A DISSERTATION ON

"A STUDY OF CONSERVATIVE MANAGEMENT OF LIVER INJURY IN BLUNT ABDOMINAL TRAUMA"

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DECLARATION

I **Dr. A.LOKESHWARAN ,** solemnly declare that this dissertation entitled **"A STUDY OF CONSERVATIVE MANAGEMENT OF LIVER INJURY IN BLUNT ABDOMINAL TRAUMA ",** is a bonafide work done by me in the department of general surgery, Govt. Stanley Medical College and Hospital, Chennai under the supervision of **Prof. Dr. T. SIVAKUMAR M.S.,**This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the university regulations for the award of M.S,Degree (General Surgery), Branch – I Examination to be held in May 2020.

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ABBREVIATIONS USED

ARDS – Acute respiratory distress syndrome

A –V – Arterio venous

BIA – Blunt injury abdomen

CBD - Common bile duct

CECT – Contrast enhanced computed tomography

CHD - Common hepatic duct

CPR - Cardiopulmonary resuscitation

CUSA - Cavitron ultrasonic surgical aspirator

CVP - Central venous pressure

DPL - Diagnostic peritoneal lavage

ERCP - Endoscopic retrograde cholangiopancreatography

FAST – Focused assessment by sonography for trauma

FQA – Four quadrant aspiration

GCS - Glasgow coma scale

ICD – Intercostal drainage

ICU - Intensive care unit

IVU – Intravenous Urography

KUB - Kidney, ureter, bladder x ray film

MRI - Magnetic resonance imaging

RTA – Road traffic accident USG – Ultrasonography

TABLE OF CONTENTS

INTRODUCTION

The liver is the most commonly injured intraabdominal organ with an incidence of 30% to 40%. The overwhelming majority of liver injuries, however, are minor, with spontaneous cessation of hemorrhage almost always the rule, and operative intervention is rarely required. On the other hand, complex hepatic injuries continue to challenge even the most experienced trauma surgeons. Hepatic injuries have been a fascinating topic since the publication of "Notes on the Arrest of Hepatic Hemorrhage Due to Trauma" in 1908 by J. Hogarth Pringle of the Glasgow Royal Infirmaries who provided the first published scientific foray into the management of severe hepatic trauma and describes one of the operative maneuvers that remains a mainstay in hepatic hemorrhage control to this day.

Perhaps the single greatest advance in the management of hepatic trauma over the past two decades has been advancement and remarkable success of on operative management of blunt hepatic injuries. Other advances include the combination of portal triad occlusion, finger-fracture technique (hepatotomy) and omental packing for complex hepatic injuries, and perihepatic packing with planned reexploration in trauma patients demonstrating signs of the "triad of death" (acidosis, coagulopathy, and hypothermia) as well as evolving transfusion strategies stressing 1:1:1 ratio of packed red blood cells (PRBCs), fresh frozen plasma (FFP), and platelets with the goal of prevention of intraoperative coagulopathy.

In the new millennium, a "multidisciplinary approach" concept

has evolved as the standard of care in the treatment of complex hepatic trauma. In addition to prompt surgical intervention, when indicated, adjunctive interventional techniques such as hepatic angiography, endoscopic retrograde cholangiopancreatography (ERCP), biliary stenting, and percutaneous computed tomography (CT)–guided drainage have become a part of the trauma surgeon's armamentarium.

AIM OF THE STUDY

1) To study the sex and age distribution of liver injury in blunt abdominal trauma.
2) To evaluate the morbidity and mortality due to severity of the injury.
3) To evaluate the various modes of injury causing the blunt abdominal trauma.
4) To evaluate the various diagnostic methods and techniques available in the management of blunt abdominal trauma.

REVIEW OF LITERATURE

Since from the historical times, blunt injury has been recognised as one of the cause for the abdominal injuries.

- Visceral injuries due to blunt trauma was first recorded by Aristotle .
- Distinct triage and surgical protocol was developed in Babylonia under the rule of Hammurabi.
- Blunt and penetrating injury to pancreas was first recorded by Trausse in 1827.
- Peritoneal lavage was first performed by Solomen.
- Diagnostic methods in the abdominal injuries were first introduced by Ainhum in 1934
- During assassination , the Chinese would cause blunt trauma resulting in the puncture of spleen leading to death.
- Voorhes described the synthetic grafts in 1952

The flushing of sterile solution through the peritoneal cavity to obtain peritoneal contents was first introduced by Root in 1965. Imaging techniques play an important role in the diagnosis of blunt injury abdomen.

ANATOMY OF ABDOMINAL CAVITY

Abdominal cavity is divided into nine regions by four imaginary planes, two horizontal and two vertical planes. Transpyloric plane of Addison passes

anteriorly through the tips of ninth costal cartilage and posteriorly through the body of L1 vertebra. Transtubercular plane passes through the tubercles of iliac crest and body of L5 vertebra. Right and left lateral planes correspond to the midclavicular lines and passes through the midinguinal point and tip of ninth costal cartilage. Sometimes subcotal plane is used instead of transpyloric plane, which passes through the 10th costal cartilage and body of L3. The different zones are right and left hypochondrium, epigastrium, right and left lumbar, umbilical, right and left iliac, hypogastrium. Additionally, external genitalia is considered as 10th quadrant and left supraclavicular region as 11th quadrant.

PERITONEAL CAVITY

The peritoneum is a large serous membrane lining the abdominal cavity and is in the form of a closed sac which is invaginated by a number of viscera. It is broadly divided into two parts- greater and lesser sacs, which communicate through the epiploic foramen or foramen of Winslow.

SOLID ORGANS: LIVER

EMBRYOLOGY

Developmental biologists have often marveled at the enormous regenerative capacity of the liver after injury, and such growth represents one of the fastest by mammalian tissues. Presumably, this process is based on the recapitulation of embryonic signals in the liver, but our understanding of the mechanisms is relatively poor. The liver is considered the largest internal organ and consists of

diverse cell types that arise from various embryologic origins. It is a vital organ that has an array of diverse functions, including endocrine, exocrine, and essential metabolic functions. The two principal cell types of the liver are the hepatocytes, which comprise nearly 70% of the mass of the adult organ and are responsible for the majority of the metabolic liver functions, and the cholangiocytes. Both cells are derived from the embryonic endoderm. Other cell types of the liver include hematopoietic, Kuppfer, stromal, and stellate cells, which are of mesodermal origin (Fig. 1.1). Despite their homogenous appearance, hepatocytes do not all function identically; they perform various tasks depending on their physical location within a hepatic lobule, the primary functional unit of the liver. For instance, periportal hepatocytes are responsible for the urea cycle enzymes, whereas pericentral hepatocytes express glutamine synthase and utilize ammonia to generate glutamine. Thus, liver development entails not only hepatocyte and cholangiocyte differentiation but also cellular differentiation within the hepatocyte population (see Fig. 1.1).

During the third week of gestation, liver primordium first appears as an outgrowth of the ventral foregut endoderm at the caudal end of the foregut. The proliferation of the epithelial cells in this liver bud leads to its outgrowth and branching into the surrounding mesenchyme, giving rise to the liver and intrahepatic biliary tree. As it grows caudally, traversing the septum transversum, the persistent connection between the branching epithelium and the foregut develops into the extrahepatic bile ducts and gallbladder (Sadler & Langman, 2006). The bipotential hepatoblasts eventually differentiate into hepatocytes and cholangiocytes. The final liver structure continues to develop through the postnatal period. In addition to giving rise to the

liver and biliary tract, the proximal endoderm (foregut) also gives rise to respiratory epithelium and the glandular cells of the thyroid, thymus, and pancreas due to its pluripotent nature (Fig. 1.2).

ENDODERMAL PATTERNING

Embryologically, the liver, biliary tract and pancreas develop through a series of reciprocal interactions between the endoderm and the surrounding mesenchyme. The primitive gut tube, which is derived from the endodermal germ layer during gastrulation, is divided into the foregut, midgut, and hindgut domains, each of which gives rise to specialized regions (Grapin-Botton, 2005). This specialization is initiated by transcription factors expressed in different regions. For example, the coexpression of pancreatic and duodenal homeobox gene 1 *(PDX1)* and pancreas-specific Transcription factor (PTF1A) in the endoderm gives rise to the pancreas.. The definitive endoderm is formed at the ventral side of the vertebrate embryo during gastrulation.

Evagination of the endoderm at the anterior end of the embryo generates the ventral foregut, which will eventually give rise to the liver,lung, thyroid, and the ventral pancreas. The dorsal region of the definitive endoderm develops into the intestines and the dorsal pancreas .

This complex dialogue between the endoderm and mesoderm appears to be critical for the final patterning of the gut tube, where several signaling pathways have been implicated in the regulation of foregut endoderm development, promoting organ specification along its anterior-posterior axis for organs such as the thyroid, lung,

liver, and pancreas. The plasticity of the endoderm was demonstrated by experimentally recombining the posterior mesoderm with early foregut endoderm, leading to repression of liver and pancreatic development in favor of intestinal development (Kumar et al, 2003; Wells & Melton, 2000).

Specific signals from the surrounding mesoderm in the foregut region lead to hepatic specification and subsequent morphogenesis (Gualdi et al, 1996). The molecular pathway linking endodermal patterning to the initiation of liver and pancreatic development has been partially elucidated by recent studies in the *Xenopus* model, supporting a role for FGF4 and WNT signaling from the posterior mesoderm in inhibiting foregut fate and promoting hindgut formation; these signals are inhibited in the anterior endoderm to allow foregut development (McLin et al, 2007).

The β-catenin signaling pathway is essential in liver and pancreas development. Specifically, repression of β-catenin (a downstream mediator of canonical WNT signaling) in endoderm is necessary to initiate reciprocal signaling from the mesoderm, leading to hepatic induction. This role for β-catenin is illustrated by the fact that forced β-catenin expression in the anterior endoderm leads to downregulation of the hematopoietically expressed homeobox gene *(HHEX)* and inhibition of liver formation, whereas forced repression of β-catenin in the posterior endoderm (future hindgut that normally expresses β-catenin) induced ectopicHHEX .

Conversely, inhibiting β-catenin in the posterior mesoderm led to ectopic expression of other liver and pancreas markers(FOR1, PDX1, elastase, and amylase) along with the inhibition of the intestinal marker ENDOCUT (McLin et al, 2007). Conversely, inhibiting the WNT receptor FZD7 resulted in loss of the liver primordium in *Xenopus* embryo. This result indicates that low levels of WNT signaling are necessary to maintain foregut fate, with hepatic specification a dynamic process.

