Changes in the Management of Injuries to the Liver and Spleen

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At the completion of my residency training in 1976, there were relatively few controversies in the treatment of injuries to the liver and spleen. Diagnosis of these injuries usually could be made by physical examination, positive diagnostic peritoneal lavage (DPL), or at operation mandated by a penetrating wound. If injury to the spleen occurred, splenectomy was indicated. Diagnosis of liver injury was likewise noncontroversial, and only the methods of treatment of the hepatic lesions engendered debate. But by the end of the last century, virtually none of the dogma believed to be unequivocally true 25 years earlier was practiced. The majority of splenic injuries were detected by CT scan and treated without operation. Liver injuries that resulted in several liters of blood and bile in the abdomen were observed if the blood pressure could be maintained with several units of blood. A surgical Rip Van Winkle, who awoke in 2000 after 25 years of slumber, would never have believed the radical changes that occurred in the treatment of injuries to these solid organs, whose diagnosis and management had once seemed so straightforward.

The purpose of this discussion is to review the changes in the treatment of injuries to the liver and spleen that occurred during the past century. It is hoped that the exhaustive literature search that is the lynchpin of this presentation will serve as an evidence-based review that either validates or challenges some of our current concepts about solid organ injury management.

METHODS OF REVIEW

Full-length papers (not abstracts) pertaining to injuries of the liver and spleen were reviewed. Citations were extracted from the *Current List of Medical Literature* and its successor, *Index Medicus*, and from the bibliographies of other publications on these injuries. Virtually every publication on hepatic and splenic trauma written in the English language before 1950 was reviewed, as were several sentinel papers written in German and French. Most of the major reports on these injuries in the latter half of the century were also examined, but the prodigious growth in publication on these topics precluded a complete literature review. Nonetheless, more than 500 reports were reviewed for this article.

A comparison of the etiology of splenic rupture and liver hemorrhage between the first and latter halves of the 20th century is also instructive. From 1930 to 1940, citations on spontaneous splenic rupture greatly outnumbered those on traumatic injury. Splenic hemorrhage secondary to malaria, typhoid, and mononucleosis were reported much more commonly than hemorrhage produced by trauma. The 1930s produced fewer than 30 citations on liver and spleen injuries in the English language; the 1990s produced more than 1,300 reports on those topics.

MECHANISM OF INJURY: CHANGES IN PATTERNS

The changes in mechanism of injury are illustrated (Fig. 1) in three time intervals: the early, middle, and latter portions of the last century. In the early period, nearly one-third of all splenic injuries and one-fifth of liver injuries reported were caused by a variety of mechanisms classified as miscellaneous. These include industrial and farm injuries, falls, and mechanisms other than motorcycle or motor vehicle crashes. Gunshot wounds were much less common than were reported later in the century. By midcentury, miscellaneous injuries became a minor part of the reported cases as a percentage because the number of liver and spleen injuries increased dramatically. Stab wounds as a mechanism of penetrating trauma increased greatly, accounting for 40% and 15% of liver and spleen injuries, respectively.

What is often underappreciated is the impact of mechanism on mortality rates. The increase in stab wounds reported had a dramatic impact on improvements in mortality data, particularly for liver injuries.

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Abbreviations and Acronyms

DPL = diagnostic peritoneal lavage
EAST = Eastern Association for the Surgery of Trauma
NOM = nonoperative management

Before World War II, the mortality rate for liver injury was often reported at 30% to 40%.[1-11] One of the larger articles on hepatic trauma published a few years after World War II reported a mortality rate of only 10%.[12] Although the results of this report were hailed as a major advance, they may be somewhat deceiving. Of the eight blunt injuries, only three were severe. Thirty-four stab wounds occurred, and no patient with an isolated stab wound died, which greatly improved overall results. Amerson and Blair[13] described 189 patients treated between 1947 and 1958, with a total mortality rate of 16.4%. Twenty-two patients with blunt injury had a mortality rate of more than 45%, and the 99 patients with gunshot wounds had a 21% death rate. But none of the 63 stab wound victims died, greatly lowering the total mortality rate. The first huge series of civilian liver injuries[14] reviewed the experience from the Jefferson Davis Hospital in Houston, TX, from 1939 to 1961. The overall mortality rate from 640 consecutive patients was 17.3%. Mortality rates from gunshot wounds and blunt trauma were 26% and 45%, respectively, but the 296 stab wounds had a mortality rate of only 3.4%.

Reports from the latter portion of the century show a major decline in stab and gunshot wounds, with a tremendous increase in blunt injuries. Because blunt trauma generally has the highest mortality rate, any improvement in mortality is likely from improvement in management.

DIAGNOSTIC ERAS

As with changes in mechanism, differences in diagnostic modalities have been dramatic over time; such changes in diagnosis have had a major impact on not only differences in management but in reported mortality rates. Although numerous iterations of diagnostic eras could be conceived, four will be considered (Table 1).

Figure 1. The mechanism of spleen and liver injury from selected series in three periods of time. In the early period there were a large number of miscellaneous injuries, which accounted for less than 1% during later periods. In the mid-portion of the 20th century, stab wounds were commonly encountered in liver injuries. Blunt trauma is now the predominant mechanism of injury for both spleen and liver injury. GSW, gunshot wound.

Table 1. Diagnostic Eras

1) Operation or autopsy
2) Primitive diagnostic efforts
3) Diagnostic peritoneal lavage
4) Focused imaging techniques

From the beginning of operative treatment of traumatic wounds in the late 19th century through World War I, diagnosis was made definitively by either operation or autopsy. Although one could assert that is still true, in that era, there were no major reports of other diagnostic strategies. Operations were usually based on physical signs or on mechanism of injury, as in the case of penetrating wounds. Emphasis in the literature was on physical findings that might suggest solid organ injuries, given the lack of other diagnostic methods. Ballance's sign described dullness to percussion or shifting dullness in the left upper quadrant. Kehr's sign, referred to pain to the left supraclavicular region, was believed to be an important sign of splenic injury. Tenderness and shock were also clinical indications of the need for operation.[4,8-10,12]

Not surprisingly perhaps, several reports based on autopsy studies concluded that nonoperative therapy for liver injuries was uniformly fatal. One early study reported a 100% mortality rate for nonoperative treatment, with a 50% mortality rate for patients who had operations. Although several reports assumed that some injuries must heal without operation, the lack of precise diagnostic studies made it difficult to accurately determine the incidence of either liver or spleen injury or their actual mortality rates. The next phase of attempts at diagnosis involved an indirect effort to establish a solid organ injury by use of radiographs or by attempts to confirm the presence of hemoperitoneum by needle puncture. Several papers in the 1940s emphasized the following radiologic features for splenic injuries: obliteration of the splenic shadow; indentation of the gastric bubble; reflex distention of the stomach; and tenting of the left diaphragm. But there were few reports on radiologic features of liver injuries.[15]

Beginning in the 1930s, many surgeons attempted to aspirate blood from the peritoneal cavity of trauma victims with suspected solid organ injury. Whether by paracentesis or four-quadrant tap, confirmation of blood in the peritoneum mandated operation. The diagnostic accuracy rate for blunt liver and spleen injuries ranged from 30% to 70%, but all reports on this technique noted that its accuracy did not approach 100%.The inaccuracy of these indirect means of diagnosis led to the development of DPL.

Root and colleagues[16] developed the technique in the laboratory by infusing blood into the peritoneum and then aspirating the contents after peritoneal lavage of the cavity with saline. This technique proved highly sensitive for blood in the peritoneal cavity and was the diagnostic method of choice for detection of hemoperitoneum for more than 30 years. Although DPL was very sensitive for blood, its high degree of accuracy made it less useful at times. Virtually all surgeons who used DPL noted that in some patients, injuries that produced bleeding had either ceased or the injuries were so inconsequential that no therapy was required. Nonetheless, DPL was a major advance because it greatly diminished the incidence of missed solid organ injuries in the era before accurate scanning was available. The impact of DPL on reported improvements in mortality rates for liver and spleen injury cannot be underestimated. Prompt use of this technique decreased missed injuries and allowed rapid operative planning in emergent cases. Clearly, treatment improved during this era, but patients with minor injuries were always treated operatively and rarely died. This inclusion of less severely injured patients certainly enhanced reported mortality rates.

