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Long term outcomes of nonoperative treatment of blunt abdominal trauma

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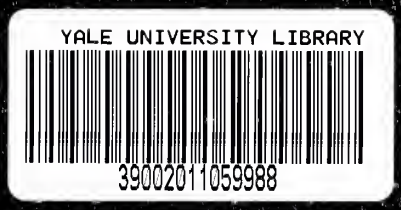

Long Term Outcomes of Nonoperative Treatment of Blunt Abdominal Trauma

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by
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LONG TERM OUTCOMES OF NONOPERATIVE TREATMENT OF BLUNT ABDOMINAL TRAUMA. Matthew A. Gutierrez and Peter B. Angood. Department of Surgery, Washington University Medical Center, Washington University, St. Louis, MO. (Sponsored by Manish Tandon, Department of Surgery, Yale University School of Medicine).

The purpose of this study was to determine if nonoperative treatment of blunt liver and splenic injuries has any long-term consequences that may be detrimental to patients. A retrospective review was conducted of 112 adult trauma patients that were treated nonoperatively for blunt abdominal trauma between 1991 and 1998. The patient records were followed up to see if this patient population had an increased occurrence of medical problems that could be related to nonoperative management. Approximately one patient developed post transfusion hepatitis, two patients developed delayed hemorrhage of their injuries but continued to be managed nonoperatively, one patient failed nonoperative treatment and required splenectomy, three patients developed sepsis, and one patient died during nonoperative management from an asthma exacerbation. There was no incidence of small bowel obstruction in the series. It appears from this data that long-term complications related to nonoperative management are minimal, and it should continue to be the treatment of choice in hemodynamically stable patients who sustain blunt abdominal trauma.

Acknowledgements

This thesis is dedicated to the memory of my grandfather David Gutierrez who passed away while I was interviewing for medical school, to my grandmother Frances Fischer who passed away during my second year of medical school, and to my grandfather Eric Fischer who passed away long before his time. May their memories live on and may they rest in peace.

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Introduction

For centuries, surgeons have struggled with the management of traumatic liver and splenic injuries. Both are solid organs that are commonly injured in blunt abdominal trauma, with the liver and spleen being the first and second most commonly injured organs, respectively (1). Anatomically, the liver receives blood flow from the hepatic arteries as well as the portal vein, and is a very vascular structure that functions in detoxification and absorption of materials in the blood. As a result, about 29% of resting cardiac output flows through the liver at any time. In addition, it can be a reservoir for up to 450 cc of blood in a healthy person or up to 1 liter of blood in a person with congestive heart failure (2).

The spleen is also a very vascular organ, with a dual arterial blood supply. Because the spleen functions as an immunologic organ, it receives approximately 5% of the resting cardiac output (3), and can be a reservoir for up to 100 cc of blood (2). Because both the liver and spleen are so highly vascularized, injuries to either organ can lead to severe hemorrhage and shock. Surgery on these organs is very difficult since there can often be a large amount of blood loss associated with operations, increasing the morbidity and mortality rate.

<u>The Spleen</u>

In 1866, Evans suggested that if injured, the spleen might bleed catastrophically at a time remote from that of the initial injury. He believed that the thin splenic capsule would not be able to withstand continuous arterial pressure if injured (4). In 1881, the first evidence that the spleen was capable of healing itself

was shown by Billroth in an autopsy based study (5). However, in other pre 20th century literature, techniques for both splenic preservation and excision were described (6). In 1892, the first successful splenectomy after blunt injury was reported by Riegner (7), and in 1911, Kocher's Textbook of Operative Surgery stated that, "injuries of the spleen demand excision of the gland, no evil effects follow its removal while the danger of hemorrhage is effectively stopped." (7). By the beginning of the 20th century, splenectomy was well established as the treatment of choice for trauma to the spleen.

In a 1912 report by Bland-Sutton, the splenectomy dictum was reinforced after nonoperative treatment was shown to have a high mortality rate (8). In 1932, McIndoe reported that delayed hemorrhage from splenic injury frequently occurs and has a comparable mortality rate to that seen with primary splenic rupture (9), once again reinforcing splenectomy as the treatment of choice for splenic trauma.

During the early 20th century, new information about the function of the spleen began to emerge. In 1918, Pearce reported that 25% of animals die from peritonitis or pneumonia post splenectomy, although at the time these infections were not thought to be due to the removal of the spleen (10). However, in 1919, Morris and Bullock reported asplenia as a cause of increased infection in animals, concluding that removal of the spleen may increase the risk of infection in humans as well (11). It wasn't until 1929 that O'Donnell reported the first case of post-splenectomy infection in a human (12), and in 1952 King and Shumacker reported 5 cases of severe infection in infants undergoing splenectomy (13). In 1973, Singer described 119 cases of sepsis in a review of 2795 patients from all age groups who

underwent splenectomy for various reasons (14). Of these cases, he reported 25 incidents of fulminant sepsis in 688 trauma patients aged 1 to 70 who underwent splenectomy for traumatic rupture, although "most" patients who became septic were less than 15 years of age (14). However, in 1986, Green et al reported major septic complications in 5.9% of their adult post splenectomy trauma population, and recommended attempts at splenic salvage when possible because of the increased risk of late septic complications (15).

It was these reports and the surgeons' awareness of the increased risk of infection post-splenectomy that first prompted the trend towards splenic preservation (16-20) and nonoperative management of splenic injuries in the pediatric population. Nonoperative management was first described in 1968 by Upadhyaya and Simpson (21), and as the success of this method in pediatrics became established, surgeons began to try conservative management on adults. At first the patient population was limited to isolated splenic injuries of very low grade and minimal hemoperitoneum (22-27), but as the practice became more widespread, nonoperative management began to be applied to more severe splenic injuries and in multiply injured patients (28-34). Although age over 55 years was originally thought to be a contraindication to nonoperative management because of decreased success rates (7, 35), most practitioners currently agree that nonoperative management can be applied to any hemodynamically stable patient (36-39). Nonoperative management should now be attempted before surgical intervention as long as the patient is clinically stable.

The Liver

The major impetus for attempting nonoperative treatment of liver injuries was uncontrollable hemorrhage during surgery. Although many physicians believed that surgical hemostasis was necessary for controlling bleeding due to liver injury, pediatric surgeons were the first to show that nonoperative management of liver injuries was a viable alternative. As early as 1908, Pringle showed minor liver injuries could heal without operation (40), but noted that "While small lacerations of the liver substance may be, and no doubt are, recovered from without surgical interference: if the laceration be extensive and vessels of any magnitude are torn, hemorrhage will, owing to the structural arrangement of the liver, go on continuously" (40). Because of this, operative management remained the treatment of choice for hepatic injuries for almost eight decades. However, in 1977, Stone et al showed that 70% of blunt liver injuries stop bleeding spontaneously by the time the patient is taken to the operating room (41), and in 1982, Carmona et al reported that 72%-85% of all traumatic liver injuries are amenable to simple techniques such as packing, draining, or suturing (42). Since only a minority needed intensive surgical procedures to control bleeding, it followed that a large number of patients with traumatic liver injuries could probably be managed nonoperatively.

In 1985, Cywes et al published a paper describing successful nonoperative management of pediatric patients with blunt liver trauma (43). In 1988, Farnell et al suggested that adult patients with blunt liver injuries might also be candidates for nonoperative treatment (44). As has happened with nonoperative management of splenic trauma, nonoperative management of hepatic injuries has also become

widespread. As with splenic injuries, the main criterion for attempting nonoperative management is hemodynamic stability (1, 45-48).

Nonoperative management

Since it was first attempted in pediatric patients, the use of non-operative measures to manage abdominal solid organ injuries has been steadily increasing in this country (49). This trend has come about due to several factors, including the desire to cut down on expensive operations unless absolutely necessary, the increased use of CT-scans and decreased use of peritoneal lavage to assess the severity of abdominal solid organ injury, and the improved short-term outcomes over surgery in patients treated nonoperatively (50). Especially with the routine use of CT scans to noninvasively assess internal injuries in hemodynamically stable patients (51-55), physicians have felt more comfortable with initiating nonoperative management.

While there has been one study that specifically looked at delayed complications of nonoperative treatment of splenic injury (56), one study that looked at long-term outcomes of blunt trauma relating to functional status (57), and several case reports of delayed complications of nonoperative management for both liver and splenic injuries (58-62), there has not been any study that specifically looks at long-term medical outcomes that may have occurred secondary to nonoperative management.

In a study by Cocanour et al in 1998, approximately 7 of their 87 patients (8%) treated nonoperatively developed delayed complications directly attributable to

their splenic injury that required intervention. However, the latest of these complications occurred 1 month after injury (two patients developed splenic abscesses), while the remainder of these complications involved bleeding that occurred within the first 3-8 days (56). Patients in this study were identified as having delayed complications by reviewing the proceedings of the trauma morbidity and mortality conference, so only complications that occurred in house after 48 hours were examined. There was no attempt to review follow-up treatment records or later readmissions to find out how patients did post-discharge. It is unknown if any of these patients had complications that occurred or medical problems that developed in the future since they weren't followed after discharge in this study. Because of these limitations, no conclusions about long-term outcomes after nonoperative treatment can be reached.

In another study looking at the long-term functional status after blunt trauma, almost half of their multiple system blunt trauma patients remained unemployed 1 year after discharge. Those that returned to work had higher self reported scores for physical and mental health, but also had a statistically significant lower mean Injury Severity Score. Unfortunately, this study did not look at what specific medical conditions the unemployed patients complained of in follow-up, and only relied on self reported assessments of how the patients felt they were doing. This study was also not limited to patients with blunt trauma to the abdomen or nonoperatively treated patients. Also the patients in the study were only followed up one year after discharge as the definition for long-term (57). Because of the limitations of this

study as well, no assessment of long-term complications of nonoperative management of blunt abdominal trauma can be made.