Studies in mouse and chick models demonstrate the need for FGF2 signals from the cardiogenic mesoderm and bone morphogenic proteins (BMPs) from the septum transversum mesenchyme (Rossi et al, 2001; Zhang et al, 2004

Hepatic Competence

Hepatic competence, or the ability to form liver from the foregut endoderm, is considered the first of a two-step process for the specification of liver in vertebrates. Competence is a prerequisite for the endoderm to respond to specific signals, such as FGFs from the surrounding mesoderm, which then leads to the second step, the induction of liver-specific genes such as albumin *(ALB),* α-fetoprotein *(AFP),* and hepatocyte nuclear factor 4α *(HNF4α).* This "competence" is facilitated through the *FOXA* gene transcription factor family that includes FOXA1 and FOXA2 (forkhead box proteins A1 and A2) and the GATAbinding proteins 4 and 6. *FOXA* gene expression precedes the induction of the hepatic program by FGF signals in the endoderm, and FOXA2 binding reverses chromatin-mediated repression of *AFP* gene

transcription in vitro (Crowe et al,1999). Deletion of *FOXA1* and *FOXA2* led to the absence of liver bud formation with the loss of AFP expression in the ventral foregut, which indicates that hepatic specification had failed to initiate (Lee et al, 2005a). Similarly, deletion of either *Gata4* or *Gata6* in mouse embryos resulted in failed liver expansion, with the hepatic endoderm still expressing hepatic genes (Watt et al, 2007; Zhao et al, 2005). The forkhead box (FOX) proteins are an extensive family of transcription factors that share homology with the winged helix/fork head DNA-binding domains (Kaestner et al, 2000) that play important roles in regulating expression of genes involved in cellular differentiation, proliferation, transformation, and metabolic homeostasis (Duncan, 2000; Wang et al, 2001; Zaret, 1999). In vitro explant cultures demonstrated that FGF was sufficient to induce ventral foregut endodermal cells to differentiate into hepatoblasts (Gualdi et al, 1996). However, when FOXA1/FOXA2-deficient endoderm was cultured with exogenous FGF2, no liverexpression was seen (Lee et al, 2005a). This result indicates the necessity for FOXA1 and FOXA2 for hepatogenesis. In vivo DNA-binding studies revealed that the liver-specific *ALB* has an important upstream regulatory binding site for FOXA factors (Bossard & Zaret, 1998; Gualdi et al, 1996). Before FOXA or GATA4 binding, the *ALB* gene is transcriptionally silent, with a closed chromatin. After binding with FOXA and GATA4, the chromatin domain is thought to become exposed (Cirillo et al,2002), thus increasing the ability of the gene to be activated. By the E9.5 gestation age, other transcription factors CCAAT/ enhancer binding protein- β and nuclear factor 1 bind to sites adjacent to the

FOXA site, and as a result, the albumin gene becomes active (Zaret, 2002). Therefore, FOXA binding to chromatin is the critical step in hepatic competence by increasing gene expression and allowing binding of other transcription factors.

Hepatic Induction

FGF from the cardiogenic mesoderm and BMPs from the septum transversum mesenchyme (STM) have been implicated in the induction of liver fate in mouse and chick embryonic endoderm. In vitro studies demonstrated that when ventral foregut and cardiogenic mesoderm were cocultured in the presence of fibroblast growth factor inhibitors, liver induction was inhibited (Jung et al, 1999). Culturing foregut endoderm without the cardiogenic mesoderm in the presence of low concentrations of exogenous FGF2 (2 to 5 ng/mL) rescued hepatic gene expression (Jung et al, 1999) and simultaneously suppressed the expression of the pancreas program (PDX1)(Deutsch et al, 2001) whereas a high concentration of FGF2 (10 to 500 ng/mL) induced NKX2-1, an early marker for respiratory epithelium, but not albumin gene expression (Serls et al,2005). When foregut endoderm was cocultured with noggin (NOG), a BMP inhibitor, inhibited hepatic gene induction was observed, an effect that was reversed when BMP2 or BMP4 were added. Despite these results, however, embryos that were homozygous mutants for *BMP4* still exhibited normal hepatic gene induction (Rossi et al, 2001; Smith & Harland, 1992).

These data indicate that the cardiac mesoderm, a source for FGF signaling, is crucial for the hepatic induction from the ventral foregut endoderm and subsequent morphogenesis,whereas BMP aides in the process.

Morphogenesis of the Hepatic Bud

Following hepatic specification (E8.5 to E9.0), the "liver" starts to express liver-specific genes *(ALB, AFP, HNF4α),* and eventually form the liver bud. Hepatic bud morphogenesis is facilitated by two transcription factors, HHEX (hematopoietically expressed homeobox, discussed earlier) (Crompton et al, 1992) and PROX1 (prospero-related homeobox 1) (Oliver et al, 1993). HHEX is expressed in the anterior endoderm at E7.0, which eventually gives rise to the liver as well as the ventral pancreas. *HHEX*-null embryos grow without a liver or thyroid and develop forebrain defects at E11.5; however, evidence of endodermal epithelial thickening suggests a possible defect in differentiation as evidence of possible hepatic induction (Martinez Barbera et al, 2000).

In a separate study on *HHEX*-null embryos, liver genes *ALB* and *PROX1* were expressed in the ventral foregut endoderm at around E8.5, with the thickening of the hepatic endoderm region at E9.0 being smaller compared with heterozygote embryos. In addition, a significantly lower proliferation rate seen in the prospective hepatic domain when compared with the control group, demonstrated by bromodeoxyuridine (BrdU) staining, with no evidence apoptosis (Bort et al, 2004). The basal membrane layer, which is rich in laminin, surrounds the hepatic endoderm

and degrades around E9.0 to E9.5, so that the hepatocytes start migrating into the STM to form the liver bud. This degradation is facilitated by the hepatoblasts, which under normal conditions downregulate E-cadherin. However, in the *PROX1*-null mutant embryos, the progenitor liver cells fail to migrate into the STM as a result of excess E-cadherin and basement membrane proteins laminin and collagen 4. The basal lamina fails to degrade, and the cells remain trapped in the hepatic diverticulum, with an overall reduction in liver size (Sosa-Pineda et al, 2000). The bulk of the liver lobe lacks hepatocytes, suggesting that the mesenchymal component contributes most of the liver mass in *PROX1*-null mutant embryos. A similar phenotype is seen with *ONECUT1* (also called *HNF6*) and *ONECUT2* double mutants, which are required for basal lamina degradation (Margagliotti et al, 2007). Furthermore, a pharmacologic inhibition of matrix metalloproteinases (MMPs), extracellular matrix remodeling enzymes usually expressed by the hepatoblasts and STM cells, inhibit hepatoblast migration in culture (Margagliotti et al, 2008). To further illustrate the importance of the extracellular matrix in hepatic bud morphogenesis, hepatoblasts deficient in laminin receptor β 1-integrin fail to colonize the liver bud (Fassler & Meyer, 1995). These β 1-integrins are among those that act as receptors for extracellular matrix proteins, such as laminins and collagens (Hynes, 1992).

In summary, PROX1 and ONECUT factors are important in regulating delamination and in controlling hepatoblast migration through regulating MMPs and hepatoblast interactions with the extracellular matrix. Without an appropriate extracellular matrix, cell migration into the STM will be disrupted.

The role of endothelial cells in liver organogenesis has also been studied, with the liver vasculature providing a vital source for hematopoiesis in early life. Endothelial cells are positioned as a loose necklace of cells interceding between the thickening hepatic epithelium and the STM (Matsumoto et al, 2001). Embryos that were null mutants for *VEGFR2* (also known as *KDR*) had failure of delamination and subsequent migration by the hepatoblasts (Matsumoto et al, 2001). These results imply that the endothelial cells interact with nascent hepatic cells and aid in liver bud outgrowth.

Liver Bud Growth

The STM cells are closely related to the ventral endoderm and contribute to hepatic induction and growth. This epithelial mesenchymal interaction is essential for liver bud formation, expansion and differentiation. During this phase (E9.5 to E15), the liver bud undergoes a tremendous amount of growth and becomes an important site for hematopoiesis. Signals regulating this stage arise from both the hepatic mesenchyme and the STM. These signals include FGF and BMP, which promote liver growth in addition to aiding in hepatic specification. BMP4 is strongly expressed in the STM and continues to be expressed at E9.0, during which the liver bud migrates into the STM (Rossi et al, 2001). *BMP4*-null mutant mice embryos had a delay in the growth of the liver bud, which indicates that BMP constitutes an important growth signal for the liver (Rossi et al, 2001).

Another factor that has been implicated in liver growth is HLX, which is expressed prominently in the visceral mesenchyme (STM), into which the liver will expand (Hentsch et al,1996). *HLX*-null embryos exhibited severe liver hypoplasia without affecting liver specification (Hentsch et al, 1996). A similar finding was observed in embryos that were null mutants for hepatocyte growth factor (HGF) (Schmidt et al, 1995). In contrast to *HLX−/−* embryos, apoptosis was the underlying cause for the severe liver hypoplasia in *HGF*-null embryos. HGF is produced by the cells lining the sinusoids, which are of mesenchymal origin, mediating its effects through the c-MET tyrosine kinase receptor produced by hepatocytes (Schmidt et al, 1995). Transforming growth factor-β (TGF-β) signalling is also involved in mediating their signals through SMAD2 and SMAD3, which translocate to the nucleus to either upregulate or downregulate gene expression. Embryos that were heterozygous mutants for *SMAD2* and *SMAD3* exhibited severe liver hypoplasia as a result of a decrease in β1-integrin expression (Weinstein et al, 2001). Intriguingly, this phenotype was rescued when HGF, a potent hepatotrophic growth factor, was added to the culture medium. Presumably, this was a result of β1-integrin expression (Weinstein et al, 2001), which plays an important role in hepatocytic adhesion to the extracellular matrix; TGF-β and HGF are known to induce β1-integrin expression (Kagami et al, 1996; Kawakami-Kimura et al, 1997).

OVERVIEW OF HEPATOBLAST DIFFERENTIATION

Hepatoblasts begin to differentiate into mature hepatocytes and biliary epithelial cells at about E13.5. Before differentiation, the hepatoblasts express genes for adult hepatocytes (albumin, *HNF4α*), biliary epithelial cells *(KRT19)*, and fetal liver *(AFP)*(Lemaigre, 2003; Shiojiri et al, 1991).Hepatoblasts in proximity to the portal vein form a bilayer architecture and eventually differentiate into biliary epithelium cells by upregulating biliary specific cytokeratin-19 *(KRT19)* and downregulating the other hepatic genes. This bilayer around the portal vein begins to form focal dilatations incorporated into the portal mesenchyme to form intrahepatic biliary duct at E17.0 until birth. Areas of the ductal bilayer plate not involved in the formation of the ducts progressively regress.

The remaining hepatoblasts differentiate into mature hepatocytes, arranged in hepatic chords with the bile canaliculi on the apical surfaces (Lemaigre, 2003). In the mature hepatobiliary system, the bile is produced by the hepatocytes and is secreted into the canaliculi, which are connected to the network of intrahepatic biliary ducts. The bile then flows to the hepatic ducts, transits through the cystic duct, and is stored in the gallbladder; eventually, the bile is excreted into the bowel via the common bile duct. The biliary epithelial cells delineate the lumen of the intrahepatic, extrahepatic biliary tree (hepatic, cystic and common bile duct), and the gallbladder

Biliary Epithelial Cell Differentiation and Formation of the Ductal Plate

The exact origin of the biliary epithelial cells has been greatly debated; however, the popular school of thought is that they are derived from bipotential hepatoblasts that can differentiate into either hepatocytes or biliary epithelial cells. This theory is based on the observation that immature hepatoblasts coexpress markers of both hepatocytes (ALB) and biliary epithelial cells (KRT19). The biliary specific marker KRT19 becomes strongly expressed at a later gestational age, as the cells become ductal cells, whereas other cells transiently express the hepatocyte markers ALB and AFP as they develop into mature hepatocytes (Shiojiri et al, 1991). This theory was further supported when embryonic liver, before the intrahepatic biliary ducts form, was transplanted into the testis of syngeneic animals, giving rise to both hepatocytes and typical bile ducts (Shiojiri et al, 1991).

Suzuki and colleagues (Suzuki et al, 2002) used in vitro studies on hepatic "stem" cells from E13 embryonic livers, identified with self-renewing capability and multilineage differentiation potential, to demonstrate that these cells could form differentiated hepatocytes, biliary epithelial cells, pancreatic, and intestinal cells.