The current diagnostic era relies primarily on imaging techniques to aid in diagnosis of solid organ injury. The use of abdominal ultrasound has proved extremely efficacious for detecting blood in the peritoneal cavity. This technology has the advantage of portability, and when done by the surgeon or emergency physician, can become a part of the physical examination. But abdominal ultrasound lacks specificity in terms of predicting the source of blood and cannot grade organ injuries.

CT scanning has become the gold standard for diagnosis of solid organ injury. CT scanning allows reasonably accurate grading of organ injuries and provides crude quantitation of the degree of hemoperitoneum. Additionally, the use of oral contrast material permits a degree of diagnostic accuracy in excluding visceral injuries. CT scanning is mandatory for patients with blunt trauma whose solid organ injuries are to be managed nonoperatively. CT has also been useful for detecting missile tracts in penetrating trauma patients. Such information is imperative for surgeons who want to attempt nonoperative management of penetrating wounds.[17-20]

MANAGEMENT OF LIVER INJURIES

The management of hepatic trauma can be divided into three eras: from the beginning of operative treatment to World War II; the postwar period to the mid-1980s (characterized by aggressive operative treatment); and from the late 1980s to the present. In this latter period, there have been several major philosophical changes in treatment that represent radical departures from previously accepted standards of care.

Late 19th century through World War II

Some of the advances in the operative treatment of hepatic injuries developed from the late 19th century through World War II are outlined in Table 2.[1,3,20-26] One of the notable aspects of this historical list has been the rediscovery of many of the concepts that were introduced about a century ago but failed to find a niche in the treatment of liver injuries, or were attempted and then subsequently rejected. Examples of the former included creation of an omental pedicle to be sutured into a liver wound, which was described in 1910,[22] but did not gain popularity for another 75 years. Topical fibrin was introduced in 1915 and did not gain acceptance. Although it is occasionally used today, it is not regarded as standard therapy. Packing was reported in the late 1800s in Europe and in the United States in 1906.[25] It was a mainstay of therapy in World War I and in the period between the world wars. During World War II, the value of direct repair of liver wounds along with drainage of bile was highlighted. The resultant dramatic improvement in mortality provoked condemnation of packing as a means of treating liver injuries.

The most common treatment administered to nearly 600 civilian patients treated from the mid-1930s to 1945 was drainage alone followed by suture repair of the liver (usually with catgut). The development of absorbable hemostatic agents led to an explosion in their use.

Table 2. Advances in Operative Treatment of Liver Wounds from 19th Century Through World War II

Date	Surgeon	Advance
1888	Langenbuch	Mass ligature controlled liver bleeding
1890	Clementi	Introduced inflow occlusion by clamping portal triad
1894	Ceccharelli and Bianchi	Tamponade bleeding liver with decalcified bone sutured on superior and inferior liver surface
1896	Kousnetzoff and Penski	Special liver needle to suture hepatic wounds
1902	Beck	Rubber catheter suture around the liver wound for compression
1902	Kocher	Gastrointestinal clamp left on liver to compress bleeding
1905	Gillette	Mattress through liver and skin (two survivors)
1906	Schroeder	Perihepatic packing
1908	Pringle	Compression of artery and vein for inflow occlusion
1910	Boljarski	Omental pedicle sutured over liver wound
1911	Cushing	Topical striated muscle on bleeding liver for hemostasis
1913	Halstead	Rubber under packing
1915	Grey	Topical sheep fibrin
1918	Harvey	Topical beef fibrin
1939	Seegers	Topical thrombin
1943	Frantz	Oxidized cellulose (Oxycel)
1944	Ingraham	Topical thrombin and Gelfoam (absorbable sponges)
1945	Light and Prentice	Gelfoam

Impact of World War II

The mortality rate reported for operative treatment of liver injuries in World War I was 66.2% when packing was used as the primary mode of therapy. After World War II, Madding and colleagues[27] reported on 829 operations performed for liver injuries by the Second Auxiliary Surgical Group. Although gauze packing was used in 28% of the total group of treated patients, this method was predominantly used early in the war years. In 1941, more than 34% of patients were packed; that number declined to 9.6% by 1945. Only a small percentage (about 5%) required hepatorrhaphy in either time period. Drainage was used in nearly 90% of patients by the end of the war. So, several trends developed that were extended into civilian practice after the war: abandonment of gauze packing; suture control only for active bleeding; and nearly uniform drainage of all wounds. This collected group of patients suffered a 27% mortality rate. Although the death rate was high by current standards, it was a dramatic improvement from the two-thirds who died in World War I. Likewise, the authors noted only a 9.7% mortality rate when the liver wound was isolated. Interestingly, these authors gave little credit for improvement in mortality rates to general advances in perioperative care. They asserted a direct link between two operative lessons and mortality, ie, discontinuation of gauze packing and routine use of drains. These lessons were widely applied in the civilian experiences for the next 40 years.[28-31]

Operative treatment: 1945 to 1985

After the end of World War II, there was a great increase in hepatic trauma from both blunt and penetrating injuries. Beginning in the 1950s, multiple large series of liver injuries were reported.[14,31-36] In addition to the standard treatment of hepatorrhaphy for bleeding, packing with absorbable hemostatic agents, and routine drainage of bile leaks, several additional strategies were developed (Table 3). In response to more serious injuries undoubtedly caused by more gunshot wounds and high-speed motor vehicle crashes, additional treatment options were required. Better resuscitative techniques and improved blood banking made early death from shock less common and increased the number of patients with operable, severe liver injuries. So increasing numbers of patients died of hemorrhage during this period. Although most reports did not discuss the source of hepatic hemorrhage, four types were noted: arterial hemorrhage; major venous including the retrohepatic caval injury; ooze from injured or devitalized tissue; and a combination of these. Operative treatments were often developed to treat specific patterns of hemorrhage.

Table 3. Surgical Trends from 1945 to 1985: Period of Aggressive Surgical Treatment

No gauze packing
Uniform drainage
Increased use of liver debridement
Tractomy
Popularization of omental patch
Brief rise of anatomic resection
Selective hepatic artery ligation
Atriocaval shunt for retrohepatic vena caval injury
Rediscovery of temporary inflow occlusion

Treatment of arterial hemorrhage

Several specific modalities began to be used more often to treat arterial bleeding. Hepatorrhaphy was used with increased frequency. When the arterial bleeding occurred deep within the hepatic parenchyma, a tractomy was advocated to expouse and suture ligate the arterial flow. But control of deep arterial bleeding was often technically difficult to accomplish.[31,34-36]

In response to futile attempts to directly suture ligate arterial bleeding, Dr Aaron's group performed ligation of the hepatic artery.[37] Initially performed at the Louisville General Hospital to control arterial hemorrhage from a ruptured hepatic adenoma, Mays found this technique useful to control arterial bleeding in trauma patients. A literal explosion in its use occurred in Louisville, and surgeons there proposed it to prevent rebleeding.[38,39] A high rate of infection led to reconsideration of its use, and it was subsequently used less frequently, although it remained an operation that could occasionally be life-saving.[40]

Major venous bleeding was recognized as a major source of mortality, particularly in patients who had been in high-speed motor vehicle crashes. The nearly uniform lethality of retrohepatic vena caval injuries with attempt at direct repair led to the development of the atriocaval shunt. This technique, developed by Schrock and associates,[41] theoretically bypassed the caval injury and allowed direct suture repair of the cava itself and main hepatic veins. The operation required opening the chest to expose the atria. This bicavitary exposure accelerated hypothermia and coagulopathy in many patients. Consequently, the mortality rate remained high, but the concept of direct repair of this deadly injury was very important.

