}

Untreated, approximately 30\% of surviving patients who are admitted to a hospital with blunt aortic injury will die within the first 24 hours.\textsuperscript{3} On the basis of the landmark study by Parmley et al.,\textsuperscript{3} aortography was considered the best study to identify blunt aortic injury for more than 40 years. However, aortography is invasive and requires a special team for its performance, rendering it a poor screening study. Efforts have been made to iden-
tify features found on plain chest radiographs to
guide the use of angiography (Fig. 2). It has been
suggested that the absence of the following signs
were valuable in helping to exclude the diagnosis
of aortic injury: loss of the aorticopulmonary win-
dow, abnormality of the aortic arch, rightward
tracheal shift, and widening of the left paraspinal
line without associated fracture.

However, other investigators have noted an un-
acceptable rate of missed injury and death associ-
ated with the use of chest radiography and have
recommended routine angiography for all patients
with a substantial deceleration injury. Between
7.3% and 44% of patients with blunt aortic injury
may have a normal mediastinum on chest radio-
graphy. Computed tomography (CT) is now
the diagnostic test of choice (Fig. 3A and 3B). Helical CT of the thorax is more sensitive for blunt
aortic injury than angiography and is estimated
to have a sensitivity of 100%, as compared with
92% for angiography. In one follow-up study of
patients with blunt chest trauma in whom blunt
aortic injury was ruled out by helical CT, none of
272 patients required procedures for or died of
injuries to the aorta or great vessels. This ap-
proach to diagnosis is both highly sensitive and
has a high negative predictive value.

Although some observers have suggested that
screening helical CT is overused, others note
a 28% rate of missed diagnoses and recommend
that helical CT be performed in all patients
with a history of a motor-vehicle crash at a speed of
10 mph or faster for unrestrained drivers and of
30 mph or faster for restrained drivers. The abil-
ity of helical CT to accurately diagnose blunt aortic
injury as well other serious injuries has led to its
liberal use in our institution. Other options for the
diagnosis of blunt aortic injury include transesoph-
ageal echocardiography, intravascular ultrasonog-
raphy, and magnetic resonance imaging.

Minimal Aortic Injury

With improvements in imaging technology, ever-
more-subtle lesions are being identified. The term
“minimal aortic injury” is often used to describe
a lesion of the aorta associated with blunt injury
that is believed to carry a relatively low risk of
rupture. Minimal aortic injury can be present in
approximately 10% of patients whose blunt aortic
injury is identified by helical CT. It has been re-
ported that up to 50% of minimal aortic injuries
that are identified by helical CT are missed on
angiography. Despite attempts to categorize such
lesions, minimal aortic injury and its treatment
remain ill-defined. The term “minimal aortic in-
jury” does not make a clear distinction between an
isolated intimal defect and a small pseudoaneu-
rysm. The natural history of minimal aortic injury
is unclear as well. In one study, in which minimal
aortic injury was defined as an intimal flap of less
than 1 cm with no or minimal periaortic hema-
toma, 50% of minimal aortic injuries that were
followed up had developed pseudoaneurysms by
8 weeks after injury.

It is likely that a small intimal flap in the ab-

Copyright © 2008 Massachusetts Medical Society. All rights reserved.
periaortic hematoma, lumen encroachment, or pseudoaneurysm, it is our practice to proceed with endograft coverage, particularly if the anatomy is favorable.

**PERIOPERATIVE DECISION MAKING**

Once the diagnosis is made, treatment must be properly timed. Immediate operative repair used to be the rule. However, patients often have multisystem injuries that complicate aortic repair. These injuries include head injury, exsanguinating abdominal or pelvic injury, and coexisting lung injury. Several studies have demonstrated the relative safety of a delayed approach, particularly if there are substantial coinjuries, using a regimen of beta-blockers and antihypertensive agents to decrease the shear force on the aortic wall. Fabian et al. performed a prospective study using beta-blockers with or without vasodilators to maintain a systolic blood pressure of approximately 100 mm Hg (or 110 to 120 mm Hg in older patients) and a pulse rate of under 100 beats per minute in selected patients with blunt aortic injury and a coexisting head injury, pulmonary injury, or cardiac insufficiency. In this study, no patient had an aortic rupture while awaiting repair. Patients with no other substantial coinjury that would otherwise complicate repair underwent emergency surgery.