Of the case reports in the literature, the most common complications described are delayed rupture of the spleen (61), delayed rupture of a subcapsular hematoma (59,62), delayed rupture of a pseudoaneurysm (58), hepatic abscess formation with sepsis (62), hemobilia (58,60), and biliary stricture formation (63). Occasionally these complications have led to hemorrhage and death (59, 62). Although this has been reported, these complications are now often recognized and interventions made before there is a poor outcome (1). Diagnostic delay of associated small bowel injury has also been described (53-54, 64-69), but according to Bensard et al, this delay in children treated nonoperatively for blunt abdominal trauma does not alter the hospital course (70). Although the study involved patients 18 and under, it brings into question whether or not diagnostic delay would be important for adults as well.

The initial studies looking at the efficacy of nonoperative treatment of blunt abdominal trauma do report that patients followed up to 1 year after management do well without complications (22-24, 29, 33, 43, 48, 60, 71-75). However, besides one study by Farnell et al that reports of 17 patients followed an average of 27 months (range 3 months to 6 ½ years) who did not have complications (44), there have not been any studies documenting how patients do greater than 2 years after nonoperative management. Because of this it is difficult to comment on any possible long-term ill effects that nonoperatve management may cause. Although nonoperative management has become more widespread, as late as 1998, the Eastern

Association for the Surgery of Trauma published guidelines stating that, "There is insufficient data to suggest non-operative management (NOM) as a Level I recommendation for the initial management of blunt injuries to the liver and/or spleen in the hemodynamically stable patient", because of a lack of prospective randomized studies that would give appropriate outcome data (76). Although an appropriate prospective study still needs to be done, there is no published retrospective data examining long-term outcomes to see if there are any ill effects far removed from the trauma that may be related to nonoperative management.

Statement of Purpose and Hypothesis

Several studies have been done comparing the short-term outcomes of nonoperative management of abdominal solid organ injuries to operative management of these injuries, and have shown an equal or higher success rate for nonoperative treatment (23, 29, 31, 34, 47, 74-75, 77). However, a long-term follow-up study has not been done to assess whether or not the nonoperatively managed patients have an increase in long-term complications. Although Knudson et al reported that hemoperitoneum after liver or splenic injuries should be resolved by day 5 after trauma (78), it is not known whether or not the presence of blood in the abdominal cavity can increase the incidence of small bowel obstruction and adhesions, or have other ill effects in the long-term. In addition, the use of non-operative treatment for abdominal trauma may cause other internal injuries to be missed (64-69), and other studies warn about an increased amount of blood products being used secondary to failure of nonoperative management (73, 79), which could lead to higher rates of blood-borne illnesses.

This study aims to determine whether there may be any long-term consequences of treating abdominal solid organ injuries non-operatively, by looking at how patients are doing during follow-up visits, and by looking at what medical problems they present with many years after nonoperative treatment. Our belief is that there should be only minimal long-term complications for patients treated nonoperatively compared to historical data of those treated operatively for abdominal solid organ injuries, and therefore nonoperative management is a safe and effective treatment for hemodynamically stable patients who suffer blunt abdominal trauma.

Methods

A retrospective review from January 1991 to January 1998 of patients undergoing nonoperative management of blunt abdominal trauma to the liver and/or spleen was conducted at this level I trauma center. Four hundred and fifty five patients were selected from the Yale Trauma Registry by Dr. H. David Stein on the basis of the discharge diagnosis of liver and/or splenic injury from ICD-9 codes. From this, charts were obtained and reviewed by this author for approximately one hundred and twelve patients that were selected by limiting patients to over 16 years of age and excluding patients if they underwent abdominal surgery for any reason within the first 72 hours after admission.

A data collection form was designed and information such as age, gender, Injury Severity Score, presence of hemoperitoneum, number of units of blood products transfused, length of hospital stay, and hemodynamic stability was gathered for each patient. The hospital database (SDK) and patient records were then

consulted by this author to see if the patient had any further admissions and/or reassessments after nonoperative management of their solid organ injuries. If further admissions were discovered, then the patient record was pulled and the nature of the admission was recorded. Long-term was defined in this study as any outcome taking place greater than 72 hours after admission for nonoperative treatment, and a complication was defined as a symptom or disease directly attributable to nonoperative interventions, or not explainable by the patient's prior medical history but potentially explainable by nonoperative interventions. Failure of nonoperative management was defined in this study as any death or abdominal operation that occurred greater than 72 hours after admission but before discharge. The data was then compiled to see if there was an increased occurrence of a particular type of medical problem in this patient population, and to determine if such medical problems might have a higher occurrence than has been described in operatively managed patients.

Results

Of the 112 patients surveyed (Table 1), approximately 47 were found to be doing well in follow-up. Approximately 23 had no further re-admissions or follow-up re-evaluations on record, and were lost to follow-up. Approximately 4 patients failed nonoperative treatment, with three of these patients requiring abdominal surgery during their admission. One patient died during nonoperative management secondary to an asthma exacerbation. The remaining 38 patients were re-evaluated for diverse medical problems after discharge, including those both related and

unrelated to nonoperative treatment of their abdominal injuries. Of the 85 nonoperatively managed patients that were not lost to follow-up, the mean length of follow-up was 632.8 days, with a standard deviation of 858.6 days (range 1 day – 9 years).

The average age of the patients surveyed was 39.0 (SD +/- 18.9, range 16 to 93 years old), with a mean length of stay of 7.5 days (SD +/- 9.3 days, range 1 to 64 days). In the patient population surveyed, there were 84 males and 28 females. Approximately 54 of the nonoperatively treated patients surveyed had hemoperitoneum identified on CT scan at admission, with 17 patients requiring transfusion of blood products. The mean number of units of blood products transfused was 3.4 units (SD +/- 3.7 units, range 1 to 17 units) in the nonoperatively managed patients that received blood products. If all nonoperatively managed patients are included, then the mean number of units transfused was 0.54 units (SD +/- 1.9 units), with approximately 15.7% of the nonoperatively treated patients receiving one or more units (Table 1). The patients surveyed that did not fail nonoperative management had a mean Injury Severity Score of 11.4 (SD +/- 7.4, range 4 to 43) with liver and/or splenic injuries ranging from Grade I to Grade IV (Table 1). The mean grade for liver injuries was Grade II (mean = 1.7, SD +/- 0.83), and the mean grade for splenic injuries was also Grade II (mean = 1.7, SD +/- 0.79) (Table 1).

Of the four patients that failed nonoperative management (Table 2), the mean Injury Severity Score was 23.5 (SD +/- 14) versus 11.4 for those patients that did not fail nonoperative management ($p = 0.0024$), with the mean length of stay

significantly longer (37.3 days versus 6.4 days, $p < 0.0005$). These patients also required transfusion of significantly more blood products than those that did not fail nonoperative management (14.3 units versus 3.4 units, $p = 0.03$). However, there was no significant difference between the average severity of liver or splenic injuries between those who failed nonoperative treatment and those who didn't (mean liver grade 1.5 versus 1.7, $p = 0.74$; mean splenic grade 2.0 versus 1.7, $p = 0.47$) (Fig. 1).

When the patients were stratified according to year of nonoperative management (Table 3), there was a significant difference found between the patient populations ($p = 0.032$) as far as Injury Severity Scores, but there was no significant difference between the patient populations when comparing splenic injury grade ($p = 0.31$) or liver injury grade ($p = 0.16$) (Fig. 2). There was also no significant difference between length of stay in the patient populations ($p = 0.15$) (Fig. 3) or age of the patients treated ($p = 0.94$) (Fig. 4).

Of the patients surveyed, approximately 24 had medical conditions or complaints that were abdominal/gastrointestinal in origin (Table 4). Of these complaints, approximately 4 involved the upper GI tract (patients #485, #2683, #3000, #3009), 6 involved the lower GI tract (patients #2281, #2321, #2345, #2393, #2486, #2901), 8 involved the liver (patients #158, #539, #900, #992, #1727, #1930, #3910, #4372), 2 involved the spleen (patients #726, #928), and 4 involved various other structures in the abdominal cavity (#674, #1472, #2505, #3370). There were no patients in this series that developed small bowel obstruction. Of these complaints, only 4 are likely related to nonoperative management and will be described further.

Three patients were diagnosed with hepatitis after nonoperative treatment. Out of these patients, only one likely received hepatitis from transfusion of blood products during nonoperative management, since the other two patients (#2901, #3910) had no record of transfusions in their chart and a history of IV drug abuse. Of these patients, the one patient (#992) confirmed with post transfusion hepatitis had received approximately 13 units of PRBCs and 4 units of whole blood for a Grade II splenic and Grade I liver laceration, and was diagnosed with Hepatitis C approximately 3 months after discharge. Approximately 8 years after diagnosis, the patient remains asymptomatic with normal liver function and a low viral load according to his clinic chart. There was no history of previous transfusions or IV drug abuse in this patient's past medical history, and the hepatitis was believed by the patient's physician to be transfusion related.

One patient (#674) surveyed was treated for idiopathic retroperitoneal fibrosis approximately ½ year after nonoperative treatment. His past medical history was only significant for tobacco use and hypertension, and he was nonoperatively treated for a Grade I liver laceration with no evidence of hemoperitoneum or retroperitoneal bleed on CT scan. However, his admission CT scan did show bilateral ureteropyelocalyectasis, no excretion of contrast to the left kidney, and significant periaortic fibrous tissue, indicating that this condition was likely preexistent to his abdominal trauma.

Two other patients were found to have extension of their injuries on repeat CT scans several days after admission, and another patient (#726) was noted to have persistent heterogeneity in the spleen approximately 23 days after admission. One

patient (#928) with a Grade II splenic injury was found on a repeat CT scan 5 days after admission to have had a delayed splenic hemorrhage. The patient was hemodynamically stable throughout his admission, and did not require transfusion of any blood products. He was discharged on hospital day #6. A second patient (#1727) with a Grade II liver laceration was found to have extension of a liver hematoma on a CT scan 3 days after admission. This patient also remained hemodynamically stable and did not require transfusion of blood products. This patient was discharged on hospital day #7.