Both previously mentioned bleeding problems often were treated initially with temporary inflow occlusion by clamping the portal triad. The concept of inflow occlusion actually predated Pringle,[26] but his work published in 1908 was rediscovered and popularized in the 1960s after rarely being mentioned in the literature for more than 50 years.

Diffuse bleeding from damaged or devitalized liver increasingly required surgical treatment. Reports on civilian liver injuries from the 1950s generally cautioned against debridement of damaged liver for fear it would worsen preexisting hemorrhage. Absorbable gauze packing and drainage were mostly used for this problem. As the forces of injury increased, other techniques were required. Resectioned debridement was increasingly used. There was a brief flurry of activity with use of major anatomic resections, but the high mortality rate of this procedure led to discontinuing its use in most American centers.[42,43] The omental pedicle described for liver injury in 1910 and mentioned occasionally through the years was reintroduced by Stone and Lamb[44] and gained widespread popularity.

Viewed from the perspective of several decades removed, this era produced relatively few techniques in widespread use today. Clearly, debridement of devitalized hepatic tissue is a concept that remains valid but seems to be required less frequently than in the past. Omental pedicle flaps are very useful in a variety of clinical scenarios in which operation is required. The atriocaval shunt is still widely mentioned as a procedure for retrohepatic caval injuries, but its very high mortality rate and the technical tour de force required for its placement (particularly because it's not often needed) dictates its infrequent use today. Likewise, selective hepatic artery ligation is rarely used today,[45] and tractomy is rarely needed. Hepatic lobectomy for trauma is almost never done unless the injury itself performs the dissection.

Advances in treatment: 1985 to present

In the 40 years from World War II to 1985, there were enormous advances in diagnosis and treatment that markedly reduced the lethality of hepatic injuries. The advent of DPL reduced missed injuries, but was beginning to be challenged by CT scanning. CT scanning allowed greater specificity of diagnosis and permitted a range of therapeutic options based on the ability to determine degree of hemoperitoneum and grade of injury that had not been imagined previously. Concurrently, the high incidence of nontherapeutic operation performed as a consequence of DPL was increasingly recognized. Deaths from perihepatic infection had diminished, presumably because of better debridement of devitalized tissue, better resuscitation, better use of drains, and perhaps better antibiotic therapy. Despite these advances, patients were still dying in great numbers secondary to hemorrhage from the liver, from both blunt and penetrating trauma. Several major problems were identified as causative factors in death from hemorrhage. There were numerous deaths from the vicious cycle of hypothermia and coagulopathy produced by major hepatic bleeding.[46] Clearly, in many patients, direct control of bleeding using standard surgical therapy was not efficacious. Likewise, techniques advocated for control of perihepatic venous injuries usually failed. New strategies were once again needed to address old problems and newer ones created by more seriously injured patients.[47-49]

Several reports in the late 1980s and early 1990s focused on these severe liver injuries and the continuing problem of death from hemorrhage. Four strategies (Table 4) that appear to have significantly decreased the mortality rate associated with hemorrhage evolved over the latter part of the century.

Table 4. Treatment Trends: 1985 to Present

Direct repair of perihepatic venous injuries
Perihepatic packing and damage control strategies
Arteriographic embolization of hepatic arterial hemorrhage
Nonoperative treatment

Direct repair of perihepatic venous injuries

Injuries to the perihepatic veins remain an unsolved problem. Although major venous injuries are fairly uncommon, they are, nonetheless, highly lethal. Atriocaval shunting was generally regarded as the optimal means to treat this problem, but a review of its results are bleak. Although some results appear reasonable given the seriousness of the injury, the combined mortality rate reported in the literature is high. The results from San Francisco General Hospital from 1968 to 1987 using this technique on 27 patients produced a mortality rate of 55%.[50] This was the best experience reported in the literature. Cogbill and associates[47] reported on a multicenter experience that treated 38 patients, with only 4 survivors. No patient with blunt injury survived when a shunt was placed. Extracting mortality data from dozens of papers on liver injuries disclosed more than 412 cases of shunt placement, with an 88% mortality rate. Despite an occasional favorable report on shunting,[51,52] most reports through the years eschewed the use of atriocaval shunting in favor of direct methods of repair.[53] Bethea[54] reported three cases of direct repair using inflow occlusion, with all patients surviving. A review reported from Charity Hospital noted a 40% survival rate with perihepatic venous injuries treated without a shunt, which was comparable to the best reported results with a shunt. Coln and colleagues[55] treated four children successfully using a direct repair technique. Frustrated by their results with the atriocaval shunt, Pachter and coworkers[56] treated five patients without a shunt, and all survived. This group noted four principles that were believed important in their good outcomes: compression of the liver wound while the patients were being resuscitated; early recognition of the venous injury; portal triad occlusion with steroids and topical hypothermia for liver protection; and the use of finger fracture through hepatic parenchyma to allow access to the bleeding veins. Results from these authors have inspired more direct repairs of perihepatic venous injuries.[57,58]

Our group in Louisville reported a small series of patients using a different technique of direct repair.[59] Three patients were successfully treated by clamping bleeding hepatic veins with fine vascular clamps to control the hemorrhage. An omental flap was then created, the liver was packed, and the abdomen covered with a temporary sheet closure. The clamps were removed within 24 hours, and no patient had major bleeding after the clamps were removed. Three additional patients have now been successfully treated with this technique. Direct repairs appear to offer a better alternative to atriocaval shunting in patients who require operative treatment for this problem.

Perihepatic packing

As previously noted, perihepatic packing was a concept popularized early in the 20th century by Schroeder,[25] but it was actually used in the late 1800s. The high mortality rate before World War II appeared to cause its virtual abandonment. But several large series of hepatic wounds continued to report sporadic use of this technique. In a 1976 review of 625 patients with liver injury, Lucas and Ledgerwood[34] noted that 3 patients survived after packing. In 1979, Calne and colleagues[60] advanced the concept of packing before transfer to a center for advanced treatment, with all 4 patients treated by this method surviving. By 1981, Feliciano and associates[61] called for a reappraisal of this technique after 9 of their 10 seriously injured patients treated by packing survived. In rapid succession, two additional reports[62,63] had more than 80% survival rate of patients with difficult bleeding injuries using packing. The term *damage control* was used to describe this strategy for those patients with multiple injuries, coagulopathy, hypothermia, and diffuse oozing from the liver and other areas.[64]

Because the indications for packing included most seriously injured patients, the reported mortality rate rose. By 1988, Feliciano and associates[65] reported on a followed group of 66 patients who were packed; 17 died early in the operative or postoperative period. The mortality rate of those who survived 24 hours was 47%. Successive reports[66-70] from several institutions showed a high mortality rate, but concluded that innumerable patients were being salvaged who previously would have died.

Several lessons were learned from these extensive experiences with perihepatic packing.[66,69] First, patients must be packed before their survival is unlikely to occur under any circumstances. Packing was excellent for diffuse ooze or from venous bleeding, but was not effective for arterial hemorrhage. Pack removal should occur much sooner than had been practiced in the pre-World War II era to avoid infection.[71] Some advocated pack removal as soon as associated coagulopathies could be corrected, but the majority of reports advocated leaving packs for 1 to 2 days, for a mechanical effect as well.[69,72,73]

The tremendous amount of resuscitation these patients received and the volume occupied by the packs created another series of problems. Attempts at fascial closure of the abdomen usually met with failure. Even closure of the skin often led to an increased intraabdominal pressure and a constellation of clinical events labeled as "abdominal compartment syndrome."[74] Many problems associated with this condition could be ameliorated by leaving the abdomen open and covered with some type of temporary atraumatic material. The open abdomen itself is responsible for the considerable late morbidity rate in survivors. Intestinal fistulas and huge abdominal wall hernias have been among the most vexing. Recent efforts have focused on earlier attempts at abdominal wall closure to prevent loss of domain and development of huge midline hernias. The use of vacuum-assisted closure devices appears to be a promising step in this direction.

