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THE SCUDDER ORATION ON TRAUMA

" Acute Injuries of the Liver"
by

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Charles Locke Scudder, whose memory we honor by this annual oration on trauma, died at Boston in 1949 after more than six decades of dedication to surgery and the teaching of surgeons. His interests ranged over a variety of problems, including not only general surgery but also gynecology, neurosurgery, urology and orthopedics. The surgery of trauma, however, especially that relating to skeletal injuries, appears to have been the the file closest to his heart. In 1917 he organized the first fracture service in this country at the Massachusetts General Hospital, the institution with which he was closely associated throughout his professional life. His textbook on fractures, first published in 1900, went through eleven editions and served many generations of surgeons-- including my father (the first edition) and myself, in a later edition. He is said to have contributed more than any other man to postgraduate education in fractures and dislocations during the first half of this century. He was chairman of the American College of Surgeons Committee on Trauma for several years. It was thus peculiarly fitting that he should deliver the first Fracture Oration at the Clinical Congress in Chicago in 1929.

The list of orators chosen to deliver the 19 Orations on Fractures is indeed impressive including as it does most of the leading figures in the field of surgery for several decades.

The name was changed to the Oration on Trauma in 1952 to conform to what Estes referred to as the "change in general outlook and breadth of prespective" which had convert the Committee on Fractures to the Committee on Trauma.. Again, the choice of the first orator in the new series was particularly fitting. Sumner Koch spoke on The Working Man's Hand.

The eleven Orations on Truama are noteworthy not alone for the distinguished surgeons who delivered them but also for the greatly broadened scope of interest evinced by the titles included in this series.

In 1963 the name was again changed, this time to honor Scudder, the initiator of the otal series. The first in this new series was given by a surgeon interested not only in fractures, but also in the broader considerations of education and organization in the field of trauma. Edward French Cave that year spoke on Trauma, Specialism and the College.

The thirty-five orations in this series have presented to the Clinical Congress an outstanding group of surgeons, each of them a major contributor toward the objectives of tl Committee on Trauma and its parent, the American College of Surgeons. It is a high honor to be asked to give the thirty-sixth oration in this series and I am deeply grateful for tl privilege.

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Only two of the previous 35 orations have dealt with injury of the abdomen. William Estes, Jr., in 1953, considered Present day Problems in Non-Penetrating Abdominal Trauma, and Arnold Griswold, my predecessor in the chair of surgery at the University of Louisville spoke in 1959 on Wounds of the Abdomen and Pelvis. Estes, the first of these orators consider the abdomen, said in 1953 that his series showed an incidence of abdominal trauma to be only 1 in 1750 admissions, He concluded that " unless he has had war experience in the therapy of abdominal trauma, the individual surgeons is not likely to avoid the pitfalls that beset successful treatment of these lesions. But fifteen years later we can truthfully say that the increasing mechanization of our so-called civilization and the ever-increasing speed of our various means of transportation have combined to make the treatment of trauma a significant part of the daily obligations of every practicing surgeon.

Wounds of the liver have received increasing attention in recent years as improved resuscitative techniques and better transportation of the injured present us with more and more critically injured patients still amenable to salvage by definitive therapy. Better understanding of the principles involved in the surgical treatment of liver injuries has brought significant lowering of the mortality rate as attested to by many reports from a variety of several centers. Perhaps no area in the surgery of trauma has shown such marked improvement in the last quarter century. Yet, still far too many patients who might have been salvaged continue to die from severe injuries of the liver, Hence, it seems proper to add a third edition on abdominal trauma to those of Estes and Griswold, this one specially concerned with injuries of the liver.

