Conservative Management of Ruptured Spleen

B. SHANDLING

SUMMARY

For many years it was accepted that splenic injury was best managed by splenectomy. However, it has more recently been realized that patients whose spleen has been removed are more liable to life-threatening infection. This is of particular consequence if splenectomy is performed in childhood, with a long life expectancy. The extra mortality rate associated with sepsis after splenectomy is noted from reports in the literature, and the immunological functions of the spleen are discussed. It is pointed out that with the present diagnostic means splenic rupture or splenic injury need not necessarily indicate splenectomy. The features of conservative management are outlined, and the results in 75 patients, of whom only 21 underwent splenectomy, are stated.


Until the turn of the century, splenic injuries were usually managed without operation, generally with disastrous results. The mortality was close to 100%, and it was this figure that led to the widespread acceptance of splenectomy which dramatically reduced mortality.

Not only is the operation of splenectomy fairly straightforward but it is the sort of operation most surgeons enjoy doing. The anatomy is not difficult to memorize, the organ is easy to identify, and its removal, provided the incision is of adequate size, is really quite easy. Indeed, the larger the spleen the easier the splenectomy. True, it is possible to damage the tail of the pancreas, the greater curvature of the stomach, or even the diaphragm, but to do this one has to be quite clumsy and ignorant and most surgeons are very unlikely to fulfill these criteria. Thus, up to the present, every textbook of surgery states that unquestionably there is only one treatment for rupture of the spleen, namely splenectomy. So widely accepted is this thesis that some authorities advise that, once the diagnosis has been made or is highly suspected, laparotomy is indicated.

Paediatric surgeons have a particularly awesome obligation to ensure that even remote complications of any treatment are avoided. In paediatric surgery one is not looking for a few salvaged years but for a 60-80-year survival. It was, therefore, appropriate that paediatric surgeons gave the impetus to the present interest and research into the long-term effects of splenectomy.

The anatomy of the organ and its peculiar vulnerability in abdominothoracic trauma are well known, situated as it is under the lower three ribs on the left. The fact that catastrophic haemorrhage may sometimes follow a laceration is universally appreciated. In sharp contrast to the certainty of our knowledge of splenic anatomy is the uncertainty of our knowledge of splenic physiology. The ancients regarded the spleen as determining the mood or humour of the person in which it resided. If an animal's kidneys are excised the animal eventually dies of renal failure. In contrast, the success of splenectomy in dealing with certain clinical problems and the lack of any clearly unfavourable effects of splenectomy have given rise to the idea that the spleen is not physiologically essential. This idea has gained widespread acceptance. Conversely, failure to remove the spleen in cases of trauma has been reported to cause secondary complications of delayed, traumatic splenic cyst formation, splenosis, and delayed rupture leading to death. The possibility that splenic injury might be resolved by haemostasis, clot resorption, and repair of disrupted tissue has generally been considered unlikely and potentially disastrous.

THE CASE FOR CONSERVATISM

‘It is an observation of great antiquity that the operation of splenectomy was not followed by death. Indeed, one may live for years without suffering any apparent ill effects from the absence of the organ; but this does not settle the problem as to whether or not a splenectomized person can weather a critical illness’, wrote Morris and Bulloch in 1919. These authors, who must be long dead, stated that removal of the spleen would very likely result in increased susceptibility to infection. They based their prediction on a series of experiments done on rats. Their gloomy prognostication lay ignored for over 30 years, until 1952, when King and Shumacher reported fulminant sepsis in infants who had had the spleen removed as part of the treatment of congenital spherocytic anaemia. This precipitated a spate of papers on the subject of overwhelming post-splenectomy infection (OPS). It was thought originally that late deaths as a result of sepsis after splenectomy did not occur, but this is not correct for there are increasing reports of such fatalities. The literature for 1976 alone contains 7 reports documenting overwhelming sepsis after previous splenectomy. The unfortunate aspect of these reports is that they appear mostly in haematological, paediatric, and other non-surgical journals. Singer provided significant insight into the entire syndrome spectrum. He collected data on over 3000 splenectomies and found a morbidity from sepsis of 4.25% and a mortality of 2.52%. Krivit voices the view that the incidence of OPSI after splenectomy for trauma is 50 times greater than that which occurs in the general population, while Singer, in his extensive review, puts this figure even higher at 58 times. More-
over, this figure rises to 86 if the operation has been performed as an incidental to another surgical procedure. In patients with idiopathic thrombocytopenic purpura the figure is 70 - 140 times. Splenectomy for congenital spherocytosis carries a mortality due to sepsis 200 times as great as that which can be expected in the population at large; with acquired haemolytic anaemia the figure is 300 times, in portal hypertension 600 times, with reticulo-endothelial cell abnormalities 300 times, and in thalassaemia 22 times. There is also a definite influence of age on post-splenectomy sepsis; there is little doubt that the younger the patient the more serious the likelihood and the greater the incidence of post-splenectomy sepsis.31