Even 20 years after the resurrection of packing as a treatment alternative, it remains an important part of the armamentarium of surgeons in managing difficult hepatic injuries.

Angiography and transcatheter embolization

Another important advance of the last 20 years has been the development of transcatheter embolization for bleeding arterial injuries within the liver. Numerous reports[75-78] on this technique have been published, and technical success rates are usually greater than 80%.

Mohr and coworkers[79] reported on 26 patients who underwent angiographic embolization either early or later in the course of treatment. Two patients bled again and were successfully treated by a second angiogram. These authors noted that the mortality rate of this group of patients was low, but considerable morbidity occurred. These complications included five patients with hepatic necrosis, four of whom had an infarcted gallbladder. Gallbladder infarction has been noted in several other experiences.[80,81]

Indications for this procedure vary to some degree among institutions, but often include the presence of a contrast blush on CT scan, particularly in patients who have required blood transfusion. Some centers recommended angiography for most severe liver injuries in blunt trauma patients. Angiography may be helpful in conjunction with other treatment modalities such as packing or nonoperative treatment.[82-85]

Nonoperative treatment of blunt liver trauma

Nonoperative treatment, thought to be a novel concept in the past 15 years, also represents ideas once practiced and then abandoned. Tellmans reported experimental observations in 1879 in which wedges of liver were removed from animals and the abdomen closed. He noted most animals spontaneously ceased bleeding promptly and generally survived. Clinically, Hinton,[86] in 1926, noted liver injuries were relatively common and advocated "conservative" or nonoperative treatment because, as he noted, most bleeding from the liver spontaneously ceased. Numerous reports discussed a selective approach to the management of hepatic injuries based on clinical factors such as hemodynamic stability.

The countercurrent argument was that nonoperative treatment was associated with virtually a 100% mortality rate, so all patients with suspected or diagnosed liver injuries must have an operation. Improved mortality rates during and after World War II assured the primacy of operative treatment.

Three observations prompted the move toward nonoperative treatment. Several reports of injuries to children demonstrated the efficacy of nonoperative treatment for blunt injuries.[87,88] First, the practice of nonoperative treatment was initially advocated for splenic injuries and then extended to liver wounds. The success in children led to attempts of nonoperative treatment in adults. Second, the high rate of nontherapeutic operations in many patients with blunt hepatic injuries was not in patients' best interest. Third, the advent of CT scanning greatly facilitated both diagnosis and grading of injuries and gave some reassurance that the intestinal injuries had not occurred.

Grading and stratification of both liver and spleen injuries represented a major advance[89] and furthered the concept of selective treatment based on injury severity. Initially, nonoperative treatment was used only in patients who required no blood transfusion, and only patients in the most stable condition received such treatment. Success with the method led to liberalization of indications for observation alone. In 1990, Knudson and colleagues[90] reported on 52 adults treated in the decade of the 1980s with no delayed hemorrhage or deaths. Additionally, her group collected 21 reports on nonoperative treatment including both children and adults. In the 286 combined patients, only 1 patient died after starting warfarin sodium therapy postinjury. Myriad reports have subsequently validated the safety and efficacy of this approach.[91-100] Currently, up to 90% of patients with blunt liver injuries are managed nonoperatively.

Although most reports on nonoperative therapy have focused on avoidance of operation as the primary benefit, our group[101] has suggested that mortality rates may be directly improved by this treatment as well. Despite the fact that the number of total liver injuries treated has increased in the past 25 years, and the number of severe liver injuries as judged by CT scanning has slowly increased, the mortality rate has declined. Particularly, the number of patients who required operative treatment for perihepatic venous injuries has declined because we have used observation as the primary treatment. In our review of 25 years' experience,[101] we treated 2.7 juxtahepatic venous injuries per year from 1975 to 1994, but now operate on only 1.5 patients per year. Because the number of patients and the grade of injury based on CT scan have increased, it would seem that the number of venous injuries should have actually increased. It is our hypothesis that many patients with venous injuries that are low-pressure wounds cease bleeding spontaneously and heal if they are left alone. The anecdotal reflections of many experienced trauma surgeons note that some patients who were stable preoperatively die during operation, after the venous injury is disturbed and profuse bleeding is initiated.

Current protocols for nonoperative management of hepatic injuries are relatively standard in most trauma centers. The key feature in the ability to offer nonoperative treatment is hemodynamic stability. Patients who are not stable must have operation or angiography (in selected patients). Most protocols will allow continued observation with up to 4 U of blood transfusion for the hepatic injury itself. CT scans should be performed to confirm the diagnosis and attempt to exclude other injuries, particularly to the hollow viscera. Patients are usually admitted to a closely monitored unit and kept on bed rest for several days.

The success rate of nonoperative treatment has been remarkably high. The necessity for operations for ongoing hemorrhage has been reported to be from 5% to 15%. There remains a concern over missed bowel injuries that have been reported from 1% to 3%.[102-106]

Nonoperative treatment has created additional problems less frequently encountered in the operative era. Hemobilia is seen not uncommonly and may require angiographic embolization. Perihepatic fluid collections might need to be drained. Endoscopic retrograde cholangiopancreatography (ERCP) is occasionally needed for larger bile leaks. Our unit reported that a significant number of patients treated nonoperatively needed some form of intervention to treat secondary problems created by the initial injury.[107,108] Major bile collections should not be allowed to remain in the abdomen.[109] We have practiced routine drainage of large collections through laparoscopy and have treated more than 30 such patients by this method. The procedure is performed several days after admission using a gasless system. Laparoscopy permits thorough irrigation and suctioning of old blood and bile, which we remove from around the liver, the gutters, and the pelvis. A suction drain is usually placed as well. No patient has bled after the procedure, air embolism has not occurred, and no technical complications have been observed. The amount of fluid removed has ranged from 800 to 4,500 mL. In addition to subjective improvement, many patients have decreases in heart rate, better respiratory mechanics, decreased leukocytosis, and decreased temperature after this procedure.

There are numerous unresolved questions in the nonoperative management of these patients, including the importance of bed rest, the timing of return to normal activities and exercise, and the role of followup scans. But nonoperative treatment seems to be a secure treatment at this point. There are no firm recommendations about followup scans.[110] Our unit generally obtains such scans, but we have no defined protocol to recommend their timing. Likewise, we have patients avoid strenuous activity for several months, but this recommendation is intuitive rather than data-driven.

Nonoperative treatment for penetrating hepatic injuries

Nonoperative treatment of abdominal stab wounds has been practiced successfully in numerous centers for many years, and some patients with liver injury have been so treated. Nonoperative treatment of gunshot wounds has been more controversial. Demetriades and associates[111] reported 36 patients with gunshot wounds to the liver, of which 16 were initially treated without operation. Five required delayed operation. Four patients required operation because of bleeding and one developed abdominal compartment syndrome. Because nearly a third of the patients assigned to nonoperative treatment failed, this result would not seem strongly positive, but the authors believed it was a useful practice in selected patients. Moore,[112] in an accompanying editorial, questioned the wisdom of this form of management. At least one other experience on this treatment strategy has been reported with reasonable success.[113]

The use of CT scanning potentially permits the missile tract to be outlined, and if the tract is confined to the liver and the patient is stable, operation may be avoided. Our unit has had two patients with missed colon injuries in a small group of patients treated nonoperatively. This led to discontinuation of the practice of nonoperative treatment of hepatic gunshot wounds. Clearly, this is a technique that must be used with great caution in highly selected patients.

Recommendations for treatment of liver injuries

The simple algorithm in Figure 2 outlines current recommendations for treatment of liver injuries. Obviously, clinical circumstances and capabilities within the treating institution will have an impact on the treatment of individual patients.