Wounds of the liver have long posed serious problems in management. A large richly vascularized organ, its pulpy glandular tissue is enclosed by a capsule which is easily lacerated, torn or ruptured. Normally protected by the rib cage and the abdominal musculature, only deeply penetrating wounds or blunt force of considerable degree will ordinarily produce serious injury. The severity of the causative trauma and its frequent association with injuries of other organs, notably other abdominal viscera, presents the surgeon with a difficult diagnostic problem and a formidable reparative task required by a patient already the victim of serious blood loss and shock. Nowhere are the words of Hippocrates more appropriate: " Life is short and the art long; the time is urgent; experiment is dangerous and decision is difficult. "

Mortality rates are difficult to evaluate since there is such a variety of injuries and because of the frequent association with other critical injuries, of themselves so often lethal. From Hippocrates to the close of the last century most authors, including Larrey, the famous military surgeon, considered injuries to the liver lethal. Prior to the first World War most reports ranged from 80 - 90 % in mortality. The World War I figure was said to be 66.3%. Between the wars several series were reported, most with death rates in excess of 60%. The most noteworthy improvement came with the contribution of the Second Auxiliary Surgical Group in World War II when Madding and his associates achieved a noteworthy mortality of but 27%, largely through abandonment of gauze packing and the substitution therefore of adequate surgical drainage. During the Korean War the mortality again fell, this time to 14%, as a result of proper local therapy, but even more because of greatly improved techniques of resuscitation and better supportive patient care. The current mortality is reported to range from 10 to 20%. More widespread appreciation of the basic principles of patient care and awareness of the best techniques for specific types of injury should be expected to hold future mortality to less than 10%.

The factors responsible for the recent reduction in mortality are without doubt (1) the abandonment of gauze packing, (2) recognition of the paramount importance of drainage, and (3) appreciation of the need for resectional debridement or lobar resection

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in the cases associated with extensive destruction of liver tissue. These, together with more effective care for the systemic needs of the patients have brought about the current reduction in death rate.

Hemorrhage, the leakage of bile and infection are the major factors in the high mortality. Gauze packing has been the traditional method for control of hemorrhage, and most writers up to the time of World War II described this as the primary approach to the problem. Credit for correction of this erroneous approach must go to Madding and his associates who pointed out that gauze packing not only fails to drain the liver, but, in fact, obstructs drainage and predisposes to the accumulation of blood, bile and the products of tissue degeneration. While it may provide immediate control of hemorrhage, recurrence upon removal of the packs has been a common occurrence, not infrequently fatal. In the liver, as everywhere else in the body, there is no good substitute for exposure and ligation of bleeding vessels.

The use of absorbable agents to control venous ooze has been proposed. Absorbably gelatin sponge (Gelfoam) and oxidized cellulose (Oxycel) have been rather widely used in recent years. Madding has called attention to their tendency to retard healing because of delayed absorption, and has cited instances of reported serious complications following their use. He concludes that "bleeding or bile drainage, if significant, should be controlled by individual ligation of the vessels."

Infection has been one of the principal causes of death. It often relates to the frequent association with other intraperitoneal wounds, particularly of the gastrointestinal tract. Meticulous attention to primary closure of all such wounds, good debridement and drainage where needed will do much to minimize his hazard. The advent of antibiotics has provided us with another valuable ally in the post-operative management of these patients.

Drains, to be effective, must be properly placed and of a suitable type. The familiar Penrose drain is often an effective mechanism, but its tendency to kink and lose its effectiveness are familiar to all. To avoid these possibilities and to ensure continuously effective drainage, there is nothing so effective as the now familiar "sump" drain whose air vent insures continuing potency. A variety of these have been devised and several are commercially available. They can be easily improvised by the insertion of a small plastic tube to serve as a vent by passing it through a larger rubber drainage tube. Madding made the ingenious suggestion that a sump can readily be obtained simply by cutting off the balloon from a Foley catheter. Waterman, in our research department, has devised a particularly effective mechanism for pancreatic drainage by placing a sump within a fenestrated Penrose tube. We have found this very effective in drainage of the liver. Whatever type of drain is selected it is important to provide adequate wounds of exit for the drains, preferably brought out through an incision independent of the operative incision. A length of 1½ inches, or a size admitting two fingers are good rough guides to proper size of the drainage site. Wherever possible they should be in dependent position. Especially useful after resection of the right lobe of the liver are drains brought out posteriorly through the bed of the resected twelfth rib.