Approximately three patients in this series were noted to have developed sepsis during admission. One patient (#1111) developed overwhelming infection during nonoperative management. This patient had a Grade I splenic and Grade II liver laceration, and developed both pneumonia and a wound infection before he became septic. Two other patients (#1520 and #4027) that developed sepsis were operatively managed. One patient (#1520) had a splenectomy and became septic with Candida post op. The other patient (#4027) had a Grade II splenic injury and developed MRSA line sepsis after operations for gangrenous cholecystitis and colon carcinoma.

Approximately four patients failed nonoperative treatment, with one patient death secondary to a coexisting condition. This patient (#1030) died on hospital day #4 secondary to acute decompensation from asthma. His past medical history included COPD, a remote history of tuberculosis, history of asthma, and oxygen dependency at home. The patient had remained hemodynamically stable and did not

require transfusion of blood products, but had significant respiratory issues that contributed to his death.

The three other patients required operative intervention greater than 72 hours after admission, with a mean interval of 15.7 days (SD +/- 15.1 days, range 5-33 days) until abdominal surgery was needed. Two patients (#202 and #1520) required exploratory laparotomies secondary to hemodynamic decompensation, with one patient (#1520) eventually receiving a splenectomy and excision of gastric ulcers. The exploratory laparotomy on patient #202 on hospital day #5 did not find any source of bleeding and less than 50 cc of old blood in the abdomen, and the patient was discharged on hospital day #22. The third patient (#4027) required a sigmoid colectomy for metastatic colon carcinoma and a cholecystectomy for gangrenous cholecystitis approximately 33 days after admission, despite clinical suspicion of cholecystitis as early as 6 days after admission. This patient also developed abdominal wound dehiscence approximately 4 days after his operation and needed to have retention sutures placed. Although this patient did not truly fail nonoperative management, he is included in this group because of the abdominal operation during his admission. This allows for a more fair comparison to be made when looking at long-term complications that may be secondary to operative intervention versus nonoperative intervention.

Two other patients in this series (#539 and #1929/1930) were admitted a second time for blunt abdominal trauma with injuries to the liver. No complications developed in either patient. Details of the second visit for patient #539 is not

included in this study since his readmission fell outside of the time period from which the patients were selected.

Discussion

No previous study has been done to look at long-term outcomes of patients treated nonoperatively for blunt abdominal injuries. While it has been shown that in the short-term, patients nonoperatively treated do as well or better than patients treated surgically for blunt abdominal injuries (23, 29, 31, 34, 47, 74-75, 77), it has only been within the past decade (33, 45) that nonoperative management has become the standard of care, making a long-term study such as this one difficult to carry out because both the length of time and number of patients that have been nonoperatively treated has not been sufficient. Because no data has been available, it has been assumed that nonoperative management does not have any long-term consequences to the patient's health, and that the consequences are less than if patients were treated surgically.

Looking at the outcome studies for patients treated surgically for blunt abdominal injuries to the liver and spleen, it is easy to see why conservative management has become so prominent. Some of the complications from surgical management include overwhelming post-splenectomy sepsis, infection, small bowel obstruction, hemorrhage, a higher risk of hepatitis B, hepatitis C, and HIV from an increased use of blood products, death, and numerous other complications (50, 80). In examining the data from this series, it would appear that the actual number of

long-term complications that are likely due to nonoperative treatment is extremely small.

According a meta-analysis by Holdsworth et al, the incidence of overwhelming post-splenectomy sepsis in adults is 0.9 percent with a mortality rate of 0.8 percent, with 52% of all first infections occurring within the first 2 years after splenectomy (81). According to Pimpl et al, splenectomy generates a considerable life long risk of both severe infection and thromboembolism (82). Although the incidence of infection is much lower than in children (an incidence of 4.4 percent with a mortality rate of 2.2 percent), adults who do develop a post-splenectomy infection appear to develop a septicemic illness with a higher mortality rate, making this complication in adults extremely worrisome (81). Because the spleen is not removed in nonoperative management, overwhelming post-splenectomy sepsis would not be expected to occur in conservatively managed patients. In this series, there was one patient (#1111) that developed overwhelming infection during nonoperative management. This patient had a Grade I splenic and Grade II liver laceration, and developed both pneumonia and a wound infection before he became septic. Because of this, it is unclear what role his splenic injury may have had in the development of his sepsis. Two other cases of sepsis did occur in two of the patients (#1520 and #4027) that failed nonoperative treatment and had to be operatively managed. One patient (#1520) had a splenectomy and became septic with Candida post op. The other patient (#4027) had a Grade II splenic injury and was operated on for gangrenous cholecystitis and had a sigmoid colectomy, but did not have splenectomy or splenorrhapy performed. He was noted to have developed MRSA

line sepsis, so appears to have become septic from an indwelling catheter rather than because of compromised splenic function. Although the 2.7% of patients in this series that developed sepsis is greater than the 0.9% incidence of post-splenectomy sepsis described by Holdsworth et al (81), the cause of sepsis in two of the patients is most likely unrelated to their splenic injuries. If these two patients are excluded, then 0.9% of the patients in this series developed sepsis, which is agreeable to the rate described by Holdsworth et al. No patient in this series was noted to develop sepsis or be readmitted for sepsis after discharge.

The incidence of small bowel obstruction after surgery is reported to be 0.69 percent (80), with a 1.12 percent incidence for trauma patients (80) within the first 4 weeks after surgery. The mortality rate for small bowel obstruction is reported to be 17.8 percent (80). According to Tortella et al, the incidence for small bowel obstruction after celiotomy for penetrating abdominal trauma is 7.4 percent (83), and although the incidence is not likely to be the same for nonpenetrating trauma, these patients have similar risk factors for adhesions and small bowel obstruction such as possible contamination from bowel contents, hemoperitoneum, and tissue damage secondary to trauma. In this series, approximately 54 patients were noted to have hemoperitoneum on their CT scan. Neither any of these patients, those operatively treated, or any patients without hemoperitoneum noted on CT scan developed small bowel obstruction as a complication. The 0.0% incidence of small bowel obstruction noted in this study compares favorably to both the reported 1.12% incidence for all trauma patients, and the 7.4% incidence for post-celiotomy patients with penetrating abdominal trauma. However, because the sample size is limited, the actual

incidence of small bowel obstruction after nonoperative treatment may be larger than indicated by this study.

Numerous studies have shown that nonoperatively treated patients require fewer units of blood products (44, 74-75), and are therefore at decreased risk for acquiring blood borne infections. In a review by Goan et al, the average number of blood products received by nonoperatively managed versus operatively managed patients was 3.3 units versus 7.8 units (47). In this series, the average number of units of blood products received in nonoperatively managed patients transfused was comparable to that found by Goan, at approximately 3.4 units per patient, while those that failed nonoperative treatment received on average 14.3 units. This reinforces the results of previous studies that show nonoperatively treated patients receive fewer blood products than operatively managed patients, and are therefore at lower risk for transfusion related infections.

Current estimates of the risk of infection from blood transfusion range from 1 in 40,000 to 1 in 225,000 per unit for HIV (84-86), 1 in 200,000 per unit for Hepatitis B (86), 1 in 3,300 per unit for Hepatitis C (87), and 1 in 50,000 per unit for HTLV-I and HTLV-II (86). It would appear from this data that because of the decreased number of blood products needed, nonoperatively treated patients would have a much lower incidence of transfusion related diseases. In this survey, only one patient (#992) in the series developed a post transfusion infection (Hepatitis C). Even though this patient was nonoperatively managed, this patient required many more units of blood products than most patients that are operatively managed, requiring 17 units.

Two patients (#202 and #1520) that required exploratory laparotomies secondary to hemodynamic decompensation were transfused more blood products than the patients nonoperatively managed. Patient #202 required only slightly more blood products than the average patient in this study (4 units versus 3.4 units), but during exploratory laparotomy was not found to have any source of bleeding within the abdomen. This patient did not need to be operated on, so was not truly a failure of nonoperative management. However, patient #1520 required 37 units of blood products during his entire admission, and was emergently taken to the operating room for an exploratory laparotomy on hospital day 9. No source of bleeding was found at that time, but 10 days later was taken back to the operating room because of hemorrhage from erosive gastritis. Because this patient had a Grade III splenic injury, a splenectomy was performed as well secondary to his blood loss. The third patient (#4027) that required operative management for metastatic colon carcinoma and gangrenous cholecystitis only required 2 units of PRBCs for his colectomy. None of these patients developed blood borne diseases post transfusion.

Although only one of the patients in this series (#992) developed a post transfusion infection, this complication reinforces the fact that transfusion of blood products does carry with it a significant risk. Every attempt should be made to minimize transfusion of blood products whenever possible. In some cases this may require the patient to be nonoperatively managed, but in other cases operative management may be the best choice for the patient.

In this series, there was one patient death that occurred during admission. Patient #1030 died on hospital day #4 secondary to acute decompensation from

asthma. His past medical history included COPD, a remote history of tuberculosis, history of asthma, and oxygen dependency at home. His splenic injury and liver injury were not very severe (both Grade I), and his only other associated injury was a small occipital scalp laceration. Although the patient did have abdominal injuries, it would seem that his death cannot be directly attributable to those injuries or to nonoperative management, making it difficult to truly consider this complication a failure of nonoperative management.

For the rest of the patients described, it seems very unlikely that their current medical conditions are related to either their abdominal injuries or to their nonoperative management. Most had preexisting conditions to account for their medical problems, or had risk factors that makes the contribution of nonoperative management likely very small. With a few of the patients, such as those with advanced liver disease, the blunt hepatic injuries did not likely help their condition, but most likely did not contribute significantly to their cirrhosis.

Of course, this survey has some serious limitations. First of all, the patients surveyed did not all follow-up after discharge, so it is impossible to determine their long-term outcomes. If one assumes that if a serious complication arose that they would return to the hospital, then this would not be a problem. By looking at our data, it would appear that this would bear out since 47 patients were seen again for reasons other than routine follow-up. However, if patients died outside of the hospital system, obtained their follow-up care at another institution, or decided not to return to the hospital despite serious medical illnesses, looking for further admission

or re-evaluation data in the patient chart would fail to survey this patient sub-population.