**SURGICAL REPAIR**

Surgical repair requires intubation with a double-lumen endotracheal tube and exposure of the injury through an incision in the left fourth intercos-
tal space with unilateral ventilation of the right lung to improve access to the injury. The proximal aorta is clamped distal to the origin of the left subclavian artery. In cases in which the tear impinges on this location, the proximal aorta is clamped between the left carotid artery and the left subclavian artery. Although simple repair can sometimes suffice, placement of an interposition graft is usually necessary.

The operative repair for blunt aortic injury has undergone a number of modifications that have reduced the morbidity associated with the procedure (Table 1). Until the mid-1970s, most of these procedures were completed with an expeditious clamp-and-sew technique that usually included an interposition graft of woven or knitted Dacron to bridge the defect. Although there are isolated reports of reasonable outcomes, a meta-analysis of this technique reported an associated mortality of 16% and a striking 19% incidence of paraplegia.

Various methods of distal aortic perfusion have evolved for use during the period of aortic clamping in order to protect the spinal cord. Early techniques incorporated the use of heparin-bonded proximal aorta-to-distal aortic shunts that passively detoured blood around the site of injury (passive perfusion). These unregulated circuits modestly reduced spinal-cord ischemia but were displaced by the use of those that incorporate a blood pump (active perfusion), which further reduced the rate of paralysis, to 2.3%. Most centers with extensive experience in such procedures now use active bypass.

Active perfusion can be performed by two main techniques. Bypass from the left atrium to the femoral artery (or descending aorta) can be performed with a relatively small amount of heparin and a simple circuit with a centrifugal pump (Fig. 4A). Venoarterial bypass involves cannulation of the pulmonary artery and the femoral artery (or descending aorta). Alternatively, venous cannulation can be performed by inserting a long cannula through the femoral vein into the right atrium (Fig. 4B).

Advantages of the venoarterial bypass include the ability to cool the patient, which potentially enhances spinal-cord protection. The presence of an oxygenator in the circuit is very helpful when coexisting lung injury precludes single-lung ventilation. Venovenous bypass with minimal or no administration of systemic heparin is made possible by the use of heparin-coated tubing. In a 30-year report of our own experience with operative repair, 73 of 219 patients were supported with venoarterial bypass, and none of the procedures resulted in paraplegia.

Despite technical advances, a prospective series of 274 cases of blunt aortic injury collected from 50 trauma centers during a 2.5-year period involving a variety of operative techniques showed an overall rate of death of 31% and rate of paraplegia of 8.7%. The rate of death in patients who did not undergo surgery (not including patients arriving in extremis) was 55%. Patients with severe brain injury require continuous monitoring of measures such as intracranial pressure. They often must be nursed with the head of the bed elevated. Hemorrhage can

<table>
<thead>
<tr>
<th>Variable</th>
<th>Clamp and Sew</th>
<th>Shunt–Bypass</th>
<th>Endovascular Repair</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complication</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operative stress</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Medium</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Operative time</td>
<td>Medium</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Paraplegia</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Clinical scenario</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient with high surgical risk</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Patient with severe lung injury</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Patient with severe head injury</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Patient with challenging anatomy</td>
<td>Medium</td>
<td>Low</td>
<td>High</td>
</tr>
</tbody>
</table>

* Relative degree of risk refers to a general comparison among the three operative procedures.
Figure 4. Two Bypass Procedures for Repair of Blunt Aortic Injury.

Panel A shows a bypass procedure from the pulmonary vein (left atrium) to the femoral artery with a centrifugal pump. The main drawing depicts open repair with an interposition graft at a typical location in the isthmus. Panel B shows femoral venous–femoral arterial cardiopulmonary bypass, which is performed by inserting a long cannula through the femoral vein into the right atrium.
worsen the long-term outcome from brain injury. Patients with pelvic fractures may require angiographic embolization and can rebleed when positioned for a thoracotomy. Finally, fractures of long bones should be repaired early, but left thoracotomy can be problematic in patients in traction for long-bone fractures. Patients with multiple trauma requiring ongoing resuscitation may have wide swings in blood pressure, making the use of beta-blockers problematic at best. Thus, the patients who are arguably most in need of early repair often are poor candidates.