Merendino and others have called attention to the value of surgical biliary decompression in the management of liver trauma. Severance of biliary ducts being a part of every severe injury of this organ, common bile duct drainage

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is as logical here as in elective extrahepatic biliary surgery. Such drainage should be an essential part of all operative therapy for severe damage of the liver.

Hemobilia, the collection of blood within a cavity deep in the substance of the liver followed by intermittent discharge through the biliary system, is almost invariably an indication of improper or inadequate handling of the primary injury. Drainage to the depths of all deeply penetrating wounds is essential to prevent this complication. Failure to resect a portion of the liver with extensive deep damage may also result in hemobilia and other grave consequences to be described later.

More accurate knowledge of the detailed anatomy of the liver, especially with respect to its blood supply, has made possible the development of current resectional techniques. Dissections, together with material prepared by injection and corrosion techniques have demonstrated a lobar and lobular distribution of the hepatic vasculature which facilitates segmental resection not unlike that practiced with the lung. We now know that the right and left lobes are separated not by the line of the falciform ligament as was formerly believed, but rather by a line extending from the gall bladder fossa to the inferior vena cava. The blood supply, paralleled by the biliary ductal system, thus divides into right and left lobes, with subdivisions equally well demarcated. Lines of division for major resections are clearly indicated by the vascular distribution, facilitating not only lobectomies but segmental resections as well. A number of common anomalies have been well described, and the surgeon dealing with liver surgery would do well to familiarize himself with these and always to practice careful identification of each major vascular element as he proceeds with his resection.

Much has been written of the diagnosis of liver injuries and there are many excellent accounts of the various diagnostic criteria. Penetrating wounds, by their very presence, indicate exploration. The once traditional probing is purposeless at best, and deceiving at worst. Nor do I confess to any enthusiasm for injection of barium or other such time-consuming attempts to demonstrate peritoneal penetration. The latter can easily be established by a small incision at no risk if the peritoneum is intact and capable of enlargement for further exploration if it has been entered. "Negative explorations" are associated with minimal morbidity and mortality.

Blunt trauma offers a far more difficult problem. Signs of peritoneal irritation are indicative of intraperitoneal injury if present, but we have all seen severe damage in the abdomen with minimal physical signs. Shock, or evidence of severe hemorrhage, especially if not responsive to blood replacement, points strongly to the need for exploration. The presence of multiple injuries adds to the difficulty of diagnosis and it is here that experience with trauma will prove most helpful. Shock must be explainable; and it should be remembered that cranial trauma alone rarely causes shock. Evidence of severe trauma which may have included the abdomen coupled with a high index of suspicion will often be sufficient indication for exploration. We have small enthusiasm for peritoneal taps or any of their variations for a negative tap is of no significance whatever. It is freely admitted, however, that aspiration of blood or blood and bile is, of itself, adequate indication for exploration.

The need for resuscitative measures before exploration is now well understood. It must be borne in mind that failure to respond to blood replacement may, of itself, be an indication of severe continuing hemorrhage most often from spleen or liver. In that case, one must explore while continuing blood replacement. The incision should be adequate and so placed as to permit extension to a thoracoabdominal approach which is usually necessary for hepatic resection. Upon finding a massive

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hemoperitonium, the blood should be aspirated and all bits of fat, liver tissue or fragments should be removed at once (should blood be in critically short supply the possibility of autotransfusion should be borne in mind--though it is less desirable if there be significant admixture of bile). Hemorrhage from the liver can be temporarily controlled by fingers or rubber clamp temporarily applied to the hepatic vessels in the right free border of the gastrohepatic omentum. Further control, temporary only, can sometime be obtained by gauze pressure while the definitive procedure is decided upon.