Another limitation to this survey is the patient population itself. Because a large amount of blunt abdominal injury is secondary to assault and because this level I trauma center sees a large amount of the indigent population, many of the patients surveyed have coexisting medical conditions, previous history of abdominal injury, and multiple risk factors for blood borne illnesses that make it more difficult to determine what role if any nonoperative management had on their current health status. Also, many patients in the survey population had previous abdominal operations that make it difficult to determine what role nonoperative management would have in the formation of adhesions, etc.

A third limitation of this survey would be the power of the study. Since no previous study has been performed, it is impossible to predict what complications, if any would be found in the long-term. It is very possible that some complications may be so rare that only a much larger study would be able to uncover them. However, without knowing what complications to expect, completing such a study would be an overwhelming task, and a smaller study, such as this one would be useful in determining an appropriate study size to look at the incidence of certain complications if they need to be further studied.

Conclusion

As nonoperative management has progressed over the years, we are now managing more severe injuries in more multiply injured patients than ever before.

Because of this, the short-term complication and failure rates of nonoperative management have changed as criteria for nonoperative management have become more liberal (1, 47), and from this, we can expect that long-term complications may change as well. Although further studies need to be done, our initial experience with the management of Grade III and IV injuries has not shown an increase in specific complications.

Improvements in care can only be made if the time is taken to look at current practices and determine what their consequences towards the patient really are. In the case of nonoperative management of blunt abdominal injuries, it appears from this study that the incidence of serious complications is likely minimal.

Because it has already been shown that nonoperative management of blunt abdominal injuries results in shorter mean lengths of stay, quicker recovery, use of fewer blood products, and a decreased morbidity and mortality rate (23, 29, 31, 34, 47, 74-75, 77), this new information that shows the long-term complications to be minimal is encouraging.

Although prospective randomized trials should be carried out to confirm these findings, the initial data presented here is promising. The long-term outcomes for patients treated nonoperatively for blunt abdominal injuries appears to be excellent from a medical standpoint, so now physicians can be comfortable when informing patients of their long-term prognosis.

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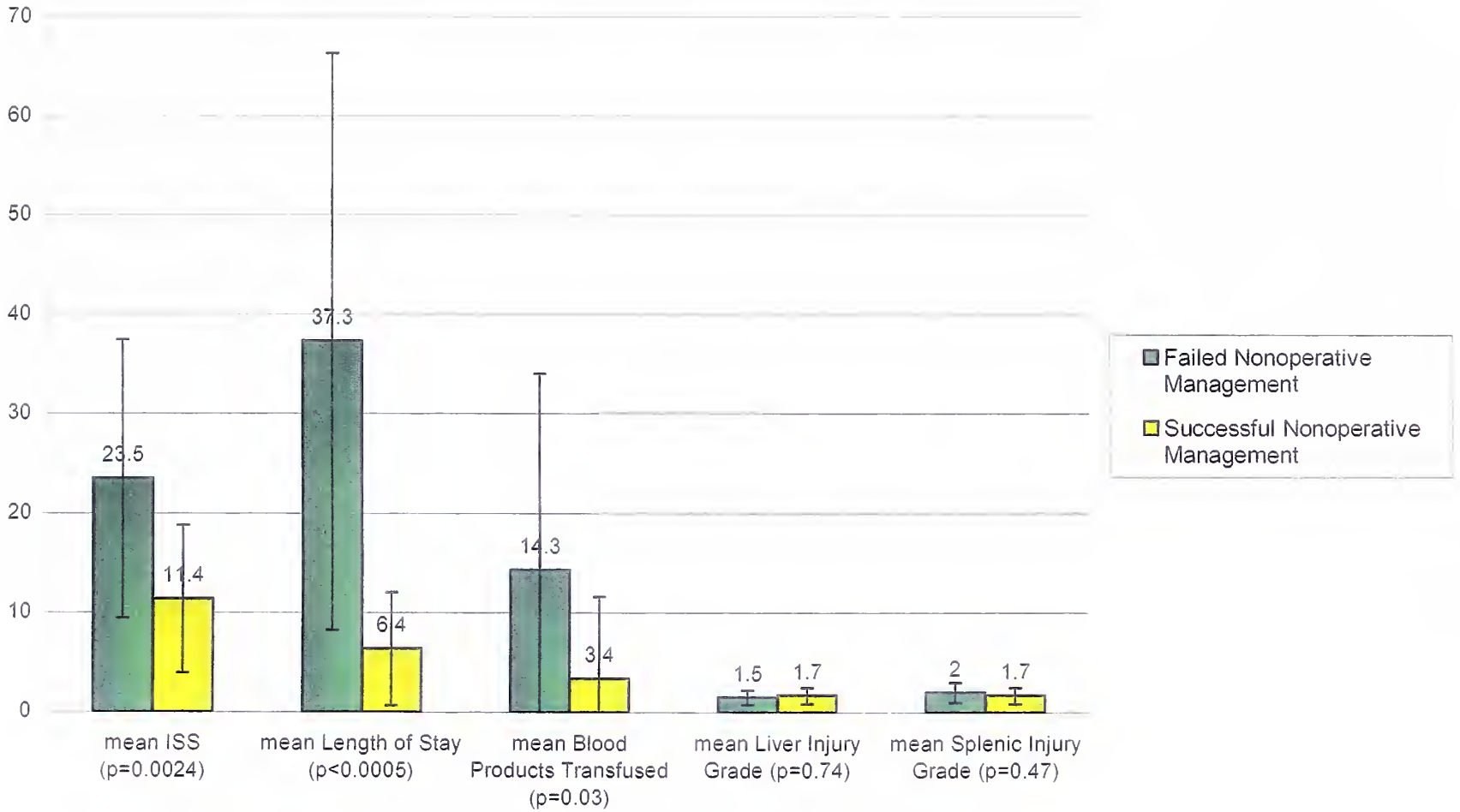

Figure 1: Comparison of Succesful and Failed Nonoperatively Managed Patients

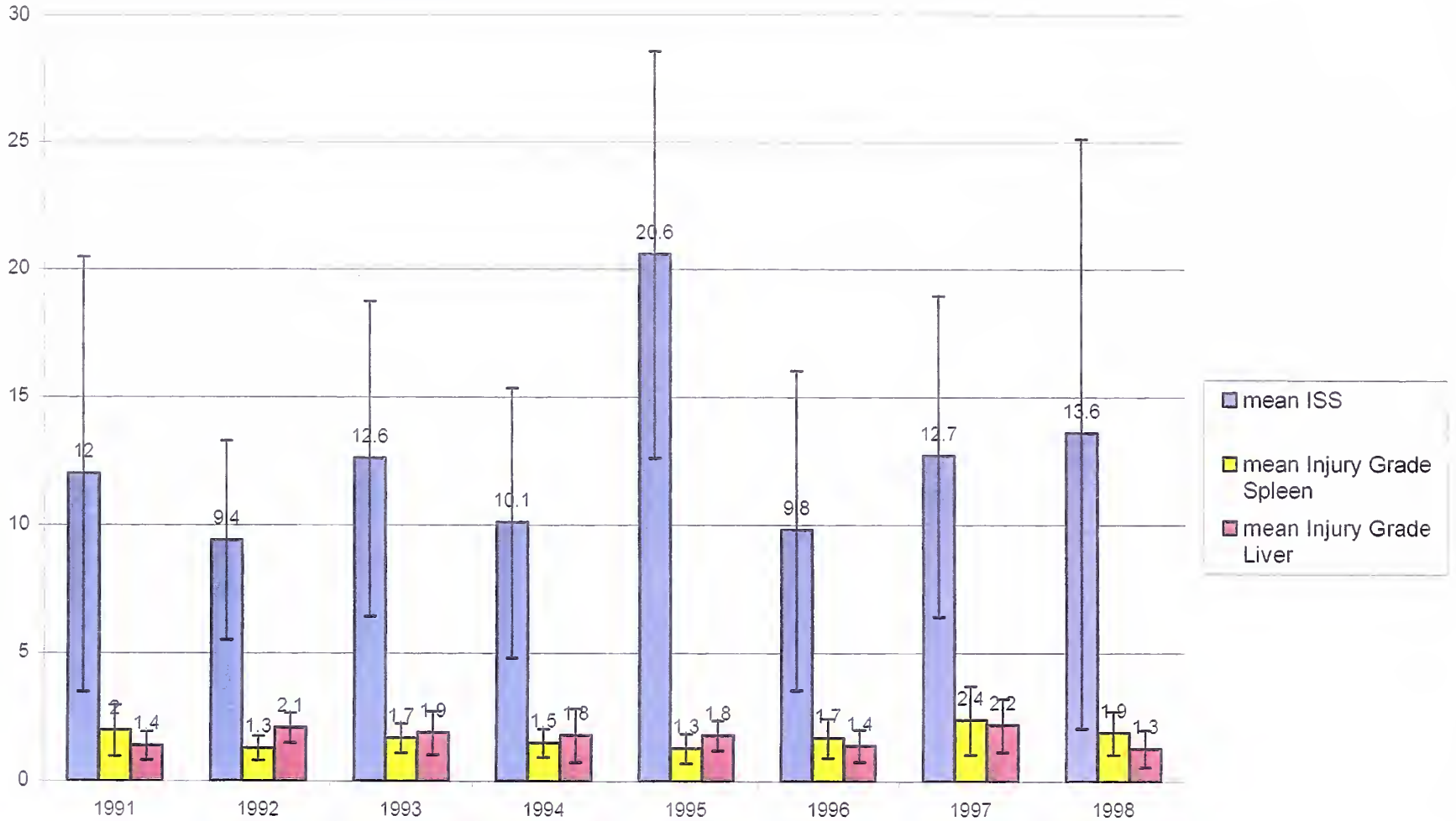

Figure 2: Patient Injury Data Stratified by Year

There is a significant difference between the mean Injury Severity Score of the patients when they are stratified according to year (p=0.032), but no difference between mean Splenic (p=0.31) or Liver (p=0.16) Injury Grades.