The first step in triggering the initiation of the transition from hepatoblast to a biliary epithelial cell is thought to be facilitated through the ONECUT transcription factor hepatocyte nuclear factor 6 (HNF6), which is expressed in the biliary epithelial cells of the developing intrahepatic biliary ducts and in hepatoblasts, gallbladder

primordium, and the extrahepatic bile ducts (Landry et al, 1997; Rausa et al, 1997). *HNF6−/−* embryos displayed severe biliary anomalies. Extrahepatically, this mutation resulted in the absence of gallbladder and the normal bile ducts; instead, there was an enlarged structure connecting the liver to the duodenum. Intrahepatically, however, it caused abnormal differentiation of biliary epithelial cells, resulting in cholestasis. Closer histologic examination revealed an increased number of KRT19-positive cells compared with control cells at E13.5, with the development of abnormal large cysts at E15.5 to E16.5 that contained an epithelium of KRT19-expressing cells. These abnormal cysts are similar to those seen in Caroli disease, an autosomal recessive disorder with ductal plate malformation and ectasias. The abnormal increase in KRT-positive cells at E13.5 lacked any proliferative marker, suggesting that they are postmitotic, resulting from hepatoblasts that have differentiated toward a biliary lineage prematurely (Clotman et al, 2002). In addition, the excess KRT-positive biliary epithelial cells formed cordlike extensions within the liver parenchyma, compared with the control group, which is restricted to the vicinity of the portal vein. This observation supports the role of HNF6 in controlling the differentiation of hepatoblasts into biliary epithelial cells and the morphogenesis of the intrahepatic biliary ducts, confining biliary epithelial cells to the periportal area. A similar morphologic defect of intrahepatic biliary ducts was observed in *HNF1β−/−* embryos, which suggests that *HNF6* controls intrahepatic biliary duct development via *HNF1β* (Clotman et al, 2002). In contrast to the intrahepatic ducts that are derived from bipotential hepatoblasts, the cholangiocytes that line the extrahepatic bile ducts are derived from the common ventral pancreatobiliary bud. The bile duct fate of

these primitive embryonic bud–derived cells is determined by the transcription factor SOX17, which is coexpressed with PDX1 in these pancreaticobiliary progenitor cells (Spence etal, 2009). The cell fate decision between pancreas-lineage PDX1-positive cells versus biliary primordium SOX17- positive cells is determined by hairy and enhancer of split 1 (HES1) (see later in the PANCREAS section). Deleting *SOX17* at E8.5 resulted in ectopic expression of pancreatic tissue in the common bile duct with PDX1-positive cells in the liver bud along with the loss of biliary structures. Conversely, overexpression of SOX17 suppressed pancreas development and promoted ectopic biliary-like tissue in the PDX1-positive domain tissue (Spence et al, 2009). Furthermore, it has been demonstrated that SOX17 regulates insulin secretion postnatally, with the mice becoming prone to developing diabetes with the deletion of *SOX17* gene in the pancreas (Jonatan et al, 2014). With regard to mesenchymal-epithelial induction of liver primordium and gallbladder, studies have suggested that the mesenchyme contributes to biliary epithelial cell differentiation, where differentiation of hepatoblasts into biliary epithelial cells was stimulated when cocultured with hepatic or lung mesenchyme (Shiojiri & Koike, 1997). It was also noted in the study of *HNF6−/−* mice that biliary epithelial cell differentiation occurred at the interface between the portal mesenchyme and the liver parenchyma (Clotman et al, 2002).

A recent study revealed that the forkhead box f1 (FOXF1) transcription factor may play an important role in the mesenchymal-epithelial signaling, an interface that is required for the development of organs derived from foregut endoderm such as the pancreas, liver, gallbladder and lung (Kalinichenko

et al, 2002). FOXF1 expression is restricted to the gallbladder mesenchyme and STM. In *FOXF1+/−*embryos, the gallbladder develops severe structural abnormalities with significant reduction in size with reduced mesenchymal cell numbers, an absent biliary epithelial cell layer, and a deficient external smooth muscle layer. The reduction in mesenchymal cell numbers was attributed to the reduction in vascular cell adhesion molecule and cell adhesion α5-integrin, both of which are essential for mesodermal formation (Mahlapuu et al, 2001; Yang et al, 1993). Defective smooth muscle layer formation was attributed to diminished levels of platelet-derived growth factor receptor α, which is essential for smooth muscle cell differentiation (Jacob et al, 1994). FOXF1 is not expressed in intrahepatic biliary duct mesenchyme in wild-type mice, and no defects were seen in the intrahepatic biliary ducts of *FOXF1+/−* mice; however, *FOXF1* mRNA levels in the liver were increased, suggesting that it may be a compensatory mechanism to prevent defects in the liver (Kalinichenko et al,2002). All these suggest that *FOXF1* is crucial for the development of the extrahepatic biliary duct and gallbladder with the mesenchyme playing an essential role in biliary epithelial cell differentiation. The interaction between the biliary epithelial cell and the extracellular matrix is also thought to contribute to biliary epithelial cell differentiation.

Integrins are membrane receptors for extracellular matrix proteins where they play an important role in mediating the interaction between differentiating cells and the extracellular matrix (Couvelard et al, 1998; Hynes, 1992). Hepatoblasts express integrin heterodimers containing the β1 subunit (α1β1, α5β1, α6β1, and α9β1), and when hepatoblasts differentiate into immature

intrahepatic biliary epithelial cells while in contact with the mesenchyme, the morphology of the integrins changes. The primitive intrahepatic biliary epithelial cells upregulate $\alpha 6 \beta 1$ expression and loses $\alpha 1 \beta 1$ while acquiring several other integrin dimmers not previously expressed on hepatoblasts such as $\alpha 2 \beta 1$, $\alpha 3 \beta 1$, $\alpha V \beta 1$, and $\alpha 6 \beta 4$ (Couvelard et al, 1998). Intrahepatic biliary epithelial cells are contacted by a basement membrane containing collagen, enactin, and laminin (Desmet, 1985); however, hepatocytes are surrounded by the perisinusoidal matrix, which is devoid of laminin or enactin (Schuppan, 1990). The increase in $\alpha 6 \beta 1$ expression along with the acquisition of biliary-specific expression of $\alpha 2 \beta 1$, $\alpha 3 \beta 1$,and $\alpha 6 \beta 4$, which are integrin receptors for laminin, correlated with the deposition of laminin at the contact points of the portal mesenchyme with the ductal plate (Couvelard et al, 1998).

Remodeling of the Ductal Plate

As mentioned earlier, the double-layered ductal plate around the portal vein begins to form focal dilatations incorporated into the portal mesenchyme to form intrahepatic biliary ducts, while the parts of the ductal bilayer plate not involved in the formation of the ducts progressively regress. This mechanism of regression is thought to be carried out by apoptosis (Sergi et al, 2000; Terada & Nakanuma, 1995). Cell-matrix interactions are also thought to contribute to the remodeling process. Tenascin was found to be expressed in the mesenchyme around the biliary epithelial cells of primitive ducts migrating into the mesenchyme. In contrast, tenascin was absent in mesenchyme of peripheral ducts.

Tubulogenesis of ductal cells is thought to be contributed by soluble factors secreted from hepatocytes or biliary epithelial cells. When coculturing human biliary epithelial cells with hepatocytes, a marked ductular morphogenic response was induced, and the biliary epithelial cells formed well-organized luminal ducts. This result was reproduced when biliary epithelial cells were grown in a conditioned medium from previous hepatocyte and biliary epithelial coculturing (Auth et al, 2001); however, it remains unclear whether soluble factors contribute to tubulogenesis in in vivo studies.

Developmental Relationship Between the Ducts,

Vessels and Mesenchyme of the Portal Tract

A functional relationship appears to exist between the contents to the portal tract (bile duct, hepatic artery, and portal vein). based on observations of a number of human diseases termed“ductal plate malformations.” These diseases include biliary atresia, Caroli disease, and Meckel and Alagille syndromes, in which abnormal biliary ducts are associated with anomalies of the portal mesenchyme and of the portal blood vessels (Lemaigre, 2003). This association was also demonstrated in studies on NOTCH pathway defects. In Alagille syndrome, an autosomal dominant disease, bile ducts are absent in the portal tract, associated with an increased number of arteries and fibrosis.

Haploinsufficiency of Jagged-1 (JAG1), a NOTCH receptor ligand, is associated with Alagille syndrome, where JAG1 is persistently expressed in the

ductal epithelium in humans (Li et al, 1997; Louis et al, 1999). It is also expressed in the endothelial cells of the developing portal vasculature (Crosnier et al, 2000). The animal model for Alagille syndrome was replicated in double-heterozygous mice for mutations in the *JAG1* and *NOTCH2* genes *(J1N2+/−).* JAG1 protein was expressed in the hepatic vasculature, and NOTCH2 was expressed in a subset of hepatoblasts surrounding the portal vein, hepatic artery, and bile ducts. Interestingly, although neither JAG1 nor NOTCH2 protein was expressed in the bile duct epithelium of these mice, JAG1 is expressed in ductal epithelium in humans (Louis et al, 1999; McCright et al, 2002). The differencesin humans (Louis et al, 1999; McCright et al, 2002). The differences in JAG1 expression between human and mouse most likely reflect species specificity rather than technical artifacts, as mice that are heterozygous or homozygous for the *JAG1* gene did not show the liver symptoms of Alagille syndrome (Xue et al, 1999). Similar biliary abnormalities were observed in *NOTCH2*-null mutant mice (McCright et al, 2002).

Hepatocyte Differentiation

During later stages of development, hepatocytes undergo a transition period from a hematopoietic support role to a mature adult hepatocyte. This change occurs under the control of the transcription factor CEBP with HNF4, the latter being a crucial factor in hepatocyte differentiation. Loss of HNF4 function led to the disruption of the expression of several genes associated with a mature hepatocyte phenotype. In *HNF4−/−* mice, hepatocytes failed to express many mature hepatic enzymes and lacked normal morphology, leading to low glycogen storage, disrupted sinusoids, and gap junction disruption.

Other factors that have been shown to promote hepatocyte differentiation include oncostatin M (OSM), an interleukin-6 (IL-6) family cytokine, HGF, and WNT (Michalopoulos et al,2003; Tan et al, 2008). Although as discussed earlier in the chapter, the repression of WNT signaling in the foregut endodermis necessary for hepatic specification, its role is reversed at later gestational stages to promoting hepatocyte differentiation. β-Catenin, a central component of the canonical WNT pathway, is essential for normal development; its aberrant activation in liver was associated with tumors, including hepatic adenomas and hepatocellular cancers (de La Coste et al, 1998; Peifer & Polakis, 2000; Zucman-Rossi et al, 2006). Livers that were deficient in β-catenin displayed decreased numbers of hepatocytes, with hepatoblasts that lacked maturation, proliferation, and function (Tan et al, 2008). In addition, CEBP α, a fundamental regulator of hepatocyte differentiation and maturation (Tan et al, 2008), was decreased. Also noted in these livers was a complete absence of CK-19–positive intrahepatic biliary ducts, suggesting that β-catenin may play a role in biliary differentiation. In vitro studies showed that OSM, produced by hematopoietic cells in fetal liver, as well as HGF, induce hepatic differentiation in the presence of dexamethasone, both working through different pathways (Kamiya et al, 2001). It was demonstrated that embryonic livers at E14.5 expressed glucose-6-phosphatase (G6Pase), tyrosine amino transferase (TAT), and accumulated glycogen, all signs of a mature and differentiated liver when cultured with HGF or OSM. This was further supported when TAT levels, as well as glycogen storage, were significantly reduced in livers derived from mice null mutant for gp130, the common receptor subunit of IL-6 family cytokines (Kamiya et al, 2001). CEBP α is also

critical for the acquisition and maintenance of hepatocyte differentiation, with knockout mice exhibiting defects in liver growth and architecture as well as increased cell proliferation (Flodby et al, 1996). CEBP α is also thought to be a key factor in controlling the switch in the differentiation of bipotential hepatoblasts to become either biliary epithelial cells or hepatocytes.