Vascular ligation without accompanying resection occasionally deserves consideration when the situation warrants. (Mays). Ligation of the right hepatic artery has been one of the most feared hazards of biliary tract surgery, for fatal necrosis of the right lobe of the liver was thought to be the inevitable consequence. Graham and Cannell reported a 50% mortality in man and invariable death in dogs after right hepatic artery ligation. Further experimental work by Markowitz and associates indicated that this was due to the gas forming anaerobes normally inhabiting the liver of carnivores, and showed that antibiotic protection brought tolerance to hepatic ligation in dogs. Many untoward results have been reported after involuntary ligation in man, but usually in the presence of cirrhosis or other disease of the liver. Survival, when it occurred, was thought to be due to collateral circulation and thus more likely to develop if the ligation was proximal to the origin of the right gastric and gastroduodenal arteries. The work of Tygstrup and associates, however, indicate that increased extraction of oxygen from the portal flow is the major compensatory mechanism, of far greater importance than collateral circulation. Mays has reported survival following ligation of the right hepatic artery at the liver after its severance by a stab wound. Of even greater interest is the experience of McKenzie who ligated the right hepatic artery, portal vein and right hepatic bile duct in a 7 year old child, preparatory to lobectomy for trauma. Patient deterioration forced termination of the operation, but the child did so well postoperatively that the parents would not consent to reoperation for completion of the lobectomy. The child made an excellent recovery. Six months later he was operated upon for intestinal obstruction at which time the liver was inspected and the right lobe, shrunken to a size slightly less than the left, appeared otherwise normal. In April of 1968, nearly two years after the injury, the boy was "very fit physically, intellectually, and emotionally." We have no explanation for this remarkable recovery; for the present we shall continue to feel that such ligation can safely be done only as the first step in a lobectomy which carries lesser risk than retention of a lobe so completely devascularized. However, it seems likely that more patients may have survived inadvertant ligation of the right hepatic artery than has been supposed. There is now good evidence that this can be done with relative safety in an otherwise healthy liver, and one should remember this possibility when dealing with an injury such as the one reported. Mays has provided an excellent outline of patient management after such ligation.

Small surface lacerations will usually have ceased bleeding by the time they are visualized, and often require no suture. Deeper lacerations may require suture ligation of individual vessels, and some are amenable to a variety of suture techniques which have been described. The importance of drainage has already been emphasized and it must never be omitted when lacerations of the liver have occurred. Madding has aptly stated that "Rarely is a liver wound too small to require drainage." He adds, "It is true that in some cases there will not be postoperative drainage, but there are no reliable criteria by which such cases can be selected preoperatively or at operation."

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Bursting injuries of the liver present the most difficult of all problems. They result from the application of sudden severe force to the liver, a fluid filled sinusoidal encapsulated mass. The force is transmitted in all directions with explosive violence, causing bursting which severely damages the internal architecture of the organ and manifests itself on the surface by stellate or linear crevices. These injuries are characteristically produced by blunt trauma, but a high velocity missile traversing the thick anteroposterior diameter can also contribute sufficient internal energy to the fluid filled mass to produce the same type of explosive effect. Intraparenchymal hemorrhage from needle biopsy has recently been reported as responsible for such bursting injury in two cases.

These bursting injuries pose problems of a different type and of greater magnitude than most injuries to this organ. They characteristically produce profuse hemorrhage and massive hemoperitoneum, profound shock, pulpefaction of the liver parenchyma and extravasation of bile. Grave metabolic and hematologic deficiencies are the rule if the patient survives. This type of injury produced nearly 100% mortality until emergency resectional therapy was recognized as the only sure method of hemostasis. It has the further great advantage of removing the damaged tissue.