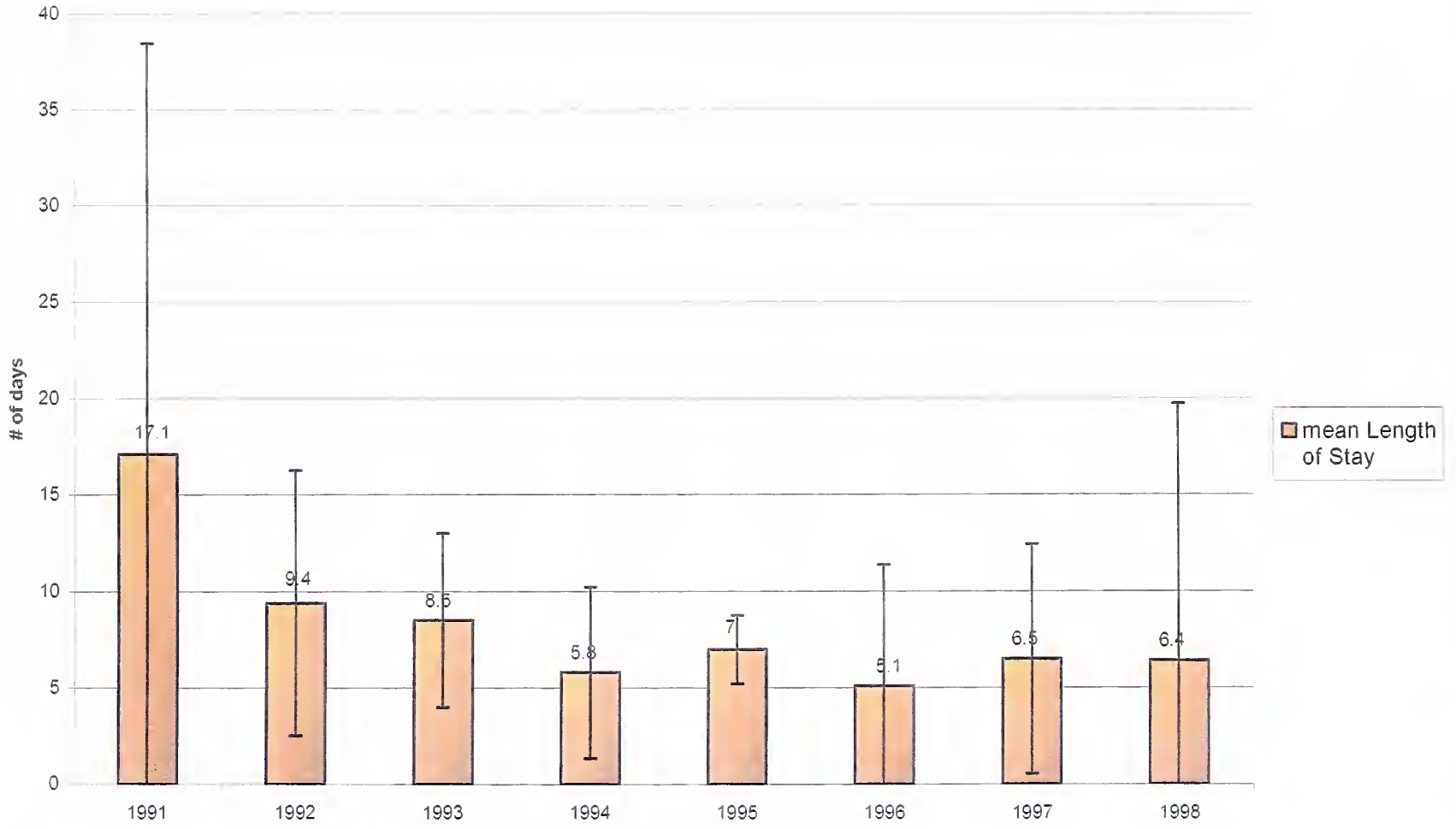

Figure 3: Mean Length of Stay Stratified by Year

There is no significant difference between the length of stay of hospital patients when stratified by year (p=0.15).

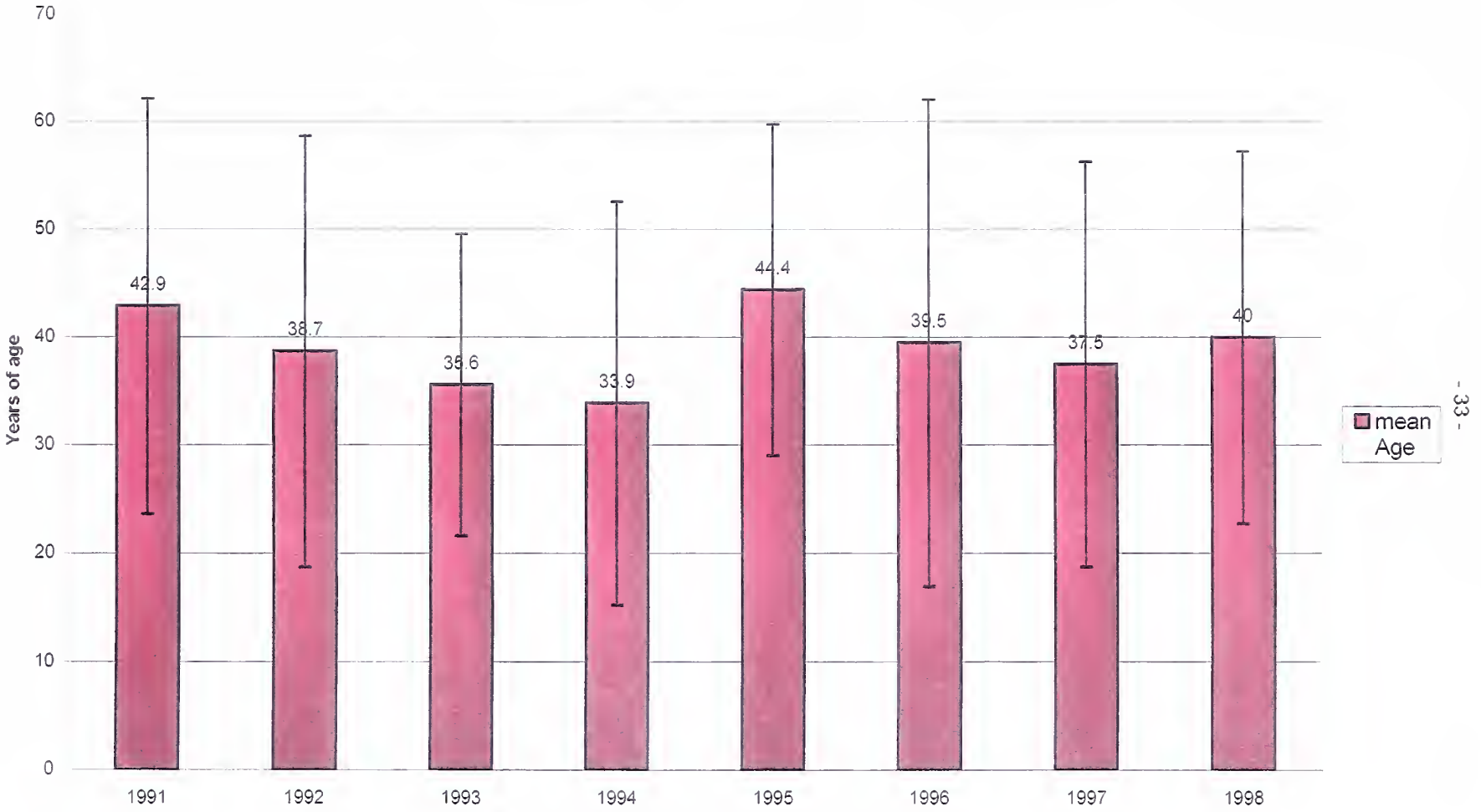

Figure 4: Mean Age Stratified by Year of Injury

There is no significant difference between the ages of the patients treated when they are stratified by year (p=0.94).

Table 1: Injury Data for Nonoperatively Treated Patients (n=108)

Pt #	Injury/ Grade	GCS	ISS	Units blood	F/U revaluations	Readmits	Abd Ops post discharge	Hemoperitoneum	Other Ops (Non-abd) during admission
123	Spleen II	15	29	1.5	Y			Y	
158	Liver I	15	8	0	Y				
234	Spleen I		13	5	Y				Y
404	Liver III	15	17	2.5	Y			Y	
428	Liver I	15	6	3					
442	Liver III	15	41	0				Y	
485	Liver III	15	14	2	Y	Y		Y	
492	Liver III	15	8	1	Y	Y			
508	Liver III	15	5	0	Y			Y	
528	Spleen I	15	12	0					
539	Liver I	14	17	0	Y	Y			
674	Liver I	15	13	0	Y	Y	Y		
678	Liver I	13	17	0					Y
681	Liver I	15	9	0	Y				
726	Spleen III	15	10	2	Y	Y		Y	
792	Liver II	15	8	0	Y			Y	Y
838	Liver II	15	5	0	Y	Y			
851	Spleen II	14	10	3	Y	Y			
900	Liver I	15	13	0	Y				
928	Spleen II	15	14	0	Y				
992	Spleen II Liver I	15	9	17	Y			Y	
1001	Spleen I	15	5	0					
1026	Liver I	15	5	0	Y	Y			
1045	Liver I	15	8	0					
1058	Spleen I	15	22	0	Y			Y	
1067	Spleen I Liver II	15	17	2	Y				Y
1111	Spleen I Liver II	15	29	6	Y	Y		Y	Y
1273	Spleen II	15	13	0	Y			Y	
1293	Spleen II	15	18	0	Y			Y	
1336	Spleen I	15	13	0					Y

Table 1 (cont.)