CEBP α starts to be expressed in the endodermal liver primordium at E9.5, and its expression in the nuclei of hepatoblasts and hepatocytes becomes stronger with development.

During biliary cell differentiation, CEBP α expression was suppressed in periportal biliary cell progenitors, suggesting that its suppression may be a prerequisite to biliary cell differentiation from hepatoblasts (Shiojiri et al, 2004). Lineage-tracing experiments have recently shown that the hepatic biliary tree, as well as the pancreatic ductal tree, and intestinal crypts, which technically are all a continuous epithelial lining (Fig. 1.6), harbor a common pool of progenitor cells, *SOX9* positive, that can all generate a continuous supply of hepatocytes, acini, and all of the mature intestinal cell types, respectively, under physiologic conditions (Furuyama et al, 2011). In hepatocyte differentiation from the lineage tagged SOX positive, bile duct cells increased during the heparegenerative process, suggesting that the biliary and pancreatic ductal tree (SOX9-expressing domains) contain a previously unappreciated pool of progenitors. It has been suggested that these progenitor-like SOX-positive cells reside in the glands (also known as peribiliary glands, or PBGs) of the extrahepatic and large intrahepatic bile ducts.

These progenitor-like cells are responsible for the renewal of surface epithelium, generating mature cells such as cholangiocytes, goblet cells, and hepatocytes. Although it is well demonstrated that the canal of Hering is a likely stem cell niche in the adult liver, harboring human hepatic stem cells, PBGs may be a newly identified reservoir. Clinically, human hepatic stem cells are considered to be the origin of some hepatocarcinomas and intrahepatic cholangiocarcinomas (Cardinale et al, 2012; Carpino et al, 2012).However, the PBG may be a site for oncogenic initiation. Specifically, endodermal-like stem cells within PBGs may be the cells of origin for mucin-producing cholangiocarcinoma cells (Carpino et al, 2012). Furthermore, cholangiocarcinomas express several markers in common with PBG cells, such as EpCAM, OCT4, and CD133 (Komuta et al, 2008). There have been several studies postulating the exact source of liver regeneration after injury, including hepatocytes (Schaub et al, 2014), ductal cells (Furuyama et al, 2011), PBGs (Carpino et al, 2012), or an identified progenitor cell source (Huch, 2015; Huch et al, 2013). In addition, it was also demonstrated through lineage tracing that hepatocytes dedifferentiate into a bile duct cell progenitor after injury, before differentiating back into functionally mature hepatocytes (Tarlow et al, 2014). A mechanism that is similar to pancreatic acinar cells dedifferentiating into ductlike progenitor cells in response to stress (Shi et al, 2013), as well as the dedifferentiation-redifferentiation pathway seen in pancreatic cells (El-Gohary et al, 2014; Puri et al, 2015; Talchai et al, 2012). However, further studies are needed to reconcile these differences into a unifying theory for the source of liver regeneration.

ANATOMY -LIVER

The liver lies protected under the lower ribs, closely applied to the undersurface of the diaphragm and on top of the inferior vena cava (IVC) posteriorly (Fig. 2.1). Most of the liver bulk lies to the right of the midline, where the lower border lies near the right costal margin. The liver extends as a wedge to the left of the midline, between the anterior surface of the stomach and the left dome of the diaphragm. The upper surface is boldly convex and molded to the diaphragm, and the surface projection on the anterior body wall extends up to the fourth intercostal space on the right and to the fifth intercostal space on the left. The convexity of the upper surface slopes down to a posterior surface that is triangular in outline. The liver is invested with peritoneum except on the posterior surface, where the peritoneum reflects onto the diaphragm, foming the right and left triangular ligaments. The undersurface of the liver is concave and extends down to a sharp anterior border. The posterior surface of the liver is triangular in outline with its base to the right, and here the liver lying between the upper and lower “leaves” of the triangular ligaments is bare and devoid of peritoneum. The peritoneum reflects onto the right posterior liver from the medial aspect of Gerota’s fascia, which is associated with the right kidney. The right adrenal gland lies beneath this reflection. The anterior border lies under cover of the right costal margin, lateral to the right rectus abdominis muscle, but it slopes upward to the left across the epigastrium. Anteriorly, the convex surface of the liver lies against the concavity of the diaphragm and is attached to it by the falciform ligament, left triangular ligament, and upper layer of the right triangular ligament.

Retrohepatic Inferior Vena Cava

The IVC runs to the right of the aorta on the bodies of the lumbar vertebrae, diverging from the aorta as it passes upward. Below the liver, the IVC lies behind the duodenum and head of the pancreas as a retroperitoneal structure passing upward behind the foramen of Winslow posterior to the right hilar structures of the liver. The renal veins lie in front of the arteries and join the IVC at almost a right angle on the left and obliquely on the right. The IVC is embraced in a groove on the posterior surface of the liver. The IVC comes to lie on the right crus of the diaphragm, behind the bare area of the liver; it extends to the central tendon of the diaphragm, which it pierces on a level with the body of T8, behind and higher than the beginning of the abdominal aorta. While the IVC courses upward, it is separated from the right crus of the diaphragm by the right celiac ganglion and, higher up, by the right phrenic artery. The right adrenal vein is a short vessel that enters the IVC behind the bare area. There may be a small accessory right adrenal vein on the right that enters into the confluence of the right renal vein and the IVC. Also, occasionally, a right adrenal vein drains directly into the posterior liver. The lumbar veins drain posterolaterally into the IVC below the level of the renal veins, but above this level, there are usually no vena caval tributaries posteriorly.

Hepatic Veins

The hepatic veins (Figs. 2.2 to 2.4) drain directly from the upper part of the posterior surface of the liver at an oblique angle directly into the vena cava. The right hepatic vein, which is larger than the left and middle hepatic veins, has a short extrahepatic course of approximately 1 to 2 cm. The left and middle

hepatic veins may drain separately into the IVC but are usually joined, after a short extrahepatic course, to form a common venous channel approximately 2 cm in length that traverses to the left part of the anterior surface of the IVC below the diaphragm. In addition to the three major hepatic veins, there is the umbilical vein, which is single in most cases and runs beneath the falciform ligament between the middle and left hepatic veins; it empties into the terminal portion of the left hepatic vein, although, rarely, it drains into the middle hepatic vein or directly into the confluence of the middle and left hepatic veins. In approximately 15% of patients, an accessory right hepatic vein is present inferiorly (see Fig. 2.3). Hepatic venous drainage of the caudate lobe is directly into the IVC, as described later. This classic description of the anatomy of the liver is sufficient for gross appreciation and for mobilization of the liver to allow access for repair of injuries, liver transplantation, or the placement of probes onto or into the liver substance. Hidden beneath this external gross appearance is a detailed internal anatomy, an understanding of which is essential to the performance of precise hepatectomy. This internal anatomy has been called the *functional* anatomy of the liver.

Functional Surgical Anatomy

The internal architecture of the liver is composed of a series of segments that combine to form sectors separated by scissurae that contain the hepatic veins (Fig. 2.5), as described by Couinaud (1957). Together or separately, these constitute the visible lobes described previously. The internal structure has been

clarified by the publications of McIndoe and Counseller (1927), Ton That Tung (1939, 1979), Hjörtsjö (1931), Healey and Schroy (1953), Goldsmith and Woodburne (1957), Couinaud (1957), and Bismuth and colleagues (1982). Essentially, the three main hepatic veins within the scissurae divide the liver into four sectors, each of which receives a portal pedicle. The main portal scissura contains the middle hepatic vein and progresses from the middle of the gallbladder bed anteriorly to the left of the vena cava posteriorly. The right and left parts of the liver, demarcated by the main portal scissura, are independent in terms of portal and arterial vascularization and biliary drainage(Fig. 2.6). These right and left livers are themselves divided into two by the remaining portal scissurae. These four subdivisions are referred to as *segments* in the description of Goldsmith and Woodburne (1957), but in Couinaud's nomenclature (1957), they are termed *sectors*.

The right portal scissura separates the right liver into two sectors: *anteromedial* (anterior) and *posterolateral* (posterior).With the body supine, this scissura is almost in the frontal plane. The right hepatic vein runs within the right scissura. The left portal scissura divides the left liver into two sectors, but the left portal scissura is not within the umbilical fissure because this fissure is not a portal scissura, and instead it contains a portal pedicle. The left portal scissura is located posterior to the ligamentum teres and within the left liver, along the course of the left hepatic vein.

Although the description by Couinaud has been used widely, it is being replaced by an alternative terminology suggested by a committee of the International Hepato-Pancreatico-Biliary Association. The main difference is

that in the alternative terminology, Couinaud's sectors arereferred to as *sections* (Table 2.1) (see Chapter 103B for differences in the terminology of the various hepatic resections). Also, note that the left medial section, in the terminology of Strasberg(2005), is composed of one segment (i.e., segment IV).

At the hilus of the liver, the right portal triad pursues a short course of approximately 1 to 1.5 cm before entering the substance of the right liver (Fig. 2.7). In some cases, the right anterior and posterior pedicles arise independently, and their origins may be separated by 2 cm. In some cases, it appears as if the left portal vein arises from the right anterior branch (see Fig. 2.40). On the left side, however, the portal triad crosses over approximately 3 to 4 cm beneath segment IV (formerly called the *quadrate lobe*), embraced in a peritoneal sheath at the upper end of the gastrohepatic ligament and separated from the undersurface of segment IV by connective tissue (hilar plate).

This prolongation of the left portal pedicle turns anteriorly and caudally within the umbilical fissure, giving branches of supply to segment II first and then segment III and recurrent branches ("feedback vessels") to segment IV (Fig. 2.8; see Fig. 2.6).Beneath segment IV, the pedicle is composed of the left branch of the portal vein and the left hepatic duct, but it is joined at the base of the umbilical fissure by the left branch of the hepatic artery.

The branching of the portal pedicle at the hilus (Fig. 2.9), the distribution of the branches to the caudate lobe (segment I) on the right and left sides, and the distribution to the segments of the right (segments V through VIII) and left (segments II through IV) hemiliver follow a remarkably symmetric pattern and, as

described by Scheele (1994), allow separation of segment IV into segment IVa superiorlyIVa superiorly and segment IVb inferiorly. This arrangement of subsegments mimics the distribution to segments V and VIII on the right side. The umbilical vein provides drainage of at least parts of segment IVb after ligation of the middle hepatic vein, and it is important in the performance of segmental resection. The caudate or segment I is the dorsal portion of the liver lying posteriorly and embraces the retrohepatic IVC.

The caudate is intimately related to major vascular structures. On the left, the caudate lies between the IVC posteriorly and the left portal triad inferiorly and the IVC and the middle and left hepatic veins superiorly. The portion of the caudate on the right varies but is usually quite small. The anterior surface within the parenchyma is covered by the posterior surface of segment IV, the limit an oblique plane slanting from the left portal vein to the left hepatic vein. Thus there is a caudate lobe with a constantly present left portion and a right portion of variable size. This portion of the caudate on the right is adjacent to the recently described segment IX, which lies between it and segment XIII. The authors find segment IX of little practical clinical significance.

The caudate is supplied by blood vessels and drained by biliary tributaries from the right and left portal triad. Small vessels from the portal vein and tributaries joining the biliary ducts also are found. The right portion of the caudate, including the caudate process, predominantly receives portal venous blood from the

right portal vein or from the bifurcation of the main portal vein, whereas on the left side, the portal supply arises from the left branch of the portal vein almost exclusively. Similarly, the arterial supply and biliary drainage of the right portion is most commonly associated with the right posterior sectional vessels and the left portion with the left main vessels.