Changes in mortality from hepatic trauma

A steady decline in mortality rate has occurred because of the inception of treatment of liver injuries (Fig. 3).[1,2,4,6,8,10,12,14,27-29,31-36,101,114-123] In 1987, Edler[114] collected 543 cases from the world's literature, with a mortality rate of 66.8%. Several collected reports before World War I disclosed mortality rates of 60% to 80%, although it appears many of the same patients were reviewed by each author.[115-118] The mortality rate during World War I was 66.2%.

Collected series between the world wars disclosed 416 patients, with a mortality range of 30% to 81% in these reports, and an overall mortality rate of 69%. Several civilian experiences reported in the mid-1940s had a mortality rate of 55%. The classic paper by Madding and associates[27] on their experiences during World War II, reported a marked decline in mortality to 27% of 829 patients treated. The experiences of several large civilian reports in the 1960s, 1970s, and 1980s show declining total mortality rates of 12.7%, 8.7%, and 6.0%, respectively. Beginning in the 1970s, many reports began to dissect liver-related mortality from total mortality. In the last three decades of the 20th century, the liver mortality

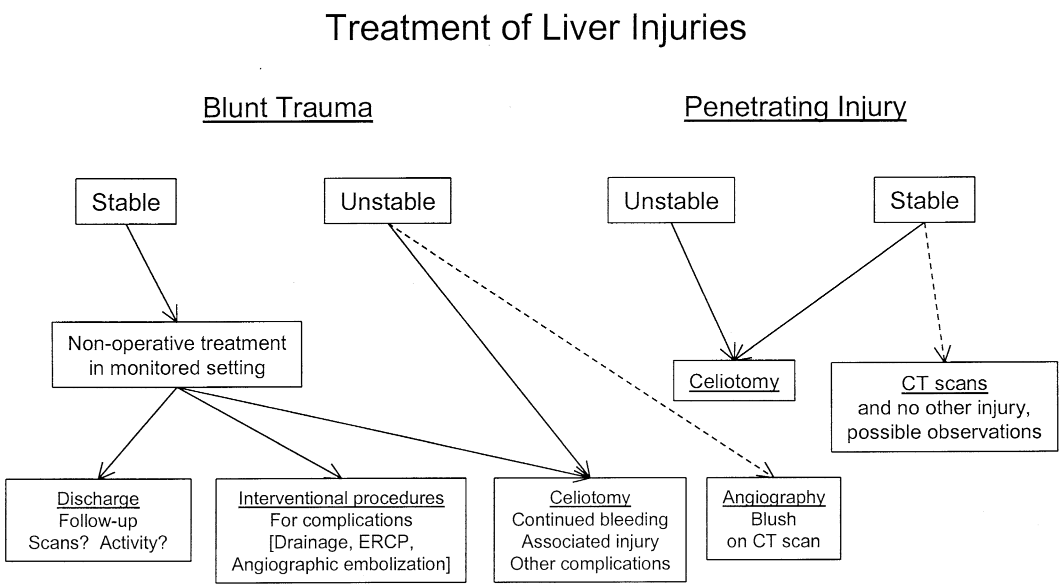

Figure 2. The demarcation point for management of either blunt or penetrating injury is hemodynamic stability. The broken lines indicate treatment protocols advocated by some that generally are not recommended by most authors.

rate was reported at 6.6%, 5.1%, and 4.2%, respectively, in these collected series. Richardson and colleagues[101] demonstrated a similar trend in a 25-year analysis of a database of more than 1,800 patients with liver injury at the University of Louisville.

The focus in the literature in the past 15 years has been on the management of complex injuries and the unsolved problems of patients with grades IV and V injuries.[124-128] Whether it is reasonable to expect further improvements in hepatic-related mortality is difficult to predict.

INJURY TO THE SPLEEN

Historical references yield conflicting information on the origins of splenectomy, although mention of the operation dates back to the mid-16th century.[129] Bessel-Hagen reported 37 splenectomies for ruptured spleen in the German literature in 1900, although the causes of the ruptured spleens were not elucidated. In fact, reports of splenic rupture until the time of World War II were more likely from mononucleosis or malaria than trauma. Regardless of the primacy of splenectomy for trauma, the operation was performed with some frequency by the early 20th century, with gradually improving results (Table 5). Although the total mortality rate is high in many of these series, then, as now, most of the deaths were from associated injuries. Although there are few reports of a large number of splenectomies before World War II, in fact, most of them were collected cases that generally analyzed the same reports and added a handful of their own. Before the mid-1930s, it appears there were less than 500 cases of ruptured spleen associated with trauma reported in the English literature; in fact, some of these traumatic injuries appear to have damaged abnormal spleens. The mortality rate for these patients was approximately 38%.

In a manual entitled *Abdominal and Genitourinary Injuries*, published by the National Research Council in 1942, it states that splenectomy for trauma "in the ex-

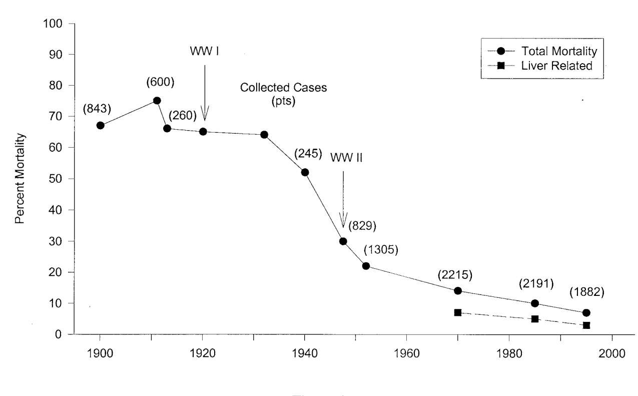

Figure 3. The mortality for liver injury from selected reports in the literature over the past century demonstrates the progress in improved mortality. Total mortality is reported in the 6% to 7% range. Mortality related to the liver injury itself has been reported from the 1970s to the present and now is often less than 5% in many experiences. WW, World War.

Table 5. Mortality Rates for Splenectomy

Year	Author	Patients, n	Mortality, %
1880	Russell	28	100
1900	Bessel-Hagen	37	47
1907	Berger (collected series)	135	38.7
1908	Johnston	108	40
1908	Lotch	138	37
1909	Brositter (collected series)	203	35
1919	Willis	—	28.8
1926	Beer	90	31
1930	Dretzka	27	33
1943	Roettig	11	9
1946	Pugh	15	6

periences of the American Expeditionary Force in the war of 1917 to 1919, was associated with a mortality rate of practically 100%." This manual, written as a guide on trauma for physicians in the military, was apparently intended to discourage splenectomy. Nonetheless, as with liver injury, the experiences of World War II brought dramatic improvement to the treatment of splenic injury. Pugh[129] reported that various experiences from that war had lowered the mortality rate to between 10% and 20% and he personally reported a death rate of only 6%. The one death in his series was from a head injury.

Eras in the management of splenic injury

Although the management of liver injury was divided into several eras, the discussion on splenic injury will be divided into two phases: the period in which splenectomy was the treatment for virtually all spleen injuries; and the era of splenic preservation. This latter treatment phase has had several iterations.

The era of splenectomy

After World War II, numerous series of splenic injuries were published.[130-139] Techniques for performing splenectomy for injury were relatively uniform, and results were fairly comparable. Although mortality rates in these reports were often high, deaths were generally related to associated injury, and a patient's demise from an isolated splenic injury was relatively uncommon. Although complication rates for general issues such as atelectasis were high, specific technical misadventures such as gastric fistulas or pancreatic injuries occurred infrequently.