Pt #	Injury/ Grade	GCS	ISS	Units blood	F/U revaluations	Readmits	Abd Ops post discharge	Hemoperitoneum	Other Ops (Non-abd) during admission
1359	Spleen II	15	8	0	Y			Y	Y
1472	Liver I	15	5	0	Y	Y	Y		
1536	Liver I	15	12	0				Y	
1557	Liver II	15	4	0					
1581	Spleen II Liver I	15	18	0	Y				
1583	Spleen I Liver I	13	9	0	Y			Y	
1640	Spleen III	15	5	3				Y	
1681	Liver II	15	17	0					
1698	Spleen I	15	24	0	Y				
1718	Liver III	15	9	0	Y			Y	Y
1727	Liver II	15	14	0	Y			Y	
1739	Spleen I	15	9	0	Y				
1788	Spleen III Liver I	15	17	0	Y				
1846	Liver II	15	9	0	Y				
1855	Spleen III	15	20	0	Y				
1904	Liver I	15	5	0	Y				
1929	Liver II	15	10	0	Y		Y	Y	
1930	Liver I	15	4	0	Y		Y		
1938	Liver III	15	10	0	Y			Y	
2050	Spleen I Liver I	15	9	0	Y			Y	
2196	Spleen I	15	9	0	Y				
2237	Liver I	15	4	0	Y				
2259	Spleen II	7	20	0	Y			Y	Y
2281	Spleen I	15	9	0	Y				Y
2321	Liver II	14	13	0	Y	Y			
2345	Spleen I	15	13	0	Y	Y		Y	
2362	Liver III	15	22	0	Y			Y	
2393	Spleen I	15	5	0	Y				

Table 1 (cont.)

Pt #	Injury/ Grade	GCS	ISS	Units blood	F/U revaluations	Readmits	Abd Ops post discharge	Hemoperitoneum	Other Ops (Non-abd) during admission
2410	Liver I	15	5	0	Y				
2486	Spleen I	15	6	0	Y				
2505	Spleen I	15	9	0	Y	Y	Y		Y
2577	Spleen I	15	17	2	Y				
2683	Spleen III Liver I	15	12	2	Y			Y	
2730	Spleen I	15	4	0	Y			Y	
2812	Liver II	15	20	0					
2849	Liver II	15	8	0	Y	Y	Y		
2856	Liver II	15	14	0				Y	
2882	Liver II	15	10	0	Y				
2896	Liver II	14	5	0	Y			Y	
2901	Liver I	15	4	0	Y				
3000	Liver I	15	5	0	Y				
3009	Liver III	6	43	0	Y			Y	
3146	Spleen I	15	4	0	Y			Y	
3200	Spleen II	15	13	0	Y			Y	
3207	Liver III	15	14	0	Y			Y	
3243	Spleen II	15	4	0				Y	
3291	Spleen II	15	5	0	Y			Y	
3328	Liver II	15	9	0	Y			Y	
3370	Liver III	15	9	2	Y	Y		Y	
3375	Spleen III Liver I	15	19	0	Y			Y	
3453	Spleen II	15	10	0	Y	Y		Y	
3457	Liver III	15	22	2	Y	Y		Y	Y
3482	Liver I	15	5	0				Y	
3531	Liver I	15	17	0	Y	Y			
3618	Spleen I		10	0	Y	Y			
3627	Spleen I Liver I	15	13	0	Y				
3706	Liver I	15	5	0	Y				
3735	Liver I	15	14	0	Y	Y			Y

Table 1 (cont.)

Pt #	Injury/ Grade	GCS	ISS	Units blood	F/U revaluations	Readmits	Abd Ops post discharge	Hemoperitoneum	Other Ops (Non-abd) during admission
3749	Spleen I Liver I	15	13	0	Y			Y	
3751	Liver IV	15	4	0	Y			Y	
3853	Spleen II	15	13	0	Y			Y	
3880	Spleen III	15	27	0	Y			Y	
3910	Spleen I	15	4	0	Y				
3950	Spleen IV	15	9	0				Y	
4029	Spleen II	15	9	0				Y	
4030	Spleen II Liver I	15	5	0				Y	
4075	Liver II	15	9	0				Y	
4132	Liver I	15	4	0				Y	
4329	Spleen II Liver I	15	25	0	Y			Y	
4353	Liver II		5	0	Y			Y	
4372	Liver I	15	9	0	Y				
4415	Spleen I Liver II	15	6	0	Y			Y	
4430	Spleen I	15	5	0	Y				
4480	Liver III	15	5	0				Y	
4516	Liver I	15	4	0					
4538	Liver I	15	4	0	Y				
4588	Spleen II	15	12	2	Y				
4701	Liver I	15	5	0				Y	
Average	*Spleen 1.7 Liver 1.7*	*14.8*	*11.4*	*0.54*					
SD	*Spleen 0.79 Liver 0.83*	*1.2*	*7.4*	*1.9*					
median	*Spleen 1.5 Liver 1.0*	*15*	*9*	*0*					
Total #					*85*	*20*	*6*	*54*	*13*

Table 2: Injury Data for Failure of Nonoperative Management Patients (n=4)

Pt #	Injury/ Grade	GCS	ISS	Units blood	F/U revaluations	Readmits	Abd Ops post discharge	Hemoperitoneum	Other Ops (Non-abd) during admission
202	Liver II	15	22	4	Y	Y		Y	
1030	Liver I, Spleen I	11	5	0	N/A	N/A			
1520	Spleen III	15	29	37	Y	Y	Y	Y	Y
4027	Spleen II	15	38	2	Y				Y
Average	*Liver 1.5 Spleen 2.0*	*14*	*23.5*	*14.3*					
SD	*Liver 0.7 Spleen 1.0*	*2.0*	*14.0*	*19.7*					
Median	*Liver 1.5 Spleen 2.0*	*15*	*25.5*	*4.0*					
Total #					*3*	*2*	*1*	*2*	*2*

Table 3: Stratification Data for all Patients (n=112)

	# of patients	mean age	mean Injury Grade Spleen	mean Injury Grade Liver	mean ISS	# patients with hemoperitoneum	# patients receiving transfusions	# units transfused	# patients readmitted	mean Length of Stay
1991	7	42.9	2.0	1.4	12	4	4	14.5	1	17.1
1992	20	38.7	1.3	2.0	9.4	8	3	2.7	4	9.4
1993	10	35.6	1.7	1.9	12.6	6	2	2.3	4	8.5
1994	14	33.9	1.5	1.8	10.1	6	0	0	4	5.8
1995	8	44.4	1.5	1.8	20.6	5	1	1.5	0	7
1996	20	39.5	1.7	1.4	9.8	8	5	3.2	6	5.1
1997	15	37.5	2.4	2.2	12.7	11	4	2.8	1	6.5
1998	18	40	1.9	1.3	13.6	6	1	2.0	2	6.4
total	*112*					*54*	*20*		*22*	

Table 4: Patient Information

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
123	Spleen II	29y PTA hysterectomy		R facial fxs (NOS) large R hemothorax bilateral sm ptx	1 wk f/u CT, lac resolving
158	Liver I	5y PTA colon CA resection	Colon CA	L tibial plateau fx	4 mo f/u, R lobectomy/choly for met CA
234	Spleen I	S/p appy	CP	Fracture of left patella Comminuted tibial plateau fx	6 y f/u ED R hip fx
404	Liver III		ETOH	Concussion with LOC < 1 hour Right medial malleolar fx	10 d f/u CT Liver lac impr, 1mo f/u doing well
428	Liver I			Rib fx's Scalp lac Tongue lac	
442	Liver III	Unknown		Rt flail chest small temporal parietal EDH kidney lac duodenal hematoma	
485	Liver III			left clavicle fracture multiple abrasions	5 d f/u CT Liver lac impr; 7 y f/u, sm hiatal hernia
492	Liver III		Parkinsons, depression	left tibial plateau fracture	Phys therapy; 8y f/u readmitted, Paralysis agitans
508	Liver III	Colostomy	ETOH	R rib fx (9th)	6d f/u CT no change
528	Spleen I	Cholecystectomy	Gallbladder	Left clavical fx Concuccion and head laceration with LOC	
539	Liver I	L hernia repair	MVC 4 y PTA, fall 12 y PTA	right zygoma fracture ethmoid sinus fracture multiple abrasions	3d f/u CT unchanged; 7 y f/u – repeat MVC, tiny new liver lac, readmitted to ortho
674	Liver I			fx >3 ribs on R side (4th, 5th, 6th, 7th)	½ y f/u CT unchanged, idiopathic retroperitoneal fibrosis; 2y f/u colon diverticulitis; 4y f/u R CEA; 7y f/u Elap, appy, ureterolysis (liver noted to be normal)

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
678	Liver I		Pit tumor	L pneumothorax Lateral nasal bone fx R globe lac Rib fx 4,7,8	
681	Liver I	TAH-BSO, omentectomy, appy, serous papillary CA of ovary 8y PTA, umbilical hernia repair	Breast cyst excision 50y PTA	Lt acetabular fx	2y f/u malig neoplasm of ovary
726	Spleen III		Hep B + 3mo PTA, HIV + 2y PTA, h/o drug abuse	Left chest wall contusion left flank contusion hemoperitoneum	23d f/u CT, resolved hemoperitoneum, persistant heterogeneity in spleen; 5y f/u Kaposi's Sarcoma; 6y f/u AML
792	Liver II			lumbar spine fracture bilateral patella fractures L clavicular fx	2 mo ortho f/u doing well; 4y f/u – MVC, d/c'd; 6y f/u – MVC, d/c'd
838	Liver II		MVC-R thigh lac	>3 rib fractures one side and <3 other side with hemo/ptx	1mo f/u no abd c/o, chest tube reinserted for PTX
851	Spleen II	Colostomy 15y PTA, cholecystectomy, hernia repair, prostatectomy, MVC 6y PTA	AdenoCA of rectum 15y PTA	forehead lac 1 2 cm	1 mo f/u, doing well; 6 mo f/u readmitted for MI, died
900	Liver I		PPD +, HIV +, HCV +, Drug Abuse	L tibial plateau displaced fx	1y HIV f/u liver dz, coagulopathy
928	Spleen II		ETOH, prev trauma	bilateral maxillary and ethmoid sinus fractures	5d f/u CT subacute splenic hemorrhage, ? cecal mass; 12d f/u, doing well; 14d f/u, no cecal mass confirmed with BE
992	Spleen II Liver I		Breast CA 5y PTA	left hip dislocation right ulna fracture right soft tissue avulsion above ankle	3d f/u CT, no change; 3 mo f/u CT stable, Hep C + (post-transfusion hepatitis); 6 mo f/u CT stable; 8y Digestive Diseases f/u for hepatitis, low viral load, nl liver function