The hepatic venous drainage of the caudate is unique in that it is the only hepatic segment that drains directly into the IVC. These veins can sometimes drain into the posterior aspect of the vena cava, if a significant retrocaval caudate component is present.

In the usual and common circumstance, the posterior edge of the caudate lobe on the left has a fibrous component, which fans out and attaches lightly to the crural area of the diaphragm; but it extends posteriorly, behind the vena link with a similar component of fibrous tissue (called the venal caval ligament) that protrudes from the posterior surface of segment VII and embraces the vena cava . In 50% of patients, this ligament is replaced by hepatic tissue, in whole or in part, and the caudate may completely encircle the IVC and may contact segment VII on the right side; a significant retrocaval component may prevent a left-sided approach to the caudate veins. The caudal margin of the caudate lobe can have a papillary projection that occasionally may attach to the rest of the lobe via a narrow connection.It is bulky in 27% of cases and can be mistaken for

an enlarged lymph node on computed tomography (CT) scan

To summarize:

1. The liver is divided into two hemilivers by the main hepatic

scissura, where the middle hepatic vein runs.

2. The left liver is divided into two sections. The Brisbane 2000 nomenclature describes the left lateral section (segments 2 and 3) and the left medial section (segment 4).

3. The right liver is divided into an anterior section (segments 5 and 8) and posterior section (segments 6 and 7).

4. Segment 1, the caudate lobe lies posteriorly and embraces the IVC, its intraparenchymal anterior surface abutting the posterior surface of segment 4 and merging with segments 6 and 7 on the right .

PATHOPHYSIOLOGY

Blunt Hepatic Injury

In MVCs, those most susceptible to hepatic injury are unrestrained front-seat passengers. These passengers are particularly vulnerable to a compression injury especially during periods of rapid deceleration. Although the anterior abdominal wall stops, the posterior abdominal wall continues to move forward, and the intra-abdominal organs are “trapped” and compressed, resulting in stretching/tearing of the liver at its vascular and structural attachments. As the liver is

only partially protected by the rib cage, liver injury from steering wheel contact is one of the most important contributing factors to driver injury.

In lateral impact (broadside or "T-bone") collisions, the target vehicle is hit on its side and accelerated rapidly at 90 degrees to its previous direction of travel. The unrestrained passenger is subject to both compression and shear injuries that cause stretching and tearing and at times result in avulsion of the liver. Furthermore, in lateral impact injuries, because the spine and posterior abdominal wall are not in the line of impact, in contrast to frontal impact injuries, more relative motion of the intraabdominal organs ensues, resulting in a greater likelihood of injury.

Penetrating Hepatic Injury

Damage caused by a penetrating injury is based on the kinetic energy of the projectile and the density and elasticity of the tissue. Lowenergy weapons such as knives only cut and do not create a temporary cavity. Medium-energy and high-energy firearms damage not only the tissue directly in the path of the missile but also the tissue on each side of the missile's path. As a missile passes through the relatively inelastic liver parenchyma, a temporary cavity (three to six times the size of the missile's front surface area, lasting for a fraction of a second) and a permanent cavity (visible to the examiner) are creatrauma patient, a rapid DPA should be performed.

American Association for the Surgery of Trauma Liver Injury Scale

I - Hematoma Subcapsular, <10% surface area Laceration Capsular tear, <1 cm parenchymal depth

II - Hematoma Subcapsular, 10%–50% surface area; intraparenchymal, <10 cm in diameter ,Laceration 1–3 cm parenchymal depth, <10 cm in length

III - Hematoma Subcapsular, >50% surface area or expanding; ruptured subcapsular or parenchymal hematoma ,Intraparenchymal hematoma >10 cm or expanding Laceration >3 cm parenchymal depth

IV Laceration Parenchymal disruption involving 25%–75% of hepatic lobe or 1–3 Couinaud segments within a single lobe

V Laceration Parenchymal disruption involving >75% of hepatic lobe or >3 Couinaud segments within a single lobe OR Vascular Juxtahepatic venous injuries, i.e., retrohepatic vena cava/central major hepatic Veins

VI Vascular Hepatic avulsion

DIAGNOSTIC AND IMAGING TECHNIQUES

DIAGNOSTIC PERITONEAL LAVAGE

Originally described by Root in 1965, diagnostic peritoneal lavage (DPL) has been a mainstay in the management of blunt abdominal trauma for over four decades. 36 Before the era of routine CT scanning, it was used as a screening tool to evaluate patients having blunt or penetrating abdominal trauma with an accuracy rate reported between 92 and 98%. 37–42 Because of its invasive nature, DPL has largely been supplanted by CT scans and FAST. However, it remains an excellent tool for further workup of occult bowel injury or in unstable patients when FAST is not available or has questionable _ ndings. In the workup for occult bowel injury, traditional parameters should be used to guide therapy. In unstable patients, a diagnostic tap is usually all that is necessary, and exploration indicated for greater than 10 mL of gross blood. _ e pitfalls of DPL are a relatively high false-positive rate, risk of creating visceral injury, and poor sensitivity for detecting injury to retroperitoneal structures such as the pancreas and duodenum. 43–45 Iatrogenic events are minimized if a Foley catheter and nasogastric tube are placed prior to the procedure. Patients with pelvic fractures and suspected retroperitoneal hematoma or pregnant females should undergo a supraumbilical approach. Visceral injury is less likely with an open approach but more time consuming and invasive. 46–49 Checking amylase or lipase in lavagate, concomitant use of CT scan, and a high index of suspicion are necessary to avoid missed retroperitoneal injury.

FOCUSED ABDOMINAL SONOGRAPHY

FOR TRAUMA

One of the most recent advances in the workup of the acutely injured patient is the use of bedside ultrasonography for detection of cardiac and intra-abdominal injury. Known as focused abdominal sonography for trauma (FAST), this technique's noninvasive nature allows the operator to perform an examination simultaneously during the initial resuscitation and stabilization of a multiply injured trauma patient. _ e technique may thereby provide evidence of signicant hemorrhage early in the course of an evaluation. An ultrasound probe is used to examine four key windows for:

1)the subxyphoid area permits visualization of the pericardium,

2)the left subcostal area visualization of the splenorenal recess,

3)Right subcostal area visualization of Morison's pouch,

4)the suprapubic area visualization of the pelvic cul-de-sac .this may indicate then presence of cardiac tamponade, intra-abdominal hemorrhage, hollow viscus perforation, hemoperitoneum, or ascites. False-positive results secondary to preexisting ascites or false negatives due to operator error and/or body habitus are the main limitations. Scanning of the suprapubic area with distension of the urinary bladder will enhance the sensitivity of the examination for the detection of pelvic fluid. A threshold of atleast 200ml of fluid in the abdominal cavity is necessary for the detection and intra abdominal injuries

COMPUTED TOMOGRAPHY

Detection of bowel injury via CT scan in patients who are intoxicated, intubated, or have associated closed-head injury or other distracting injuries can present a diagnostic challenge in the absence of a reliable abdominal exam. _ e incidence of blunt bowel injury varies from series to series but is generally reported in the 1–5% range in all blunt trauma patients admitted to level I trauma centers. 58, 59 A high index of suspicion is predicated on mechanism of injury and physical examination findings such as abdominal wall tattooing and/or the seat belt sign. CT findings may be direct such as extravasation of oral contrast or pneumoperitoneum or more commonly indirect such as bowel wall thickening, stranding of the mesentery, or free fluid in the absence of solid organ injury. Indirect findings may be fairly nonspeci and secondary to bowel edema from resuscitation or preexisting ascites. Reproductive age females may have a small amount of normal or "physiologic" pelvic fluid present sometimes adding to the complexity of the evaluation. Patients on positive pressure ventilation or with significant barotrauma may develop mediastinal or subcutaneous emphysema that can tract through the peritoneum or retroperitoneum and give the appearance of free air. Great care in the radiologic interpretation and close clinical correlation are necessary in such cases. _ e liberal use of DPL may prevent non therapeutic laparotomy. Obviously, when significant doubt remains, abdominal exploration may be necessary to confirm an injury. Contrast is usually available in the emergent setting to permit adequate opacification of the small bowel. Patients are further at risk for aspiration of

the contrast media, and administration often requires placement of a nasogastric tube. A number of reports now have shown that elimination of oral contrast media does not lead to an increased incident of missed bowel injury.58–60 Many centers have now safely eliminated the use of oral contrast media from their routine trauma protocols expediting management and ease of patient care. Resuscitation edema may cause a hazy appearance around the head of the pancreas and duodenal c-loop raising the question of a pancreas or duodenal injury. Further clarification in this situation can be obtained, when it occasionally occurs, via repeat CT scan with the administration of oral contrast and the injection of 300- to 500-cc bolus of air down the nasogastric tube and may make the pneumoperitoneum obvious.

CT may also be of great importance in identifying patients with arterial hemorrhage related to pelvic fracture. CT imaging may demonstrate an arterial blush or large hematoma in the vicinity of a pelvic fracture indicating the need for pelvic arteriography or pelvic external fixation. A “CT cystogram” may also be helpful and eliminate redundancy of x-ray evaluation. Foley catheter is clamped after placement in the trauma bay. Real-time interpretation, as the CT scan is performed by the evaluating physician, may dictate further delayed images or a formal three-view (anterior/posterior, lateral, and postvoid views) cystogram.

Hemodynamically Unstable Patients

Patients who arrive with hemodynamic instability (systolic blood pressure <90 mm Hg) and who do not immediately respond to appropriate fluid resuscitation are expeditiously taken to the operating room without delay, irrespective of mechanism of injury. Further diagnostic evaluation at this point is contraindicated, as unnecessary delays inevitably follow and are often responsible for the ensuing fatalities.

In the hemodynamically unstable patient with pelvic fractures from blunt trauma, diagnostic peritoneal lavage (DPL) which has evolved to a quick screening diagnostic peritoneal aspirate (DPA)—or DPA consisting of the initial aspiration portion of the DPL only and focused assessment with sonography in trauma (FAST) are currently the diagnostic modalities used to detect the presence of intraperitoneal blood. A grossly positive aspiration on DPA (>10 mL of gross blood) mandates immediate operative intervention. In most trauma centers, FAST has replaced DPL/DPA as the preferred diagnostic modality for the determination of hemoperitoneum in the unstable bluntly injured patient. Although FAST has a 97% sensitivity for hemoperitoneum greater than 1 L, the location of the parenchymal injury cannot be reliably identified. The sensitivity of FAST drops precipitously when the quantity of intraperitoneal fluid is less than 400 mL. Kuncir and Velmahos found that the sensitivity and specificity of DPA was 89% and 100%, respectively, whereas for FAST it was significantly less at 50% and 95% in their prospective series of hemodynamically unstable patients with blunt abdominal trauma. If a FAST

examination is equivocal in a hemodynamically unstable trauma patient, a rapid DPA should be performed.

Hemodynamically Stable Patients

The hemodynamically stable blunt trauma patient, on the other hand, may undergo further diagnostic studies. Hemodynamic stability, however, should not lull the trauma surgeon into a false sense of security, as significant intra-abdominal injuries may be present despite normal vital signs and a normal abdominal examination. The ability to accurately assess the presence or absence of significant intraabdominal injuries by physical examination alone in the blunt trauma patient is notoriously poor, as up to 20% to 30% of patients with a benign abdomen on physical examination have been shown to subsequently have significant intra-abdominal injuries on imaging or at laparotomy.