Surgeons in these eras were most intent on avoidance of major morbidity or death from hemorrhage. In addition to immediate treatment for bleeding, there was concern about the potential for delayed bleeding. Numerous publications on delayed splenic rupture were reported.[134,140-144] In 1943, Zabinski and Harkins[145] published a paper on this subject, and reports continued through each succeeding decade. In 1956, Bollinger and Fowler[134] collected 258 cases of splenic trauma from previous reviews and noted a 21.5% incidence of delayed rupture or bleeding. At least a dozen additional articles reporting several cases of delayed rupture were published before the era of nonoperative management, many reporting patients who died. Interestingly, delayed bleeding from splenic injuries continues to be a problem reported in recent literature reviews. In 1990, Farhat and colleagues[143] reported delayed splenic rupture in 75 patients with splenic injury. One of these patients died. These authors reviewed more than 30 reports outlining cases of delayed splenic rupture. In 1994, Kluger and coworkers[144] presented 3 patients with delayed rupture, all of whom were initially admitted in a stable condition. These authors collected 24 cases from the recent literature (1985 to 1992) as well. What seems clear in a literature review that spans from the 1930s to the end of the century is an incidence rate of at least 1% to 2% of patients who developed major delayed hemorrhage.

Surgeons in the splenectomy era achieved remarkable results in the treatment of potentially fatal bleeding with extremely low mortality rates because of isolated splenic injury.

Shift from emphasis on hemorrhage to postsplenectomy infection

Recognition of the spleen's role in the resistance to infection was known for most of the 20th century. In 1919, Morris and Bullock[146] found an increased death rate in splenectomized rats injected with a strain of bacteria causing rat plague. Several other studies in experimental animals indicated the importance of the spleen in resisting various infections. The classic study that raised clinical awareness of this problem was published in 1952 by King and Shumacker.[147]

Although this reference is widely quoted, its details are almost never mentioned. The authors reported five cases of congenital hemolytic anemia treated by splenectomy. Remarkably, two pairs of the five patients were siblings who tragically died. These five infants had splenectomy at 4 weeks, 3 weeks, 2 weeks, 6 months, and 25 months of age, respectively. Singer[148] reviewed 2,795 asplenic patients collected from 23 series in addition to 6 patients from the Texas Children's Hospital. These 6 patients had splenectomy at 4, 5, 10, and 17 months, and 2 and 3 years of age. The 2-year-old was a trauma patient who died 3 years later of a *Haemophilus influenza* infection. Singer found 688 trauma patients, including 388 children, with 4 deaths from sepsis. The death rate in children was 0.58% and total death rate was 0.01%. Eraklis and Filler[149] reviewed 1,413 collected patients and, not surprisingly, found similar results because there were considerable overlays in reports studied. But these reports appeared to include only studies with reported infection and ignored those without reported sepsis. It is not clear whether infants undergoing splenectomy for hematologic disorders behave similarly to older children with splenectomy for trauma and, even more uncertain, whether these results on infection could be generalized to patients older than 55 years with injuries needing splenectomies. The issue of postsplenectomy infection appears settled in children,[147-154] but the data in adults are less clear.

In 1969, Whitaker[155] described an adult who developed infection postsplenectomy, although his spleen was not removed for an injury. O'Neal and McDonald[156] noted 7 cases of fatal sepsis in 256 asplenic patients and calculated a mortality rate of 7.3 per 1,000 person-years of followup.

Several reports have now documented the hazard of postsplenectomy infection in adults.[157-162] One problem with reports on postsplenectomy sepsis is that they often review the same patients, which leads to the conclusion that the problem occurs more frequently than actually may be the case. Forty-five articles in the trauma literature devoted to this subject were reviewed, and there appeared to be nearly as many articles as cases of sepsis in asplenic adult trauma patients.

DiCataldo and colleagues[163] from Italy reviewed the world's literature to 1987 and found 12 deaths from overwhelming postsplenectomy sepsis (a rate of 0.66%). None of their personal 148 patients developed sepsis problems over a several-year followup. Luna and Dellinger[151] reviewed most of the same patients, and, not surprisingly, found 11 deaths. Pachter and associates[164] mentioned three cases in a discussion of another paper with two deaths. Table 6 reviews the incidence of infection problems in trauma patients for whom years of followup are available.

The best reports are from countries where better followup appears to be available than in the United States. In 1991, Cullingford and associates[152] reported followup on 1,490 patients undergoing splenectomy in Western Australia, of which 628 were for trauma. In 3,922 person-years of exposure, 8 infections developed. Only one had overwhelming postsplenectomy infection, and only one death occurred. In 2001, a British group[162] reported a questionnaire study of microbiologists who had data on overwhelming postsplenectomy infection. They found that 24 cases had occurred in trauma patients undergoing splenectomy. The mortality rate was 46%. This is similar to the 50% mortality rate collected by Zarrabi and Rosner[165] in the 34 adult trauma patients reported in the world's literature. Because these reports all contain tremendous overlap, it is difficult to ascertain how many cases have been reported; it seems that with the addition of patients from Australia and England there are less than 70 cases worldwide, with a death rate of about 30%.

Table 6. Postoperative Infection Rates after Splenectomy for Trauma*

Author	Sepsis	Mortality	Overwhelming postsplenectomy infection
Schwartz	3.3	0	0
Malangoni	0.25	0	0
Sekikawa	0.57	0	0
Green	1.91	0.14	0.14
Cullingford	0.21	0.03	0.04
Total	0.59	0.03	0.04

*Incidence per 100 years of patient exposure.

Our unit obtained longterm followup data on 414 splenectomy patients in the late 1980s (unpublished data), reflecting 2,167 patient-years. The major problems our patients faced in order of prevalence were sequelae of the injury in which the splenic trauma occurred, alcoholism and drug dependence, and trauma recidivism or other injuries occurring later. One patient developed pneumonia that may have been from pneumococcus 6 years postinjury and was successfully treated. An alcoholic patient suffered aspiration and polymicrobial lung abscess. One patient with an intraventricular shunt developed meninigitis believed to be related to problems with the shunt itself.

Despite this low rate of postsplenectomy infection in adults, it was one of several factors that were used as the rationale for a shift from splenectomy as the primary treatment of splenic injury to splenic conservation. Additionally, the mortality rate appears higher in patients undergoing splenectomy than in a normal population.[166,167] As with liver injuries, DPL led to celiotomy in many patients with minimal injury where the spleen was not severely injured. But the major driving force behind splenic preservation was the observation by surgeons caring for injured children that the spleen could be saved by operative means such as splenorrhaphy or through avoidance of operation altogether.[168-171]

The era of splenic preservation

Efforts at splenic preservation could be divided into three different areas: operative attempts at maintaining splenic function; embolization of the splenic artery and its branches; and nonoperative management of splenic injury.

Operative attempts at splenic salvage

With the increased awareness of the immunologic importance of the spleen, efforts at operative splenic salvage began to appear in the 1980s, led by pediatric surgeons. Splenic autotransplantation was advocated by several groups, but, eventually, placement of these implants was shown to be ineffective.[171-175] Splenorrhaphy was described in children and, within a few years, several series of splenic salvage by suturing the spleen had been reported. Overall success rates in children were reported to be high. With the exception of hilar injuries, most splenic lacerations were amenable to repair. Initially, it was believed that properties of the child's spleen made this possible, but that suturing an adult's spleen was not feasible. But within a few years, splenorrhaphy was being practiced in adults with a reasonable success rate. On an interesting historic note, William Mayo[176] described a patient managed by splenorrhaphy in 1906. Feliciano and colleagues[177] and Pachter and associates[164] reported extensive experiences, with rebleeding rates of 1.5% and 1.8%, respectively. Most articles on splenorrhaphy were positive, but Beal and Spisso[178] mentioned rebleeding as a problem and noted the higher risks of blood transfusions with splenorrhaphy.

Some splenic injuries were devitalizing or shattered the lower pole such that splenorrhaphy was not feasible. Partial resection of the spleen with oversewing of the capsule was practiced with reasonable success in experienced hands. An upper pole artery is present in the majority of spleens, which facilitates lower pole resection.[179] Multiple ingenious methods were devised to tamponade the spleen, including wrapping the injured spleen.[180-183] It is unclear what the penetrance of operative splenic salvage was into the care of trauma patients not treated in trauma centers. Several studies show considerable variations in the rate of attempts at splenic salvage between trauma centers and nontrauma centers.[95,184] Nonetheless, operative splenic salvage was a concept that had a high rate of success in many centers. When nonoperative management came to the fore, splenorrhaphy and other forms of operative splenic salvage began to decline, although they are still useful when operation is required. But most nonoperative failures are treated by splenectomy.