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
1001	Spleen I		3y PTA fall; 4y PTA fall, 6 y PTA MCC (no abd trauma), + ETOH, + intranasal cocaine	rib fxs L 9th and 11th	
1026	Liver I		HTN	rib fx	1 ½ y f/u readmit for MI
1045	Liver I	15d PTA L hernia repair, R thigh lipoma excision	Prev trauma; 16 y PTA, hematemesis +guiac, neg UGI	T12 transverse process fx T12 compression fxs	
1058	Spleen I		HTN	small right cerebral contusion right comminuted distal radius fracture	4d f/u CT with decreased fluid; 11d f/u doing well
1067	Spleen I Liver II			right Colle's fracture pulmonary contusion multiple right rib fractures pneumothorax	3wk f/u, doing well
1111	Spleen I Liver II		ETOH	R subtrochanteric femur fx L segmental proximal to midshaft femur fx R tibial plateau fx	1d f/u CT no fluid; 2y f/u MVC (left AMA)
1273	Spleen II	Ovarian cystectomy, appy, elap	Diverticulitis, mesenteric adenitis	Multiple metacarpal fxs	1 mo f/u doing well
1293	Spleen II		MVC 2 y PTA	Left 5, 6 rib fractures with pulmonary contusion and hemothorax	6d f/u CT, resolving contusion; ½ mo f/u, doing well; 1 mo f/u, doing well; 1 y f/u s/p fall, no BM probs
1336	Spleen I		Bone spur R knee	left acetabular chip fracture L1 burst fracture with 40% loss of height	
1359	Spleen II		ETOH, 6y PTA MVC with trach/epiglo-tectomy	Comminuted distal tib-fib fracture	1mo f/u doing well; 2 mo f/u doing well

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
1472	Liver I	Elap 5y PTA, liver lac s/p stab wound	Liver lac s/p stab wound 5y PTA; 12 y PTA LLQ pain given antacids, IVDA, Hep B, Hep C	lac to forehead	1y f/u superificial stab wound; 1 ½ y f/u readmission for assault, L rib pain, L jaw pain; 2y f/u readmission for GSW to pelvis/abd, surgery for multiple anastomosis, enterotomies, repair of mesenteric A&V, L common iliac ligation, LLE fasciotomy, IVC filter placement
1536	Liver I	TAH/BSO, appy, cholecystectomy	Focal adenomatous hyperplasia of uterus, ETOH with abnl LFTs, diverticulitis	L rib fx multiple R trimalleolar fx	
1557	Liver II				
1581	Spleen II Liver I			L kidney lac small ptx multiple rib fx Transverse process fx L3-L4	1mo f/u doing well
1583	Spleen I Liver I		IVDA, HIV +3y PTA, Hep B + 8y PTA	multiple abrasions concussion	1d f/u CT no progression, decreased fluid
1640	Spleen III	PEG	Esophagitis, C diff colitis,CVA, COPD, NIDDM	large lac back of head	
1681	Liver II	B/l hernia repair		R knee abrasion	
1698	Spleen I		5y PTA MVC	Left flail chest left AC seperation small left ptx	5d f/u CT increased fluid, no change spleen; 4 y f/u pulmonology
1718	Liver III			l1 l2 l3 l4 l5 transverse process fxs 1 cm kiney lac	½ mo – 5 mo f/u, doing well
1727	Liver II		Lung CA 6 y PTA, pneumonectomy 4 y PTA	R rib fx Open forehead lac outer table skull fx	3d f/u CT increased hematoma liver lac; 1 mo f/u chest clinic

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
1739	Spleen I			multiple abrasions shoulder dislocation	3d f/u CT no abnl; 2 mo f/u CT nl; 1 mo f/u doing well; 2 mo f/u doing well
1788	Spleen III Liver I			orbital fx tripod fx pterygoid fx anterior maxilla wall fx zygomatic fx	1 d f/u CT unchanged; 13d ENT f/u doing well
1846	Liver II		MVC 8 y PTA	back abrasions	1 ½ y f/u doing well
1855	Spleen III		ETOH, Hep C, IVDA	anterior rib fxs L 5,6 posterior rib fxs multiple 5-11 flail chest	½ mo f/u s/p fall, CT neg; 1y f/u vomiting and diarrhea secondary to withdrawal; 1 ¼ y ortho f/u for hand
1904	Liver I		Schizo, CHI in childhood and 7y PTA	R knee abrasion multiple head abrasions	2 ½ mo f/u, doing well; 2 ½ y f/u psych SI
1929	Liver II (1st injury)	8y PTA Classical C sec	Drug abuse, 3 mo PTA HIV neg, Hep B pos/antibody neg	lac R knee	½ mo f/u doing well; 1 mo f/u doing well; 1 ½ mo f/u readmission for repeat C sec and BTL; ½ y f/u arm abscess; 3 ½ y f/u ED for cough, left AMA
1930	Liver I (2nd injury)	Same as above	Same as above		2d f/u CT unchanged hematoma; Same as above
1938	Liver III		ETOH, schizo	forehead abrasion LOC	1d f/u abd XR with some dilated small bowel, nl lg bowel
2050	Spleen I Liver I		IVDA, spontaneous pneumothorax		2mo f/u, s/p assault to face; 2 mo f/u c/o visual field abnl; 1 ½ y f/u fibular fx
2196	Spleen I		IVDA, ETOH, HIV + by report, dog bites, s/p SI	proximal fibula fx L tibia avulsion fx facial lacs	½ mo f/u fx clinic
2237	Liver I		GC/chlam urethritis, 11y PTA human bite		11d f/u chest pain
2259	Spleen II		IDDM	hemomediastinum	19d, 24d, 33d, 42d, 46d ENT f/u trach healing well

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
2281	Spleen I		IDDM, kidney tx 4y PTA, hypercholesterolemia	right tibial plateau fracture fracture l2 and l3 transverse process 2 left lower rib fx	Mult f/u; 1 ½ y f/u bloating after meals while adjusting insulin
2321	Liver II		19d PTA, tx 1u PRBCs, 23y PTA MVC rib fx; CEA, CVA	left 7, 8 rib fractures with small pneumothorax	3d f/u abd XR colon air fluid levels, nonobstructive pattern; 3 y f/u abd XR nonspecific pattern, aspiration pneumonia
2345	Spleen I		ETOH, elevated bilis, inferior wall ischemia	L side ptx L lower back hematoma L lung contusion L mult rib fxs	11d f/u doing well; 1 mo f/u doing well, CT no evidence lac, no fluid; 1y f/u BRBPR with clots, new afib, orthostasis, ETOH
2362	Liver III	6y PTA appy	Migraines	R pneumothorax Pulmonary contusion R clavicular fx	14d f/u doing well; 1 mo f/u, doing well, CT improvement liver lac
2393	Spleen I	18y PTA b/l hernia repair	1 mo PTA MVC + ETOH	Occipital scalp laceration multiple abrasions	5d f/u doing well; 12d f/u heme + stool; 1 mo f/u no abd c/o; 1 ½ mo f/u no abd c/o
2410	Liver I		Mental retard, sz d/o; 12y PTA nl barium swallow	multiple facial and neck abrasions	5 d f/u doing well; 4y f/u for sz; 4 ½ y f/u for sz, then dilantin toxicity
2486	Spleen I	19y PTA L hernia repair	12 y PTA colonoscopy adenomatous polyp removal in rectal vault; 7y colonoscopy internal hemorrhoids; 2y PTA diverticulosis; 19y nl barium enema; 16y blunt trauma to R flank with contusion	open finger dislocation, L 4th prox interphalangeal joint back contusion	1d f/u CT unchanged; 5d f/u CT splenic lac improved; 8 d f/u CT splenic lacs improved, clot in R iliac vein; 2y f/u colonoscopy for BRBPR - tics and internal hemorrhoids found
2505	Spleen I		Posterior spinal fusion 21y PTA	5,6,7,8 right rib fractures thoracic 9 transverse proc fx	2 ½ mo f/u readmission for resection of L renal artery aneursym

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
2577	Spleen I			left hip dislocation left maxillary sinus fracture	3d f/u CT stable lacs, no fluid;1 mo f/u doing well
2683	Spleen III Liver I	30y PTA partial gastrectomy	ETOH, tobacco, MI, HTN	L chest hematoma 4th R rib fx L rib fxs	C/o odynophagia with hematemesis that resolved by d/c; 2d f/u CT increased fluid, no active bleeds; 8d f/u CT improvement liver & spleen; 1mo f/u no abd c/o; 2 ½ mo f/u doing well
2730	Spleen I		ETOH, tob, mult superficial GSW, 17y PTA MVC; 15y MCC; 2 y s/p assault scalp lac; 3 mo PTA hand fx		½ y f/u s/p assault left AMA; 1 ½ y f/u s/p stab wound L arm and leg; 2y f/u s/p stab wound upper back; 5y f/u s/p traumatic amputation of finger
2812	Liver II		ETOH, tob, smoke drugs; MVC 24y PTA muscle strain	Forehead contusion with concussion and LOC < 1 hour	
2849	Liver II	3y PTA primary C sec; 6y PTA appy	ASA overdose (8-10 ASA) 9y PTA	left ulna fracture	Excision of melanoma from back; 4y f/u readmission for C sec; 6y f/u readmission for C sec
2856	Liver II	9y PTA appy		3 cm lip laceration chin avulsion left basilar skull fracture	
2882	Liver II			small abrasion mid forehead small abrasion rt hand	9d f/u no abd c/o; 1y f/u s/p MVC; 1 ½ y f/u s/p fall stairs; 1 ¾ y f/u s/p fall stairs, s/p another fall
2896	Liver II	S/p appy	ETOH, HTN gout, R lacunar infarct	renal laceration right 4, 6, 7 rib fractures L3 transverse process fx	6d f/u CT no change, no fluid;1 mo f/u doing well
2901	Liver I		IVDA, ARF		4y f/u Hep B and C +, HIV + (no blood tx from trauma)