CT scanning is the preferred initial diagnostic modality in the hemodynamically stable patient with blunt abdominal or lower thoracic cage injuries. High-speed resolution scanning with a spiral scanner is employed after the administration of intravenous (IV) contrast agent. In most trauma centers, oral contrast material is no longer routinely given for screening abdominal pelvic CT scan for blunt abdominal trauma. Administration of oral contrast agent is usually reserved for the focused assessment of specific hollow viscus injuries such as identification of a duodenal laceration or delineation of a duodenal hematoma. Five-millimeter cuts are

obtained after 120 mL of noniodinated contrast agent (Omnipaque) is injected at a rate of 2 mL/second. Scanning commences 50 seconds after injection, a delay that corresponds to the portal venous phase of liver imaging. Scans should immediately be interpreted and classified according to the American Association for the Surgery of Trauma Liver Injury

Scale by the CT fellow or attending radiologist, always in the presence of the chief trauma resident and trauma attending. As a senior trauma attending usually has more experience than the designated in-house radiology resident, the surgical attending physician's initial impartial review of the CT scan is vital. The senior trauma attending in presence makes the final decision as to the appropriateness of nonoperative therapy.

It should be noted that the grade of injury or degree of hemoperitoneum on CT does not determine the need for operative intervention, as this decision is based primarily on the patient's hemodynamic stability and the absence of peritonealsigns and the absence of need for laparotomy if a concomitant hollow viscus injury is identified.

Instead, the CT scan merely provides the surgeon with a general anatomic overview of the injury, identifies associated abdominal injuries requiring operative intervention, and can be used as a base for comparing future healing of the hepatic injury and resorption of intraperitoneal blood. CT can also identify injuries involving the bare area of the liver, which commonly present with minimal intra-abdominal bleeding, a paucity of abdominal signs, and often a negative DPL/DPA.

The role of FAST as a screening examination in hemodynamically

stable patients is evolving. Currently, many trauma centers forgo CT scanning in stable patients with negative initial FAST examinations and merely repeat the FAST in 6 hours. However, scanning for only free fluid has its diagnostic limitations because not all blunt hepatic injuries result in hemoperitoneum.

In a recent study looking specificallyat sonographic detection of blunt hepatic trauma, Richards et al determined the overall sensitivity of FAST for blunt hepatic injuries (all grades) to be 67%, based on the detection of free fluid alone. On the other hand, it is clear that most solid organ injuries without intraperitoneal fluid on FAST are, in general, of minimal clinical significance.

At present, most trauma surgeons agree that those patients who are hemodynamically stable and who have either intraperitoneal blood on their initial FAST examination or positive findings on physical examination over the lower chest and upper abdomen should have a CT scan to specifically identify a hepatic or splenic injury that can be managed nonoperatively. Once identified, the hepatic injury may be followed with ultrasound if necessary.

Diagnostic laparoscopy (DL) is a safe procedure that has had a major impact in avoiding unnecessary abdominal explorations in patients with stab wounds or gunshot wounds that may not have penetrated the peritoneal cavity. The role of DL in patients with blunt hepatic injury is less clear. DL should allow for an accurate assessment of most hepatic injuries and, as advances in laparoscopic instrumentation progress, perhaps allow for repair of some liver injuries. However, reports of missed enteric and other intra-abdominal injuries with DL are sufficiently numerous to significantly limit the usefulness of DL.

MANAGEMENT

Nonoperative Management of Blunt Hepatic Trauma

Currently, nonoperative management of adult blunt hepatic injuries is the standard of care. Approximately 85% to 90% of all liver injuries may be successfully managed nonoperatively in both adults and the pediatric population. A recent publication examined the data on 14,919 liver injuries submitted to the National Trauma Data Bank and revealed that only 13.6% of all liver injuries underwent operation.

As the grade of liver injury increased so did the likelihood of operative intervention: grades I and II¼8.5% (n¼10,178), grade III¼21% (n¼2793), grade IV¼27.2% (n¼1462), grade V¼37.4% (n¼439), grade VI¼42.6% (n¼47). Initial hemodynamic stability or hemodynamic stability achieved and maintained with moderate fluid resuscitation is the single most crucial prerequisite qualifying patients for nonoperative management. Once hemodynamic stability has been ascertained, the following criteria must be met:

1**) Absence of peritoneal signs**

2) **Precise CT scan delineation and AAST grading**

3) **Absence of associated intra-abdominal or retroperitoneal injuries**

4) **CT scan that require operative intervention**

5) **Avoidance of excessive hepatic-related blood transfusions**

Previously cited inclusion criteria such as neurologic integrity are no longer valid, as neurologically impaired patients can be safely managed nonoperatively in a monitored setting. Furthermore, mandatory repeat CT scans to document improvement or stabilization of injury are unnecessary and contribute little to patient outcome. Rather, the patient's clinical course should dictate the need for additional evaluation.

Interestingly, in his landmark 1908 article describing clamping of the portal triad to arrest hepatic hemorrhage, Pringle also alluded to the physiologic tamponade provided by the abdominal wall and the potential advantage of nonoperative management in patients with less severe injuries: "The mere act of opening the abdomen, in some, at any rate, of these cases is, I feel certain, associated with an increase of the amount of blood that is lost to the patient. The blood pressure in the portal vein is not great and as the result of the local injury and the extravasation of blood there is produced reflexly a state of firm contraction of the abdominal muscles. The abdominal wall in these cases becomes absolutely rigid and board-like, the tension in the abdominal cavity thereby brought about must prevent at least a rapid escape of blood and may lead to its arrest altogether."

Today, the majority of blunt hepatic trauma patients can be successfully managed nonoperatively. Although nonoperative management was initially limited to AAST grades I to III injuries, it is now clear that the hemodynamic status of the patient, rather than AAST grade of injury, is the most significant factor in determining the need for operative intervention. Select patients with grades IV

and V injuries can be managed nonoperatively. However, many grade IV and V injuries will usually present with hemodynamic instability or concomitant injuries mandating surgery, thus precluding nonoperative intervention. In a multi-institutional study, grades IV and V injuries were responsible for 67% of all patients who failed nonoperative management and subsequently required operative intervention.

Therefore, although hemodynamic stability determines which patients can be managed nonoperatively, the subgroup of patients with complex hepatic injuries (grades IV and V) are at substantially higher risk for treatment failure and should therefore be closely monitored in a critical care unit.

Conversely, the same basic standards apply to patients with lower AAST-grade injuries (i.e., I through III). In these instances, the initial injury may be deemed as "not significant," and thus it becomes tempting to avoid surgical intervention despite hemodynamic instability or a decreasing hematocrit, relying instead on further fluid and blood transfusions. This course of action is fraught with pitfalls and should be avoided to minimize the morbidity and mortality risks of nonoperative management. To summarize, of all the variables monitored, hemodynamic stability appears to be the most crucial and is considered the watershed for nonoperative or operative intervention.

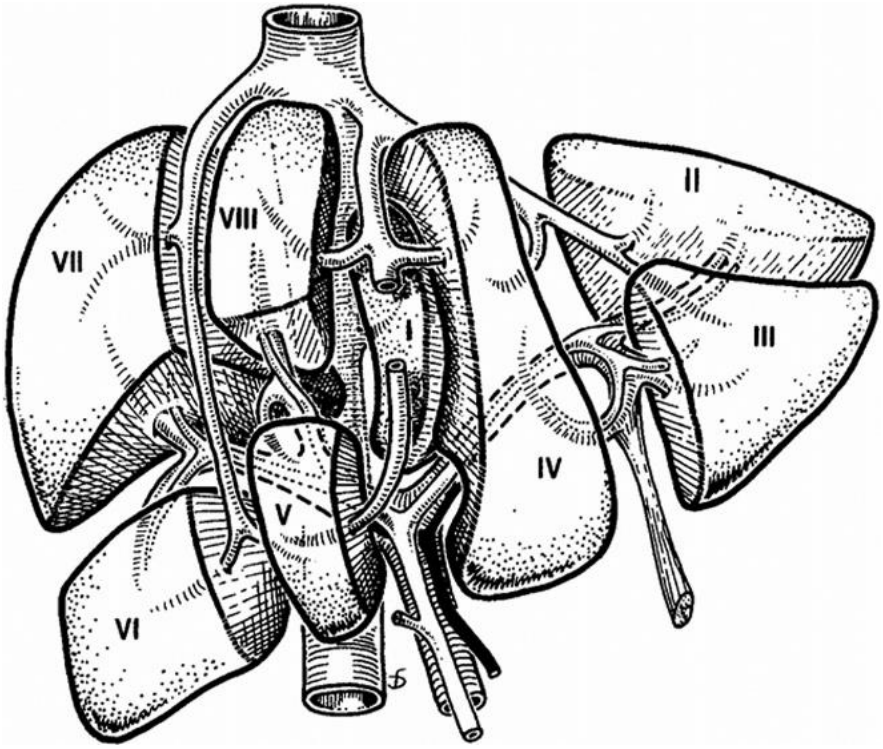
Contrast "Blush" on Computed Tomography

Specific cause for concern is the presence on the initial CT scan, after administration of IV contrast agent, of a contrast "extravasation," "blush," or "pooling" of contrast material within the hepatic parenchyma. This finding indicates active bleeding. Even in the context Of hemodynamic stability and irrespective of AAST grade of injury, preparation for possible surgical intervention should promptly be made, as patients can suddenly and unpredictably decompensate clinically. If the patient remains hemodynamically stable, angiography with the intent of embolizing the lacerated vessel should be attempted (with an operating room on standby secured).

An experienced interventional radiologist will usually have little difficulty in selectively catheterizing and embolizing the injured vessel, most often with stainless steel coils rather than Gelfoam to achieve the most dependable and permanent embolization. Successful embolization can then potentially permit further nonoperative management.

As the natural history of intrahepatic vessels with evidence of extravasation is unknown, they are best dealt with immediately so that sudden bleeding, false aneurysm formation, and late hemobilia may be avoided. Persistent and prolonged attempts at controlling the bleeding vessel through angiographic means should be discouraged. In the rare event in which angioembolization (AE) fails to control ongoing bleeding, surgical intervention using the angiogram as an anatomic marker to more rapidly achieve intrahepatic hemostasis should promptly be undertaken.

FIGURE 1 Functional division of the liver, according to Couinaud's nomenclature. (From Mattox KL, Feliciano DV, Moore EE, editors: Trauma, 4th ed, New York, 1999, McGraw-Hill, Fig. 30-1. Originally appeared in Blumgart LH, editor: Surgery of the liver and biliary tract, New York, 1988, Churchill Livingstone.)



Operative Management

General Principles

The four basic principles in the management of liver trauma requiring surgery are

1) Hemostasis
2) Adequate exposure,
3) Prevention of coagulopathy,
4) consideration of damage control.

Débridement and the need for drainage are also important considerations. With hepatic injuries, these objectives can be reached by the use of the finger-fracture technique (hepatotomy) to incise hepatic parenchyma, often combined with temporary occlusion of the portal triad for hemostasis using the Pringle maneuver. Extensive débridement of injured hepatic tissue can then be done, followed by application of a viable pedicled omental pack and closed-suction drainage.

Before the incision is made, the patient should receive a dose of antibiotics to cover aerobic and anaerobic microbes and is placed on a warming blanket. The surgeon must keep in mind that hypothermia is a frequent complication of resuscitation and operation in patients with major hepatic injuries. Appropriate maneuvers to decrease hypothermia . Adherence to these maneuvers will usually prevent the development of intraoperative coagulopathies, excessive hemorrhage, and fatal arrhythmias secondary to hypothermia.