**SPLENIC FUNCTION AND SEPSIS**

What then are the functions of the spleen? This organ accounts for one-quarter of the total mass of lymphoid tissue in the body. It is a major source of antibodies, but its removal has little effect on the net potential for antibody production and there is no serious alteration in most of the immunological reactions. However, in one important respect splenectomy results in immunological impairment — in the initial response to particulate bloodborne antigens. Splenectomized individuals do form antibody to subcutaneously injected antigens, but when particulate matter is injected intravenously for the first time they form little or no antibody. If a normal rat is injected with sheep erythrocytes, either intravenously or subcutaneously, it responds by the rapid development of a satisfactory level of antibody titre. If a rat is splenectomized and then injected subcutaneously, there is again a good antibody response. However, if the splenectomized animal is injected intravenously with sheep erythrocytes it responds with a very low level of circulating antibody titre. Rowley30 found that when the same antigen was given intravenously to 14 patients who had had a splenectomy 8 days - 4 years previously, there were 12 patients who failed to respond with a significant haemolysin titre.

The spleen serves an important phagocytic function because of its peculiar anatomical advantage and its special type of circulation. Particulate matter, colloids and specifically bacteria are cleared from the circulation by the spleen. The well-recoginized splenic response to infection of acute enlargement is due to vascular engorgement. The splenic anatomy is well designed to be effective in trapping bacteria. The spleen, with a blood flow of 350 litres per day, and with its fine reticular network, allows for maximum contact between antigens in the blood stream and lymphoreticular cells involved in phagocytosis. If there is no spleen present, then particulate matter cannot be cleared in the sinusoids by being brought into contact with the tissue macrophages.

There is another reason why splenectomized patients are prone to infection, namely because of the loss or the depression of phagocytic function, which itself may be due to alterations in the opsonin-complement system, depression of the serum immunoglobulin levels, and suppression of lymphocyte responsiveness. The body's immune defence against encapsulated organisms such as *Streptococcus pneumoniae* is dependent on adequate levels of specific opsonizing antibody. This may explain the increased incidence of serious infection in splenectomized patients with particularly well-encapsulated organisms such as *Strep. pneumoniae* or *Haemophilus influenzae*. This incidence is especially high in children and more so in children under the age of 2 years. At younger ages, when individual immune systems have had less 'experience' with strains of these bacteria before splenectomy, they are at increased risk of exposure to multiple strains for which they have no antibody. The older the individual before splenectomy, the greater his or her immune experience with multiple strains, with resultant antibody formation, and the smaller the risk of overwhelming sepsis.

It has been said that a major part of the problem is the increased rate of infection acquired in the post-operative period and related to exposure to hospital microbial flora and to medical devices used on patients (particularly respirators, tracheostomy apparatus, heated aerosols, and intravenous and urinary catheters). Bacteraemia in these patients may even occur after discharge from hospital.31

**Bacteriology**

The bacteria most commonly incriminated35 in overwhelming post-splenectomy sepsis are the pneumococcus, *H. influenzae*, and *N. meningococcus*. Less commonly encountered are *Escherichia coli*, *Streptococcus*, *Staphylococcus* and *Salmonella typhi*. It is important to remember that the duplication time of a colony of pneumococci is 20 - 30 minutes, so that the altered clearance of such a rapidly growing organism may lead to a dangerously high concentration in the blood stream in a very short period of time.