**Endovascular Repair**

The most significant advance for the treatment of blunt aortic injury in the past 50 years has been endovascular grafting, first described by Parodi et al. in 1991 for the treatment of abdominal aortic aneurysms.\(^38,39\) Endografts are placed through a femoral artery. A guide wire is advanced under fluoroscopic guidance to the site of injury. The position is identified on angiography and the stent graft deployed across the injured aorta, excluding it from the circulation (Fig. 5). Endovascular grafting has numerous advantages. There is essentially no physiological burden with this minimally invasive procedure. In patients with brain injury, the device can be deployed with the head of the bed elevated. Single-lung ventilation is not required. The procedure can be accomplished with minimal or no heparin, and there is no need for a bypass of any kind.

We recently reviewed the world medical literature and identified 23 reports that included five or more patients. Among 220 patients undergoing endovascular repair, there were 15 deaths, a rate of death of 6.8%.\(^40\) Several investigators have retrospectively compared endovascular and open repair. In these studies, there was a reduction in morbidity and mortality and no cases of paraplegia in the endograft groups.\(^41-44\) In our series of 39 consecutive patients (the first 20 were described previously), there were 5 deaths (12.8%) and no cases of paraplegia.\(^40\) None of the deaths were related to the endograft. Today at our institution, endovascular repair has supplanted open surgery as the primary treatment of blunt aortic injury.

There are currently some technical limitations to endografting. Injuries that occur adjacent to a sharp bend in the aorta may result in poor appos-
tion of the covered stent to the aortic wall. This leads not only to failure in covering the injury but also to device collapse.46,45 Device collapse can also occur when the endograft is oversized in comparison to the thoracic aorta.45 Another technical issue relates to the management of the left subclavian artery. Lesions adjacent to the left subclavian artery may require covering this vessel in order to achieve adequate repair. Although usually well tolerated, coverage of the left subclavian artery can result in ischemia of the upper extremity or territory perfused by the left vertebral artery. In such cases, bypass from the left common carotid artery to the left subclavian artery may be required. Patients with a dominant left vertebral artery (relative to the right) should be considered for left subclavian artery revascularization before coverage. Fortunately, the majority of our patients have been successfully treated without left subclavian artery coverage.40

Until recently, the only thoracic endograft that had been approved by the Food and Drug Administration (FDA) and that was available in the United States was the TAG device (W.L. Gore and Associates). However, this device was approved for use in nonruptured aneurysms of the thoracic aorta. The smallest TAG device is designed for a patient whose aorta has a diameter of 23 mm in the nonaneurysmal portion. In our experience with blunt aortic injury, the average aortic diameter is about 23 mm, with a range of 19 to 30 mm.40 To avoid oversizing and device collapse, we often used smaller endograft cuffs designed for and approved by the FDA for use in the treatment of abdominal aortic aneurysms. Recently, two additional endografts — the Talent Thoracic (Medtronic Vascular) and Zenith TX2 (Cook) — were approved by the FDA for the treatment of thoracic aortic aneurysms and penetrating ulcers. The approval of these devices provides a greater variety of device sizes than were previously available. However, there are no FDA-approved devices carrying an indication for treatment of blunt aortic injury.

There are several potential pitfalls related to off-label use of FDA-approved devices: the abdominal devices, while often of satisfactory diameter, tend to be short and require overlapping of grafts to achieve the appropriate length. In addition, the abdominal devices tend to be on shorter delivery systems, which could potentially create difficulty in treating unusually tall patients. None of the available devices are particularly suited to adaptation to the tight angulation in the distal aortic arch often encountered in young patients. Devices that are designed to be approved for trauma indications will need to address these elements.

Finally, the durability of endografts is unknown. There are questions about long-term device integrity as well as the natural history of the aorta itself after this type of injury and repair. These issues are particularly important considering the relatively young age of trauma patients, as compared with patients with aneurysmal disease. For now, we recommend that patients receive lifelong serial imaging (Fig. 3C and 3D).

In summary, the treatment of blunt aortic injury has evolved considerably. It is our opinion that endovascular repair will soon be the therapy of choice for most patients. Device technology should continue to evolve, providing appropriate-sized, conformable devices. Surgeons with endografting skills should be prepared with a wide range of device diameters to accommodate small aortas in young patients and be prepared to forgo endografting for injuries that are anatomically unsuitable. With proper preoperative planning, endograft repair provides even severely injured or frail patients with an opportunity for repair and is expected to substantially reduce procedure-related rates of death and paraplegia.

Dr. Neschis reports receiving grant support from W.L. Gore and Associates. No other potential conflict of interest relevant to this article was reported.
CURRENT CONCEPTS