The skin is prepped from the chin to the knees and a standard midline incision is made. The midline incision not only affords excellent exposure of the entire liver but also provides wide access to all peritoneal and retroperitoneal structures. The combination of a long midline incision and the use of large "upper-hand" retractors have, for the most part, eliminated the need for thoracic extension of the abdominal exposure. It should be kept in mind that extending the midline incision to the sternal notch (i.e., completing a median sternotomy) exposes the patient to two open cavities with the attendant increased risks of hypothermia and coagulopathy. Exsanguinating hemorrhage continues to remain the most immediate cause of death in patients sustaining hepatic trauma.

The initial incision into the peritoneal cavity can be accompanied by profuse hemorrhage once the tamponading effect has been lost. At this time, all efforts should be directed toward intraoperative resuscitation and consideration for utilizing the technique of damage control, temporary packing of the liver, and attendant correction of coagulopathy and hypothermia in the intensive care unit (ICU) with delayed laparotomy as an adjunct. Attempts at definitive surgical hemostasis without proper intraoperative resuscitation usually results in systemic hypothermia and profound coagulation defects with their dire consequences. This fundamental pitfall should be avoided at all costs.

Irrespective of the severity of hepatic injury, almost all liver injuries can be initially managed by manually compressing the injury with lap pads (Fig. 3), while hemodynamic and metabolic stability are restored by the anesthesia team. Failure to correct hypovolemia and acidosis before attempts at surgical control will likely lead to

cardiac arrest and subsequent death. Once intraoperative resuscitation has been achieved, manual compression of the liver is slowly released so that a more accurate assessment of the injury can be made.

Division of the falciform ligament allows for placement of an "upper-hand" self-retaining retractor in the incision. In order to better visualize injuries on the superior or lateral aspects of an injured hepatic lobe, it is often necessary to mobilize the liver into the midline wound. Once this is done, careful traction on its hepatic end can aid in exposing the dome of the liver and the suprahepatic inferior vena cava. Additional exposure is obtained by placing laparotomy pads behind the posterior surface of the liver. Mobilization of the right and left lobes proceeds with division of the triangular ligaments . If there is a hematoma within the leaves of the triangular ligament, a hepatic vein or venal caval injury is most likely. If the hematoma is not expanding and there is no immediate active hemorrhage requiring control, entering a stable retrohepatic hematoma is not advised. Extreme caution must be taken even during traction as this may disrupt a stable hematoma and can create massive bleeding.

Minor Injuries (Grades I and II)

Simple techniques of controlling hemorrhage include a 5- to 10-minute period of compression, application of topical agents including fibrin glue, electrocautery/argon beam electrocoagulation, and suture hepatorrhaphy (Fig. 5). In many patients with superficial lacerations of the capsule, a 5- to 10-minute period of compression will frequently control any hemorrhage. If there is no visible leakage of bile, no further therapy is indicated. Topical agents, such as fibrin glue, Surgicel, and

Avitene, are useful when avulsion of Glisson's capsule is present. Five minutes of compression with lap pads is performed after the application of a topical agent to the raw surface. After releasing compression, the electrocautery can be used for any remaining bleeders. Fibrin glue or the other hemostatic agents may be overlaid with a large Gelfoam pad creating a nonadherent surface to compress a gauze laparotomy pad against. Drainage is not necessary in the absence of obvious bile leakage.

Suture hepatorrhaphy has historically been the mainstay of hepatic hemostasis in grade II and some grade III injuries. It is important to first enter the hepatic wound and selectively ligate any open or avulsed bile ducts or blood vessels. Figure-of-eight 2-0 or 3-0 Prolene sutures are usually employed. Alternatively, 2-0 or 3-0 chronic sutures or hemoclips can also be used. Small defects in the hepatic parenchyma can be closed with simple interrupted 0-chromic or 2-0 chromic liver sutures either with regular or blunt-nosed needles. For deeper lacerations, attempts at primary closure of the hepatic defect should not be undertaken. Instead, a flap of omentum on a pedicle is placed within the hepatic parenchymal defect and is then held in place with interrupted liver sutures . It is important to loosely approximate the edges because portions of the liver beneath can become necrotic in the postoperative period if the sutures are tied too tightly.

Complex Injuries (Grades III and V)

If significant hemorrhage continues after the release of manual compression of the liver, the portal triad should be occluded with an atraumatic vascular clamp (the Pringle maneuver;). In over 85% of patients with complex hepatic injuries, occlusion of the portal triad will temporarily stop the bleeding. This maneuver, coupled with the finger-fracture technique to expose lacerated blood vessels for direct repair, is responsible for the dramatic decrease in deaths from exsanguination.

Complex hepatic injuries (grades III to V) can best be managed by adhering to several sequential crucial steps:

1. Portal triad occlusion **(Pringle maneuver**)
2. Finger fracture of the hepatic parenchyma (**hepatotomy**), exposing lacerated vessels and bile ducts for direct ligation/ repair
3. Consideration of **temporary packing with** laparotomy pads to allow appropriate intraoperative resuscitation
4. Consideration of temporary intrahepatic packing with hemostatic agents such as surgical Nu-Knit
5. Débridement of nonviable hepatic tissue
6. Placement of an omental pedicle, with its blood supply intact, into the injury site
7. Closed-suction drainage

Maneuvers to Prevent/Decrease

Hypothermia in Patients with Major Hepatic Injuries

1) Resuscitation with warm (37°–40° C) crystalloid solutions
2) Resuscitation with high-flow blood warmers
3) Covering the patient's head with plastic bags
4) Placing the patient on a heating blanket
5) Use of a Bair Hugger on the lower extremities and on chest if thoracotomy is not needed
6) Irrigation of open body cavities with warm saline
7) Use of heating cascade on anesthesia machine

Much controversy has surrounded the normothermic ischemic time produced by the Pringle maneuver. The data are clear that complex hepatic injuries can be managed with continuous cross-clamping of the porta hepatis for up to 75 minutes without adverse sequelae.With portal triad occlusion achieved by an atraumatic vascular clamp, the surgeon then opens the liver parenchyma (hepatotomy) in the direction of the injury (Fig. 9). Although it initially seems crude, the finger-fracture technique constitutes the benchmark of obtaining rapid, adequate exposure. Specifically, using the electrocautery, Glisson's capsule is incised in the direction of the injury. Normal hepatic parenchyma is then crushed between the surgeon's thumb and index finger (or a neurosurgical suction device), thereby rapidly exposing

injured blood vessels and bile ducts, which are repaired or ligated under direct vision. Narrow Deaver or malleable retractors can be inserted into the hepatotomy tract for better intrahepatic exposure. Large lacerated intralobar branches of the portal vein or hepatic veins can be repaired in a lateral fashion using 5-0 Prolene sutures (Fig. 11).

After intrahepatic hemostasis has been achieved, thorough débridement of devascularized hepatic tissue is essential to avoid postoperative septic complications. The use of omentum is extremely beneficial in the management of complex hepatic injuries, as it provides viable tissue to fill dead space, tamponades minor venous oozing, and provides a rich source of macrophages that may help combat infection.

The choice to drain a liver injury is controversial and debatable. If bile is noted intraoperatively, drainage is not controversial and is mandatory. The preferred method of drainage is with closed-suction Jackson-Pratt (JP) drains anterior and posterior to the injury. The data rendering drains unnecessary in elective hepatic resection cannot be applied to complex hepatic trauma, in which hypotension, and the frequent need to terminate surgery are the usual order of the day. In addition, the "zone" of injury may extend centimeters beyond what appears to be normal hepatic parenchyma, leading to eventual necrosis and abscess formation. Although routine drainage after elective hepatic resection may be superfluous, enough variables exist in the trauma setting to merit consideration of the use of closed-suction drains for complex hepatic injuries.

Data supporting a clear choice of drains can be found in a noteworthy publication by McSwain et al who reviewed 164 cases of liver trauma with 12 subsequent intra-abdominal abscesses at Charity Hospital and characterized the infection rates associated with various types of drainage catheters in these traumatic liver injuries. Thirtyfour percent of the patients had no peritoneal drainage and an abscess rate of 1.8%. Closed-suction drains of the JP variety had the lowest associated infection rate: 18% of patients with a 0% abscess rate. Fourteen percent of patients had open Penrose drains with an infectious complication rate of 8.7%. Nineteen percent of patients had the combination of a Penrose and a sump type of drainage with the highest associated complication rate of 22.5%. Closed JP circuits are the drains of choice for hepatic trauma. Use of open drains such as Penrose or sump drains should be discouraged secondary to their association with unacceptably high rates of abscess formation.

Damage Control: Perihepatic Packing and Planned Reexploration

Perihepatic packing has emerged as an essential lifesaving maneuver in patients with complex injuries refractory to conventional methods of treatment and usually complicated by brisk bleeding, hypothermia (less than 34° C), acidosis (pH <7.2), and coagulation defects from massive transfusion (over 10 units PRBCs). The effectiveness of perihepatic packing is directly related to the tamponading effect of the packs on the hepatic injury. Specifically, the packs raise intraabdominal pressure (IAP), causing tamponade of low-pressure venous and nonmechanical capillary

bleeding. The key to the success of perihepatic packing is to insert the packs early in the course of the operation before the onset of repeated episodes of hypotension. Primary indications for perihepatic packing follow:

1) Onset of intraoperative coagulopathy

2)Extensive bilobar injuries in which bleeding cannot be controlled

3)Large, expanding subcapsular hematomas or ruptured hematomas

4)The necessity to terminate surgery as a result of profound hypothermia, which usually results in hemodynamic instability

5)Failure of other maneuvers to control hemorrhage

6)Patients who require transfer to Level I trauma centers

7)Juxtahepatic venous injuries

As a general rule, the liver should be mobilized before packing to help establish a tamponading effect. If, however, a significant hematoma is encountered in the triangular ligament (indicative of a vena caval or hepatic vein injury), further mobilization is contraindicated as massive and uncontrollable bleeding may follow. Most often, dry multiple-lap pads are placed on top of the injured liver until the ipsilateral hemidiaphragm is reached (Fig. 12). In order to lessen the degree of bleeding when lap pads are peeled off the raw liver surface, an option is to routinely place a Steri-Drape (3 M, St. Paul, Minn.) directly upon the liver surface to serve as an interface between the injured liver and the lap pads. Another alternative is

to place large noncompressed sheets of Gelfoam over the raw liver surface as a hemostatic adjunct which additionally provides a layer of protection preventing bleeding when the laparotomy packs are removed.

Resorting to packing is usually synonymous with a dire situation. Under these circumstances, rapid closure of the abdomen with towel clips can be undertaken. Several large Steri-Drapes impregnated with Betadine cover the entire incision, encompassing all towel clips.

Towel-clip closure takes minutes to perform and facilitates rapid patient transfer to a critical care setting where the patient's metabolic status can be optimized. Alternatively, prosthetic abdominal wall closure with a sterilized IV bag, commonly known as a Bogata bag, can also be employed. Commercially designed wound VAC (vacuumassisted closure) systems are also available and extremely useful with both the wound VAC and the latest version of this system—the AB Thera Open Abdomen Negative Pressure Therapy System device (KCI, San Antonio, Tex.), which is now being used most of the time. Because perihepatic packing raises IAP, monitoring IAP in the perioperative period is critical to avoid the development of an abdominal compartment syndrome (ACS). Pack removal should be dictated by the reversal of the patient's hypothermia, acidosis, and coagulopathy. These goals can usually be achieved within 36 to 48 hours. Packing has been historically associated with a 20% to 30% incidence of perihepatic sepsis. However, early pack removal, the evacuation of intraperitoneal clots, and the thorough débridement of necrotic hepatic tissue have lessened the incidence of this complication.