**Clinical Features**

The clinical presentation of overwhelming sepsis is usually horribly dramatic and relentlessly downhill, often culminating in death within a few hours. Such a patient may develop a fever, vomiting, rigors, hyperpyrexia, difficulty in breathing, cyanosis, and an almost unrecordable blood pressure. Disseminated intravascular coagulation and, terminally, bilateral adrenal haemorrhages, complete the picture. Death usually occurs between 12 and 24 hours after the onset of the clinical syndrome. It is a frightening and catastrophic illness. More patients are being reported with the development of this dreaded complication. In a recent report an infection was recorded 25 years after a splenectomy for trauma. We surgeons may be long departed while our patients continue living under a veritable sword of Damocles.

**MANAGEMENT OF RUPTURED SPLEEN**

When operating for a ruptured spleen it has surely been the experience of most surgeons that often, by the time the spleen is exposed at operation, all active bleeding has ceased.36 Bleeding recurs while mobilizing the spleen preparatory to removing it. Splenic injury is therefore not
synonymous with persistent, uncontrolled intraperitoneal haemorrhage. Just as a kidney stops bleeding, so does a spleen.

At the Hospital for Sick Children, Toronto, a former chief surgeon, Dr Wansbrough in 1940 propounded the concept that splenic rupture or injury was not necessarily an absolute indication for splenectomy, but in those days there were no satisfactory means of incontrovertibly documenting rupture other than at operation. Today we have splenic scintiscans, even arteriography. Scanning is accurate, and the method is rapid and relatively non-invasive.

**Conservative Treatment**

Immediately upon conclusion of the assessment of a patient with a ruptured spleen an intravenous infusion with a large-bore needle is started in the arm, and a nasogastric suction tube is inserted. The patient is closely monitored in an intensive care unit, and if splenic damage is suspected a radio-isotope scan of the spleen is obtained. Any existing injury to the organ will be demonstrable as a laceration or as a filling defect representing a haematoma. We do not routinely perform abdominal paracentesis, the reason being that if blood is aspirated the surgeon may feel committed to a splenectomy. This manoeuvre is reserved only for the anaesthetized or unconscious patient. If necessary, a blood transfusion is given. In the great majority of children there is a favourable response within hours, and within 24 hours there is an improvement and diminution of all physical signs. After several days of close observation the patient may safely be moved out of the intensive care unit to a surgical ward. Intravenous fluids are not discontinued and the nasogastric tube is not removed until gastrointestinal function has returned to normal and the abdomen is soft and non-tender. Patients are kept on strict bed rest for at least 2 weeks in hospital, with follow-up scans at regular intervals until either the spleen has reverted to normal or no further improvement is visible on the scan. By 60 days after the injury most scans have either returned to normal or the scarring that will remain has reached its final stages.

If, when the patient is first seen, there is sustained hypovolaemic shock, severe and progressive anaemia, an increasingly distended abdomen, and left upper quadrant and/or left shoulder tip pain, a ruptured spleen is very likely, and if the patient’s condition continues to deteriorate despite the administration of blood a laparotomy will be necessary. However, this is an exceptional chain of events. If, during observation in the intensive care unit, signs and symptoms of intra-abdominal haemorrhage develop under observation, then immediate operation will be required.

**Results**

Since 1972 we have had 75 patients with proven splenic injuries as demonstrated by scintiscan. Of these 21 underwent splenectomy. In many instances, especially during recent years, laparotomy was indicated for other reasons such as a suspected liver or bowel injury. There were 54 patients with splenic injuries, on whom no laparotomy was done. Many of these patients required blood transfusion, and in some cases the volume given was substantial. We have had no cases of delayed or secondary rupture or any patients who subsequently developed a splenic cyst or symptomatic abdominal splenosis. The degree of disruption as evidenced on the scintiscan does not determine the treatment to be adopted. Severely damaged spleens (as determined radiographically) have been managed without operation.