Mays, in the Price Institute of Surgical Research, has given excellent illustrations of the effect of blunt trauma upon the liver. Previous cadaver specimens have been seen not to respond with the type of injury seen life. (?) The liver, in life, is a sinusoidal fluid-filled organ perfused by both the hepatic artery and portal vein. Livers injected with saline in the hepatic and portal veins and barium in the biliary tree, when dropped from measured heights, after the method of Glenn and associates, developed injuries comparable to the bursting seen in blunt trauma in life. Lesser degrees of trauma (27-34 ft. lb.) produced tears and lacerations in the capsule but no internal damage. Moderate trauma (106-134 ft. lb.) produced crevassing of the external surface but only occasional injury to the vascular and ductal systems. 285-360 ft. lb. caused pulpefaction with disruption of the veins, arteries and bile ducts similar to those seen in the typical bursting injury. These findings are corroborative evidence of irreparable damage to the liver and give added weight to the argument for resection in these severest of injuries. Reference has already been made to the deleterious effect shown by Atik in our laboratories illustrating the deleterious effect of retention of devitalized or devascularized liver tissue upon the experimental animal. Mortality from bursting injuries in the University of Louisville Hospitals was 100% in the last 20 such cases encountered prior to adoption of resection. Segmental or lobar resection has now been carried out in ___ cases with survival of ___. There have been numerous reports from other centers showing greatly increased survival achieved by resection in these critical situations. The approach is so logical that it is surprising that it took so long for resection to receive its proper place in therapy-- though it was actually carried out by Bruns nearly 100 years ago! It is indeed strange that it has taken so long for us to recognize that debridement - the excision of dead and devitalized tissue - long recognized as essential in other areas, is fully as important in the liver.

Recent experiences with resection in the University of Louisville Hospitals illustrates the need for primary resection of badly damaged liver tissue.

A 42 year old man was pinned between two pieces of heavy machinery and was admitted in shock. Exploration revealed a massive hemoperitoneum and a burst injury of the right lobe of the liver. Hepatectomy was carried out, the specimen removed weighing 1170 grams. The sections are characteristic of the extensive parenchymal changes already present.

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Upper left: Fatty Degeneration. Intracytoplasmic fat vacuoles are small and medium sized. The liver cells appear swollen and the sinusoids correspondingly reduced in width (X430).

Lower left: Fresh microthrombi are present in branches of the portal vein. Platelets and fibrin can be distinguished (X430).

Upper right: The liver cells show acute degenerative changes and some of them have been infiltrated by polymorphonuclear leukocytes (X430).

Bottom right: Acute hemorrhagic changes with disruption of liver cords. The liver cell nuclei are pyknotic. Platelet clumping is present in this microlaceration (X430).

This patient made an excellent recovery but died of an embolus suddenly three weeks after operation and just before planned discharge. Autopsy revealed that there was already striking evidence of liver regeneration, with a total liver weight of 1600 grams.

Another patient illustrates even more dramatically the deleterious effect of retained devitalized liver tissue and the beneficial effect of its removal.

A 31 year old man was admitted in shock after an automobile accident. Exploration revealed a massive stellate laceration of the right lobe of the liver which the surgeon elected to repair with sutures. Hemorrhage was thus adequately controlled, and the patient did well for a few days. He then became noticeably worse. There was a septic type of fever with leucocytosis. A biliary pleural effusion was drained by tube insertion. Prothrombin time and serum albumen dropped and the serum bilirubin rose. Blood culture yielded a hemolytic staphylococcus, coagulase negative. He was re-explored on the 24th postoperative day. Sutures in the liver were intact, but there was a large fluctuant area beneath them, with congestion and enlargement of the entire right lobe. A right hepatic lobectomy was carried out after ligation of its vascular supply. The resected specimen revealed a large necrotic cavity in the liver parenchyma, with necrosis, degeneration and inflammatory reaction in the adjacent liver tissue. The patient's convalescence was associated with two episodes of septic shock which responded to fluid and colloid replacement together with massive antibiotic and steroid therapy. Thereafter his recovery was uneventful and he was discharged in good condition on the 19th postoperative day after the second operation. Periodic liver scans subsequently reveal gratifying regeneration of the right lobe.