Embolization of splenic artery hemorrhage

In 1973, embolization of the splenic artery was described to decrease the splenic mass in a patient with hypersplenism. This procedure did not gain widespread popularity because of reports of splenic rupture and abscess.[185] In 1984, the transplant group[186] at the University of Illinois reported a prospective randomized trial of splenectomy versus partial splenic embolism to decrease the functional splenic mass. Several additional publications on the use of splenic artery embolization to decrease splenic function and size were subsequently reported. In 1995, Scalfani and coauthors[187] introduced the concept of embolization of splenic artery injuries, and 150 patients with all grades of splenic injuries underwent diagnostic arteriography on admission. Ninety patients had negative angiograms and were observed only; 60 had embolization of splenic vascular lesions. The total salvage rate was reported to be 98.5%, which is the highest success rate reported in the literature.

The technique of embolization has also been a matter of debate: ie, main artery coil, which may render the entire spleen ischemic and obviate the value of "preserving" the spleen versus distal embolization for active bleeding areas. A problem with this latter approach is the potential of rebleeding, because the vessels may be in spasm at the time of the initial angiogram.

A study from Memphis demonstrated that vascular lesions identified by a repeat CT after resuscitation were not present on an admission CT scan. Concern also existed about embolization of multiple arteries that could affect most of the functioning spleen.

Several studies on splenic embolization for trauma are now present in the literature, including two published within the past year.[188-192] Haan and associates[188] presented data from four institutions that performed splenic embolization from 1997 to 2002. Indications for embolization were based on CT findings and included significant hemoperitoneum (outside the perisplenic area), contrast extravasation, splenic artery pseudoaneurysm, and arteriovenous fistula, with failure rates of 10%, 17%, 12%, and 40%, respectively, depending on the injury treated. The overall mortality rate was 5%, although none appeared related to the injured spleen. The failure rate was 13.5% and complications were numerous (20%), including hemorrhage (13%), missed injuries (3%), and infection (4%). Fourteen patients underwent repeat angiography, and six developed splenic abscesses.

Thirteen of 168 patients underwent splenic embolization in a series from San Antonio.[193] Twelve were deemed a success. No mention was made of complications or outcomes other than splenic salvage. These studies augment data from Memphis, where 26 patients out of a population of 526 with splenic injury had successful embolization. These authors reported a high rate of success, and no mention was made of complications. The total mortality rate for all splenic injured patients was 10.5%, but included no deaths in those treated by angiography.

Although splenic embolization has been espoused to preserve the spleen, no studies document its effect on the organ's immunologic function. Because the perfusion of the sinusoids of the spleen are driven by arterial pressure, it is unclear if thrombosis of the artery will alter normal splenic function.

Nonoperative management of splenic injury

Nonoperative management (NOM) was initially practiced in children with splenic injury with excellent results. Virtually all studies in children have been positive, and NOM clearly is the treatment of choice in this population. As in hepatic injuries, the progress in children paved the way for trials in adults. The criteria for NOM may vary somewhat among institutions but generally include hemodynamic stability and lack of evidence of visceral injuries.[194,195] Initially, it was believed that patients older than 55 years should be treated operatively,[196] but some subsequent studies refute that assertion.[197,198] Absence of head injury was initially considered a contraindication of NOM, but that criterion is not used in many centers. Some centers are more likely to operate on higher-grade injuries or those with a vascular blush.

The success rate of NOM appears in adults to vary widely from 60% to 98%.[199-217] The multiinstitutional trials committee of the Eastern Association for the Surgery of Trauma (EAST) examined the results of treatment of 1,488 adults contributed by 27 trauma centers.[200] Grade of splenic injury, degree of hemoperitoneum, and presence of associated injury were important determinants of outcomes of NOM. A failure rate of 11% occurred in those managed nonoperatively. A second study by that group showed that patients older than age 55 had a higher rate of failure of NOM than those younger. The mortality rate was increased for those with successful NOM (8% versus 4%) in patients older than 55 years versus those younger than 55 years, which is not surprising. The death rate in NOM failures was 29% versus 12% in those over age 55 and in the less than age 55 groups, respectively.[218] Another recent study has also confirmed an increased mortality rate in older patients in whom NOM is not successful.

Several predictors of failure of NOM have been examined, although there are no universally accepted recommendations for management of patients who might be at higher risk for failure. In 1995, the Memphis group[219] noted a "contrast blush" in two-thirds of the patients with failed operative management. Several subsequent reports demonstrated vascular lesions within the spleen in patients with failed NOM.[220-222] These studies emphasize the importance of the CT scan in detection of these abnormalities. Several issues with CT scan reliability have been raised: Do early CT scans accurately depict vascular lesions or should a second scan be done after resuscitation? Issues of interrater reliability for detecting these lesions have been raised as well.[223] It does seem likely these abnormalities should heighten awareness of potential failure of NOM.

Table 7 shows the results of NOM in several studies. Success rates vary from 52% to 98% even though the criteria for inclusion were relatively uniform. With passage of time, surgeons appear to be accepting this treatment strategy with greater frequency for patients with a higher degree of hemoperitoneum and a higher grade of splenic injuries. Some now take patients in unstable condition to the angiogram suite instead of the operating room. It is interesting that most articles published on splenic injuries focused on the ability to successfully avoid operation as the optimal end point. Several recent publications made no mention of the mortality or morbidity incurred with operative or nonoperative treatment, or the fate of the spleen after NOM failure.

Table 7. Outcomes of Nonoperative Management of Splenic Injuries in Selected Series

Author	Year	No. of patients	% Immediate operation	% Successful nonoperative management
Cogbill*	1989	832	87	88
Shackford†	1990	1,254	—	69
Pachter	1990	193	87	96
Schurr	1995	309	58	87
Smith	1996	173	58	97
Gooley	1996	46	—	52
Davis	1998	524	39	94
Pachter	1998	190	47	98
Konstantakos	1999	267	33	83
Cocanour	1999	461	42	86
Myers	2000	233	31	94
Nix	2001	542	25	92
EAST Group‡	2001	1,488	39	89
Dent	2004	168	17	98

*Experience of six centers.
†Collected series from literature.
‡Experience of 27 centers.

The time of failure of NOM was reviewed in several studies where such data were available. The EAST study confirmed that nonoperative management that failed usually did so within 96 hours, but it failed in 7 patients after day 9.[200] Cogbill and colleagues[195] noted failure of NOM at days 6, 9, 10, 13, 19, and 36. A report from Cleveland showed that NOM failed in 31 adults at an average of 71 hours, but 1 patient suffered failure at 20 days.[180] A review of 14 series, in which data on time of failure were present, disclosed at least 30 patients suffered NOM failure after Day 7. One was smoking a cigarette outside the hospital on Day 8 when major bleeding occurred![211]

There may be a natural reluctance to report untoward events associated with failure of nonoperative management. Our unit has had at least 4 deaths from delayed bleeding in NOM patients over a 10-year period (unpublished data). Two older patients died in the ICU on Day 6 and Day 10; 1 died on Day 9 after transfer to the orthopaedic service, and 1 died on Day 16 at Walmart. We had another patient with an anoxic brain injury after an arrest from splenic bleeding. None of these had a drifting decline in hemoglobin but, rather, catastrophic bleeding reminiscent of reports on delayed rupture from an earlier era.