At follow-up most of the scans showed some residual minimal defect. Radionuclide scanning is accurate; the method is rapid, relatively non-invasive and quite atraumatic.

On reviewing the records of the group of 12 patients who had required a laparotomy and undergone splenectomy, it is noteworthy that 12 children had active bleeding at the time of the operation.

The group of patients not operated upon in spite of radiologically proven splenic injuries all had returned to full and normal activities, and follow-up scans revealed no late complications. One patient underwent laparotomy 3 weeks after injury because of a large lucent defect on scintiscan. At operation, this was found to be a well walled-off organizing haematoma. The spleen was not removed. In another patient the spleen presumably took 2 years to heal, since for 2 years there were serial changes as determined by scanning. Seven spleens healed completely, and all the others healed with a residual defect which became stationary by scan in 2 or 3 months.

It is interesting to note that in 1976, 1977, and 1978 all the children but 1 were treated non-operatively.

**CONCLUSION**

The risk of delayed rupture of a subcapsular haematoma is rare in children, but when it occurs the patient will develop the familiar severe signs and symptoms several days after the injury. We have only seen this happen in two instances, both within 1 week of injury. In such a situation nobody would question but that urgent laparotomy and possible splenectomy are indicated. There are increasing reports in the literature of conservative intraoperative management of a ruptured spleen. Partial splenectomy, the application of microfibrillar collagen, and repair of lacerated splenic tissue are increasingly being recommended in the literature. Of course, the spleen that is pulped and irreversibly smashed must be removed. Such patients should be warned about the possible development of overwhelming sepsis at any time in their future lives, and should be immunized with polyvalent antipneumococcal serum. The question of oral antibiotic prophylaxis is one which has not yet been settled. Antihaemophilus serum has been developed and will soon be released.

On the other hand, the spleen with a small capsular tear that is not bleeding should be left in place.

I wish to suggest to clinicians that a diagnosis of a ruptured spleen does not automatically signify a splenectomy. We must be conservative in our approach in the
literal sense of the word. As much splenic tissue as possible must be conserved; this policy will, I am sure, prevent late death in splenectomized individuals.

REFERENCES

Hepatic Injuries
A 1980 Overview
A. J. WALT

FOREWORD
It is a great honour to be invited to contribute to a 'Festschrift' for Professor Jannie Louw who has influenced the lives of so many patients and surgeons around the world in so beneficial a way. His exemplary attention to detail, the clarity of his teaching, the breadth of his knowledge and his surgical energy all serve as unattained models for the rest of us. For my part, I would like to acknowledge the great kindness, courtesy and encouragement he has given me over the 35 years I have known him. In the USA, to say that one has had part of one's training under Jannie Louw is to be rewarded immediately with a nod of approbation and some envy. So, like many others, I am deeply grateful to Professor Louw for first stimulating my interest in surgery and for subsequently helping to develop it further.

SUMMARY
The present-day management of hepatic injuries is reviewed in the light of experience with over 1,400 cases at Wayne State University. Mortality is now between 10% and 14%, although this is reduced to 5% if only the liver is injured. The disparity between degree of severity of trauma and severity of injury is noted, as is the trend toward a more conservative surgical approach recently. Over 90% of cases can now be managed with comparative ease and with less frequent use of drains and cannulas.


The liver is the largest of the solid intra-abdominal organs and the most frequently injured. Members of the surgical unit at Wayne State University in Detroit operated upon at least 1,404 patients with hepatic injury between 1961 and 1976. This unit continues to treat approximately 100 liver injuries annually. The nature of the injuries range widely from stab wounds through gunshot wounds to the crushing damage of blunt injury. Certain basic principles have become much better recognized and accepted over the past 5 years. Hepatic injuries provoke less fear today than they did in the past and are surgically treated in a much more flexible and conservative manner than before. Our mortality rate has reached a plateau between 10% and 14%. The views which follow are based on this experience.

GENERAL OVERVIEW
The mortality from hepatic injuries remains between 8% and 20% in the USA. The specific institutional figure