The illustration shows the initial injury and the primary repair inadvisedly carried out at the first operation. The dotted area shows the large area of softening palpated beneath the suture line. In the center is a cross section of the excised right lobe showing the necrotic cavity. The sections show, above, totally necrotic pigmented liver tissue from the border of the cavity. Below is a section somewhat further removed which shows some fatty degeneration. Large intracytoplasmic fat vacuoles were found mainly in the periphery of the liver lobules.

Also illustrated are the metabolic responses to hepatic trauma. Between operations there was a steady rise in serum bilirubin, a fall of prothrombin time with deficiency of factors V, VII and X, and a drop in serum albumen. Following resection of the necrotic tissue the liver function returned to normal. Bromsul-

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phalein test was normal after subsidence of jaundice.

Serial liver scans two weeks after injury, six weeks after resection and six months and two years later graphically illustrate the regeneration of the liver.

This patient, perhaps more dramatically than most, shows the importance of prompt recognition and removal of dead and devitalized liver tissue following bursting injury. Had this been done initially, a smooth recovery might have been anticipated. Without the secondary resection he would almost surely have succumbed. With present anatomical knowledge and resectional techniques segmental or lobar resection can be carried out with relative safety.

The metabolic effects of massive liver resection must be reckoned with. Excision of large portions of liver substance will cause a drop in the peripheral blood sugar level which ordinarily requires maintenance by constant glucose infusion for at least 48 hours after operation. Serum albumen levels fall and may indicate a need for administration of plasma or human serum albumen. Other derangements of metabolism and the clotting mechanism may be anticipated: Decrease in platelets and fibrinogen concentration and depression of factors V, VII, and X together with lowered prothrombin levels. These often require fresh whole blood administration together with fibrinogen and fresh frozen plasma. Should there be evidence of fibrinolysis epsilon aminocaproic acid should be given. With careful operative technique and assiduous attention to postoperative needs, prompt recovery may be expected, followed by gratifyingly rapid regeneration in the area of resection.

The so-called Hepatorenal Syndrome deserves brief consideration. The renal dysfunction and anuria often observed in the presence of severe liver damage produced by disease or trauma has received considerable attention. For some time it was thought that some toxin released by such livers was responsible for the kidney damage. Opinions were divided as to whether this was produced by a toxin released by the damaged liver tissue or whether it might rather be an exotoxin produced by anaerobic bacteria in the devitalized tissue. Better understanding of renal failure has brought general abandonment of this theory. Francis Moore has well summarized the situation as follows:

"Patients with severe liver disease are prone to the development of hypotension; they therefore are prone to renal failure. In addition, pigment derived from breakdown of the porphyrin component of hemaglobin is nephrotoxic. Little wonder that the severely jaundiced patient readily goes into renal failure. When he does, we gain no additional knowledge by terming it the 'hepatorenal syndrome.' 'Bile nephrosis is equally specious as a term. The treatment of the patient rests on the guiding principles in any renal or hepatic failure, both factors being present."

Severe injuries of the liver can now be effectively managed by early diagnosis and prompt adequate treatment:

- 1) Aggressive resuscitative measures with blood replacement supplemented by lactated Ringers solution.
- 2) Thorough exploration with adequate exposure and effective vascular control to determine laceration type and extent of injury.
- 3) Individual ligation of bleeding vessels and of bile ducts as indicated.

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- 4) Repair when or as indicated, always with drainage, dependently placed and brought out through separate incision.
- 5) Resection or resectional debridement in the presence of severe damage with devitalized or nonviable tissue present.
- 6) Conduct drainage in all injuries involving damage to biliary ductal system.
- 7) Careful attention to the postoperative metabolic needs of the patient.

Proper application of these basic principles will yield not the former 80% to 90% mortality, but instead a survival of 80% to 90% of patients with severe liver injuries.

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