Mortality and morbidity of splenic trauma

Unlike liver trauma, in which the mortality rate has declined considerably in the past 20 years, total mortality rates remain at 6% to 7% or higher in many series because of the presence of associated injuries. On the other hand, the mortality rate of isolated splenic injuries has effectively been at 0% for nearly 40 years (Fig. 4).[224-229] It is difficult to find any report where more than an occasional patient died of treatment of the splenic injury itself. This creates even greater pressure on surgeons to ensure that patients not die in their attempt to finesse a salvageable splenic injury by NOM, embolization, or any treatment other than splenectomy.

Although numerous reports of deaths associated with NOM in adults assert that the deaths are not related to the splenic injury itself, several list multiple system organ

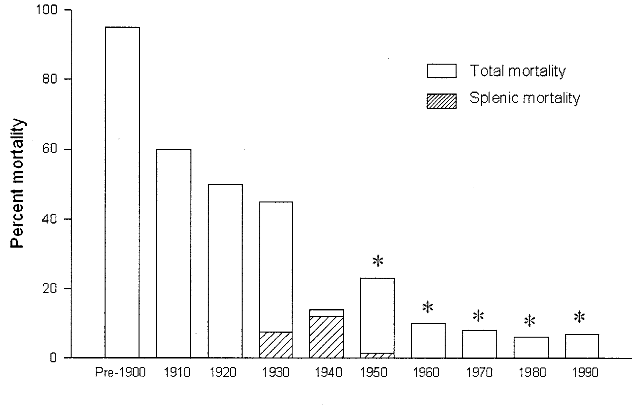

Figure 4. Total deaths in patients with splenic injury is usually about 6% to 7%. *Mortality from the splenic injury itself has been less than 1% for more than 50 years.

failure as a frequent cause of death. In such patients, the additive effect of hemorrhage from the spleen is difficult to assess. A few patients on our unit have developed multiple system organ failure without obvious cause. It remains unclear whether or not the occult blood loss from the spleen could have been a cofactor in their deaths.

Patients able to be successfully treated by NOM invariably have decreased intensive care unit (ICU) stay, decreased total length of hospital stay, and decreased blood usage when compared with patients who have operations. On the other hand, the operative group usually had a higher injury severity score and higher grade of splenic injury.

DISCUSSION

Progress in outcomes of hepatic injuries in the last 20 years has been dramatic. Major improvements in mortality rates appear to be related to declining death rates from hemorrhage. The strategies of perihepatic packing, better management of major juxtahepatic venous injuries, use of angiographic embolization for hepatic artery bleeding, and perhaps even nonoperative management itself appear to have improved the mortality rate. Surgeons caring for liver injuries must be prepared to use a variety of operative and interventional maneuvers to treat those hepatic injuries that require more than nonoperative management. The decision not to operate may be fraught with uncertainly not only for issues involving the liver but because of concerns over a missed visceral injury. Surgeons must not, in my opinion, adopt a mindset in which an operation or other interventional procedure is viewed as a defeat.

Surgeons in World War II learned that drainage of bile is very important, which is a lesson that is often forgotten by those providing nonoperative treatment of liver injuries today. Patients who have major liver injuries with a great deal of bile and old blood in the abdomen need to have them removed. If there is a major fluid collection in the right gutter and pelvis, as is often the case, percutaneous drainage will not remove this fluid. Our unit has advocated the use of laparoscopy a few days postinjury to accomplish these goals. The results have been excellent, and we certainly recommend this procedure to surgeons as a useful adjunct to nonoperative management.

The treatment of splenic injuries presents a different set of issues. Unlike liver injuries, the mortality rate from splenic injuries has not changed for 40 years. By the 1960s, deaths from isolated spleen injuries approached zero in large collected series, and a review of numerous series demonstrated a mortality rate of less than 1% for the past several decades. The total mortality rate varied among series, depending on the severity of associated injuries, but was 13.8% in the EAST study that reflected the experience of 27 trauma centers. Because relatively few patients die of isolated splenic injury after reaching the hospital alive, we must by careful that our management does not imperil the patient.

I admit a certain dismay over the current management of splenic injuries in some centers. I believe that the balance between concerns with bleeding and infection has shifted illogically to favor infection. Splenic preservation has been granted a position of "political correctness" that must be balanced against the fact that occasionally a shattered spleen must be removed. In fact, many patients are still receiving splenectomy, but the emphasis on NOM as a laudable end point in and of itself is worrisome to me. When I hear our residents apologize for removing a bleeding spleen and saving a life, I become concerned. We must not appear to be cavalier about patients with high-grade splenic injuries or a large hemoperitoneum. The manner in which NOM success rates are reported is also deceiving. Numerous reports of 95% success made little mention of the 17% to 45% of patients who must have urgent splenectomy. Several series also immediately discount the deaths from their computation.

I am also puzzled about what is termed "failure of nonoperative management." When NOM fails at Day 14 or Day 29, as has frequently been reported, what happens to the patient? The literature implies that patients are in a situation in which they can promptly receive a splenectomy. Surely, some of these patients, who are many days from injury, must be placed at risk from these "failures." It is incumbent on those who report on these experiences to provide more data on the potential risk of offering treatment other than operation.

Much more information is needed on postsplenectomy sepsis in adults. Large studies from multiple trauma centers need to be organized, using the multiinstitutional study committees of American Association for the Surgery of Trauma, EAST, or the Western Trauma Association. Those of us who care for adult trauma patients with splenic rupture should cease referring to the studies by King and Shumacker and Singer as rationale for avoidance of splenectomy. We should organize studies to determine the incidence of infections in posttraumatic asplenia. Equally importantly, data are needed on delayed bleeding. There appear to have been more "failures" of NOM occurring after Day 7 reported in the past decade than total cases of overwhelming postsplenectomy infection ever reported in the world's literature. We need reassurance that these "failures" are not being harmed. Anecdotally, I have been impressed in private discussions about deaths or "near misses" from bleeding occurring in NOM failures. These are rarely reported in the literature. Additionally, many reports list multiple organ failure as a leading cause of death. Does unrecognized shock play a role in these deaths?

The concerns I have about splenic preservation apply even more strongly to embolization of the spleen. Several algorithms recommend taking patients in unstable condition to angiography suites rather than to the operating room. A recent positive report on splenic embolization showed a 13.5% failure rate and a 20% complication rate, including 6 patients with splenic abscess. Fourteen patients required repeat angiography to achieve these results. Quite frankly, if a surgical series reported these results for splenic operation, the surgeon would undoubtedly lose his operative privileges! We now have reports of unstable patients being taken to angiography instead of to the operating suite, and then, when they develop a splenic abscess, there is an attempt at percutaneous drainage of that process. The logic involved in some of these decisions might make sense if it was being advocated by groups other than surgeons.

My real concern is not what highly skilled trauma surgeons and angiographers do in Level I trauma centers to try to push the envelope of splenic preservation further and further. My apprehension is the mindset that it creates in those who care for ruptured spleens in venues other than Level I trauma centers and the uncertain message it sends to residents. Those of us who train residents in the care of trauma patients must ensure those residents can safely remove a spleen after they are in practice. Arresting splenic hemorrhage can be a lifesaving procedure, and surgeons must not abrogate that responsibility to interventionalists who, almost certainly, will not be available in all the hospitals in which a splenectomy is urgently needed. It should also be noted that the efficacy of this procedure in preserving immunologic function remains unproved.

Having challenged our trauma community to acquire vitally needed data on several issues, it is time to pay tribute to those caring for the injured and the remarkable strides that have been made in treating liver and spleen injuries. Not only has the care for patients greatly improved, but our organizations have served us well. The learned societies in trauma have organized studies to elucidate solutions to problem areas and have promulgated grading systems for liver and spleen injuries that are now routinely applied. Most importantly, our American College of Surgeons has provided leadership in the care of the injured. Through the auspices of the Committee on Trauma, we have organized trauma care, verified centers for provisions of care to the most critically injured, and provided a wealth of educational opportunities since the founding of the College, for all surgeons who care for the injured. The improvements made in treatment of the solid intraperitoneal organs should stand as one of our best achievements.

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