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
3000	Liver I	3y PTA RUQ hernia; 1 ½ mo PTA recurrent hernia RUQ	ETOH, esoph stricture, esophagitis/ulcer, dysphagia 3y PTA, h/o Barrett's, asthma	contusion left thigh	1 mo f/u doing well, RUQ hernia; 3 mo f/u esophagitis/ulcer/dysphagia; 4 mo f/u doing well, RUQ hernia
3009	Liver III			rib cage fx 3 ribs on R side (7th, 8th, 9th) with ptx	1 ½ mo f/u difficulty swallowing solids, no abd c/o
3146	Spleen I				1 mo f/u u/s, spleen partial healing; 2 mo f/u u/s, healing spleen, 2 contusions
3200	Spleen II			5th rib fx L side L pulmonary contusion	9d f/u no abd c/o
3207	Liver III			metatarsal fx 2nd & 3rd R foot	½ mo f/u doing well
3243	Spleen II				
3291	Spleen II			L supraorbital lac	8d f/u no abd c/o; 2 ¼ mo f/u MVC, left AMA, c/o back/neck pain
3328	Liver II				7d f/u u/s, liver healing
3370	Liver III				½ mo, 1mo, 1 ½ mo, 1 ½ mo, 2 mo, 2 ½ mo f/u doing well; 1y f/u hyperglycemic nonketotic syndrome, 1 ½ y CVA
3375	Spleen III Liver I			Abrasion lt forearm Abrasion lt flank	½ mo f/u doing well; 1 mo f/u no abd c/o; 1 ½ mo f/u L chest pain; 3 ½ y ENT f/u mouth lac
3453	Spleen II		Hemophilia, Jehovah's witness	R facial contusion	1 d f/u CT no change, no fluid; 10 d f/u CT evolution splenic hematoma, decreased fluid; ½ mo f/u CT continued improvement of splenic hematoma, no fluid; 1 mo f/u sharp LUQ pain, f/u CT stable Grade II splenic injury, HCT stable, + pleuritic pain

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
3457	Liver III		Asthma; 1 ½ y PTA ankle fx; 1y PTA MVC ankle contusion	Hemopneumothorax symphysis diastasis with ant SI joint disruption R side	3d f/u CT some improvement liver; 5d f/u CT liver lac unchanged, postop ileus, L iliac fx; ½ mo f/u doing well; 1 y urology f/u s/p fall with pelvic fx; 2y f/u MVC, lumbar and sacral/pelvic contusion
3482	Liver I		Tobacco	kidney lac R rib fx	
3531	Liver I			Right superior pubic ramus fracture Right pulmonary contusion	2y f/u readmission for excision of sebaceous cyst L groin; 3 y f/u readmission for eosinophilic pneumonia
3618	Spleen I	4d no fluid splenic heterogeneity noted CT; 10d no abd c/o	3y PTA GSW R arm; 2 y PTA s/p fall motorcycle strain L knee and ankle	left elbow contusion multiple abdominal and thorax contusions	2 ½ mo f/u for illy/agitated
3627	Spleen I Liver I		4 mo PTA pinning R hand	L pneumothorax small	+hemoptysis en route to hospital, but stable HCT in house; 8d f/u occas RUQ pain/L pleuritic pain, no abd c/o
3706	Liver I		8 y PTA R forearm fx; 4y PTA R femur fx, Salter II	Major lip lac	½ mo f/u doing well; 2 ½ mo f/u doing well, CT with near complete healing liver; 4 mo f/u CT resolved liver lac
3735	Liver I			L subtrochanteric hip fx scalp lac	No blood products requested by pt unless absolutely necessary, (Hct 16 at nadir); 2d f/u abd u/s no fluid, no defects; 4d f/u posterior liver hematoma; 5y f/u readmission for MVC, chest contusion, L hip avulsion fx
3749	Spleen I Liver I			anterior L 6th rib fx L pneumothorax transverse fx l1 - l3	13d f/u doing well

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
3751	Liver IV		1 ½ y PTA MVC R knee contusion		1 d f/u CT no change, + fluid, gallbladder with blood-bile level; 4 d f/u CT no change, pericholecystic fluid/blood; 6d f/u CT organizing liver lac; 9d f/u CT improved lac, bowel with unresolved subcapsular fluid
3853	Spleen II		Microcephaly, psychomotor retardation, foot deformity	small kidney hematoma open nose fracture	18d ENTf/u s/p nasal reduction; 20d f/u doing well; 2 mo f/u doing well; 2 ½ mo f/u CT interval healing, small irreg spleen tip, min fluid; ½ y ENT f/u
3880	Spleen III			lt femur fx lt ptx lt sacrum pubic symphysis fx pelvic fx	18d f/u doing well; 1mo f/u nl abd; 2 mo f/u doing well; ¾ y f/u twice for chronic low back pain
3910	Spleen I		16 y PTA stab wound R intraclavicular; ½ y PTA abd U/S for increased LFTs shows fatty liver; IVDA 2y PTA		9d f/u doing well; 5 y f/u RLE cellulitis, Hep B and C +, HIV neg (no tx for trauma)
3950	Spleen IV		S/p patent ductus arteriosus, mild pulm stenosis	L flank abrasions	
4029	Spleen II				
4030	Spleen II Liver I		Prev trauma, tobacco	L intercostal margin abrasion	
4075	Liver II		ETOH, tob, s/p assault 1d PTA lac L eyebrow	Occipital bone fx Right 10th rib fx	
4132	Liver I				
4329	Spleen II Liver I		R eye surg, hypothyroid	rib cage fx 3 rib on L side (7th, 8th, 9th) with ptx	½ mo f/u doing well

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
4353	Liver II		Migraines, aseptic meningitis 5y PTA	multiple facial lacs nasal fx abd hematoma	3 d f/u CT improved liver, hematobilia in gallbladder, decreased fluid; 9d f/u renal u/s normal; 1 mo f/u no abd c/o X 2
4372	Liver I		Hep B, cocaine, HTN, ETOH, 4 y PTA abd CT nl; 1 y PTA retroperitoneal U/S no defects, liver spleen homogenous	multiple abrasions concussion with LOC < 1 hour left mandibular ramus fx	¾ mo f/u s/p asssault to head; 4 mo f/u s/p assault to head; 1 y f/u s/p assault L flank; 4 ½ yr f/u abd pain; 5 y f/u prostate CA ; 5 ½ y f/u MI, nl stress thal; 5 ½ y f/u prostate CA XRT, Hep B liver cirrhosis, s/p interferon therapy; 5 ½ y f/u chest pain; 6 ½ f/u angina
4415	Spleen I Liver II		Crohns, MVC 11 y PTA L thumb fx	left eighth rib fracture forehead hematoma	5d f/u CT stable spleen lacs, slight change in liver lac; ½ mo f/u RLQ pain, L chest pain, numbness L ulnar fingers; 1 ½ mo f/u, doing well, CT with resolution of liver and spleen, R colon diverticulae
4430	Spleen I			left renal contusion left posterior 11, 12 rib fx	1d f/u CT small spleen lacs, no fluid; 9d f/u doing well, no abd c/o
4480	Liver III	Laparoscopic BTL 1y PTA	Tob, ETOH, endometriosis L pelvis, cocaine use	scalp lac	
4516	Liver I				
4538	Liver I				10 d f/u doing well, 1 mo f/u doing well, CT normal
4588	Spleen II			T12 compression fx L radius fx	5d f/u CT normal
4701	Liver I		Marijuana	Head contusion Abrasion L side	
202	Liver II		HTN	Ribs 5-10 laterally fx R side L shoulder humerus fx at surgical neck	1 ½ y f/u readmit ortho closed femur fx; 3 y f/u DI

Table 4 (cont.)

Pt #	Injury/ Grade	Ab ops hx	Med hx	Assoc injuries	F/u, readmissions
1030	Spleen I, Liver I		COPD/asthma on home O2, remote h/o TB	L occipital scalp lac	Died in house from asthma exacerbation
1520	Spleen III		Tobacco, ETOH, spina bifida occulta, hypertonic bladder	L pleural effusion Open book pelvic fx Rectocosto junction disruption L flail chest L pulm contusion	7d f/u repeat CT, L lateral abd wall hernia with incarcearted valve and herniated abd contents; 3 mo f/u doing well, nl BM; 4 mo f/u incisional hernia, no ulcer or esophageal narrowing; 2y f/u acute viral syndrome, vomiting with fever; 4 ½ y f/u s/p fall; 8y f/u ortho for ORIF pelvis; 8 ½ y f/u for flouroscopic facet injection
4027	Spleen II	Appendectomy	CVA, HTN	L prox humerus fx dislocation, 3 parts L tension pneumothorax L femur fx L tib-fib fx Complex pelvic fx Multirib fx with L PTX	10d f/u CT abd with paracolic fluid, splenic lac, liver hemangioma, likely CA in sigmoid colon; 1mo f/u RUQ U/S, fatty liver, gallbladder sludge, diffuse echogeneity; 1 ½ mo f/u CT, fluid around liver (post-op), no change in spleen, no liver defects; 2 mo f/u CT abd for signs and sx of obstruction - splenic lac slightly decreased in size, no evidence of obstruction; 1 ½ y f/u doing well

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