Juxtahepatic Venous Injuries (Grade V)

Juxtahepatic venous injuries, especially from blunt trauma, are often fatal, with mortality rates up to 50%. Failure to control haemorrhage from a deep laceration, missile tract, or stab wound with a Pringle maneuver still in place strongly suggests the presence of a juxtahepatic venous injury. Regardless of the technique used to manage these devastating injuries, early recognition is essential because prompt modification of the surgical approach is necessary.

In the past, in order to try to salvage these patients, trauma surgeons inserted an atriocaval shunt with a resultant prohibitively high mortality rate (60% to 100%). Currently, the use of atriocaval shunting has been virtually abandoned, and these difficult injuries have been, at times, successfully managed using a variety of approaches.

At present, there is a general consensus among trauma surgeons that if a retrohepatic caval injury or a hepatic venous injury (grade V) can be adequately controlled with perihepatic packing, no attempts at further repair should be initiated. When adequate resuscitation has been accomplished, there may be a role for endovascular stenting of the injury before pack removal. Even without endovascular stenting, when planned reexploration is undertaken, no further bleeding is often noted. If bleeding occurs after pack removal, definitive treatment can then be undertaken with the knowledge that the patient's hemodynamic status has been optimized and that adequate personnel are available if a vascular shunt is necessary.

Another approach is direct hepatotomy through Cantlie line to

reach the injured retrohepatic cava or hepatic veins. After manual compression, vigorous resuscitation, and prolonged portal triad occlusion, mobilization of the liver is performed with medial rotation, thus providing access to the retrohepatic cava and hepatic veins.

Rapid and extensive finger fracture should be directed toward the site of injury until the lacerated retrohepatic cava or hepatic vein is found and repaired under direct vision. The surgeon must be prepared to finger fracture the hepatic parenchyma through normal and frequently nonanatomic planes. Because these patients usually have injured hepatic parenchyma as well, portal triad occlusion serves two purposes: it contributes to controlling hemorrhage from intrahepatic branches of the hepatic artery and portal vein, and it decreases the inflow to the liver, thereby aiding finger fracture and minimizing
blood loss.

A third approach comprises venovenous bypass, vascular exclusion, and primary repair. Total vascular isolation of the liver via venovenous bypass (combined with the Pringle maneuver and clamping of the suprarenal and suprahepatic cava) permits direct suture repair of the venous injury. The advantage here is that vascular isolation with venovenous bypass obviates the need for an intracaval shunt. Cannulation for bypass can be done peripherally via saphenous vein and axillary vein cutdowns. Venovenous bypass has been used in a small
number of severe retrohepatic liver injuries with an overall survival
rate of 88%.

Next is total hepatic resection and delayed liver transplantation.

Total hepatectomy and second-stage hepatic transplantation can be a drastic yet lifesaving maneuver for devastating liver injuries that have failed all conventional treatments. Perihepatic packing and total hepatectomy with portacaval shunting can be performed in the primary hospital; the anhepatic patient can then be transferred to a transplant center for eventual liver transplant. Although this radical maneuver can be associated with high morbidity and mortality rates, the prognosis is better if the decision to proceed with total hepatectomy and portacaval shunting is made before the development of intractable multiorgan failure.

Portal Triad Injuries

As exsanguination is the most common (85%) cause of death in these highly lethal and complex injuries, the first priority in portal triad trauma is hemorrhage control, specifically manual compression followed by the Pringle maneuver. A wide Kocher maneuver and mobilization of the hepatic flexure will allow medial rotation of the ascending colon to better expose the portal structures. Exposure of a retropancreatic portal vein injury may require pancreatic transection with distal pancreatectomy after the vascular repair is complete. Although portal vein ligation can be used to expeditiously manage portal vein injuries, the preferred treatment is lateral venorrhaphy, as most series report a 51% to 60% survival rate with this approach.

Hepatic artery injuries should generally be managed with ligation. However, the hepatic parenchyma must be evaluated for ischemia after ligation, especially in the presence of portal vein injury or shock. In addition, the gallbladder

should be removed if the hepatic artery is ligated. Partial extrahepatic bile duct injuries (less than 50% circumference) may be primarily repaired, with or without stenting. However, complete or complex bile duct injuries are best managed by Roux-en-Y biliary-enteric anastomosis. For the unstable patient, ligation with external drainage and delayed reconstruction is a reasonable approach.

Adjuncts to Operative Management

The original description of the multidisciplinary approach to the management of complex hepatic injuries by Asensio and colleagues described an approach consisting in immediate surgical intervention utilizing the most complex techniques in the surgical armamentarium including extensive hepatotomy, hepatography and selective deep vessel ligation, nonanatomic resection and débridement, and even hepatectomy along with packing temporary abdominal closure followed by immediate angiography and AE ligation with the ICU team present in the angiography suite to continue the resuscitationprocess. Afterward, patients were returned to the operating room after their physiologic defects were corrected for unpacking, further hepatic débridement, and nonanatomic or anatomic resection, drainage, and abdominal wall closure. Postoperative complications were treated with the use of percutaneous CT-guided drainage of hepatic collections and ERCP and stenting of major biliary leaks that were detected.

Subsequently, Asensio et al separated their results on 103 patients with AAST-OIS grade IV to V injuries in which they advocated early hepatic angiography and AE in all patients with grades IV and V hepatic injuries. Improved

survival was associated with immediate surgery to control life-threatening hemorrhage, the institution of early hepatic packing when necessary, and subsequent patient transport directly from the operating room to the angiography suite for immediate hepatic AE. Clearly, AE is essential in the management of complex hepatic injuries, whether they arise from blunt or penetrating mechanisms.

In the only prospective study in the literature Asensio et al used their multidisciplinary approach in the management of 75 AASIOIS grades IV and V complex hepatic injuries and confirmed the value of this approach as well as the value of angiography and AE and reported significant improvements in the survival rates for these injuries; 81% for grades IV and 43% for grade V. Early AE may also be useful in the multiply injured patient whose hepatic injury is being managed nonoperatively but whose serial hematocrits are noted to be dropping. Under these circumstances, the patient should immediately undergo repeat CT scanning, rather than arbitrarily receive incremental blood transfusions. If the repeat CT scan confirms that the liver injury has deteriorated and the patient remains hemodynamically stable, then AE should be attempted. Late angiography is therapeutic in the presence of hemobilia, bleeding emanating from abdominal drains in the postoperative period, and vascular abnormalities noted when follow-up CT scan is indicated.

MATERIALS AND METHODOLOGY

50 consecutive cases which are admitted in the Govt. Stanley medical college and hospital during the period of September 2018 - September 2019 are studied. It is a prospective observational study.

METHODS OF COLLECTION OF DATA

My study was collected by

1) Detailed history of the patient either directly or from the patient relatives
2) Clinical examination
3) Diagnostic investigations made to the patients

Patients admitted in the emergency surgical ward are thoroughy examined from head to foot. Patients with clinical findings of abdomen tenderness , guarding are initially resuscitated and then shifted to investigations of ultrasonography , CECT abdomen.

Head , chest and orthopaedic injuries are excluded.

Operative and non operative management mainly depends on the haemodynamical stability, clinical examination , radiological investigation CECT abdomen and pelvis

Conservative management included of strict bed rest, i.v fluids , npo depends on abdomen examination, i.v antibiotics,Analgesics.

Hemodynamically unstable patients despite the adequate fluid resuscitation and blood transfusions are shifted to operation theatre for emergency laparotomy . laparotomy findings are included.complications , outcome and duration of stay are recorded.

OBSERVATION AND RESULTS

Table -1 SEX distribution

Gender			
		Frequency	Percent
	Female	10	20.0
	Male	40	80.0
	Total	50	100.0

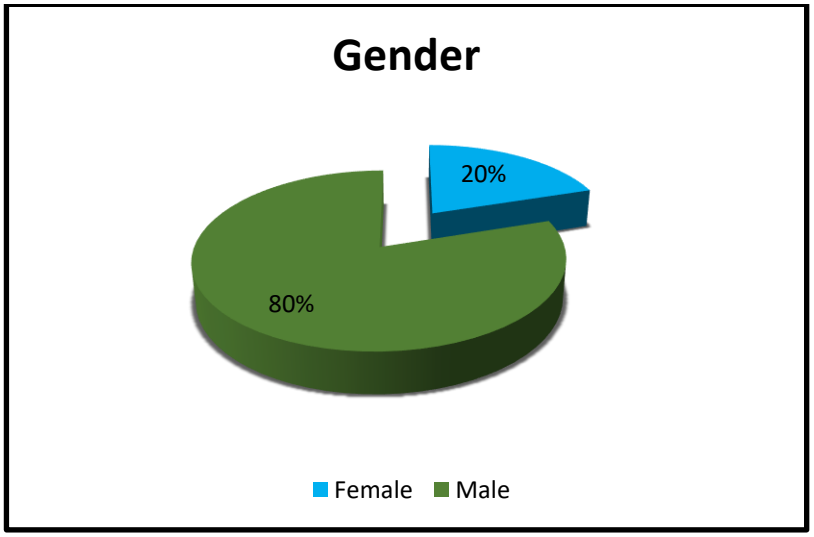


Table – 2 Age distribution

Age

	Frequency	Percent
Upto 20 yrs	8	16.0
21 - 30 yrs	13	26.0
31 - 40 yrs	11	22.0
41 - 50 yrs	8	16.0
51 - 60 yrs	4	8.0
Above 60 yrs	6	12.0
Total	50	100.0

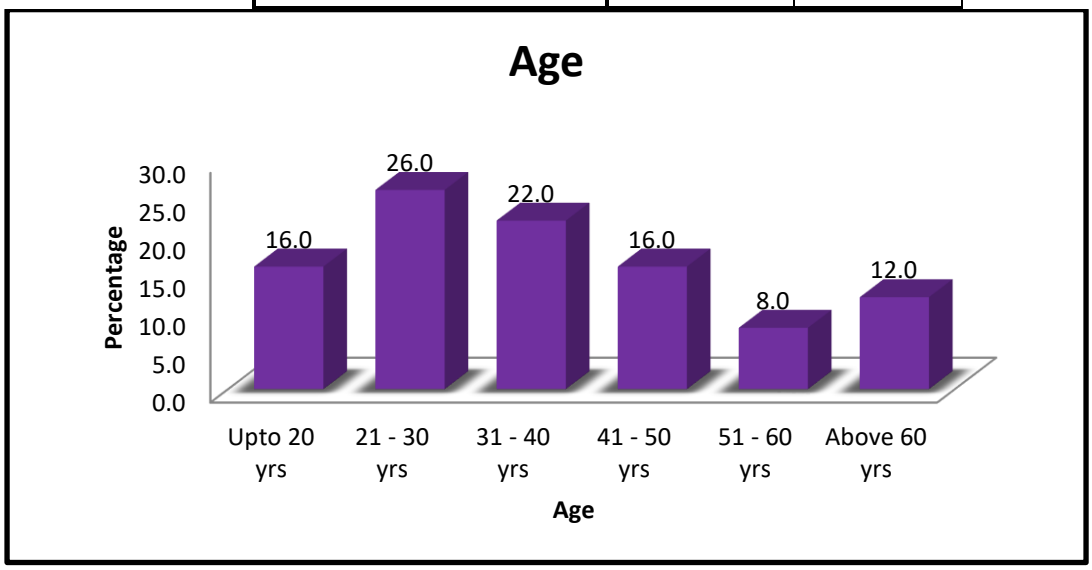


Table – 3

Mode of injury

	Frequency	Percent
BWB	13	26.0
FALL	14	28.0
RTA	23	46.0
Total	50	100.0

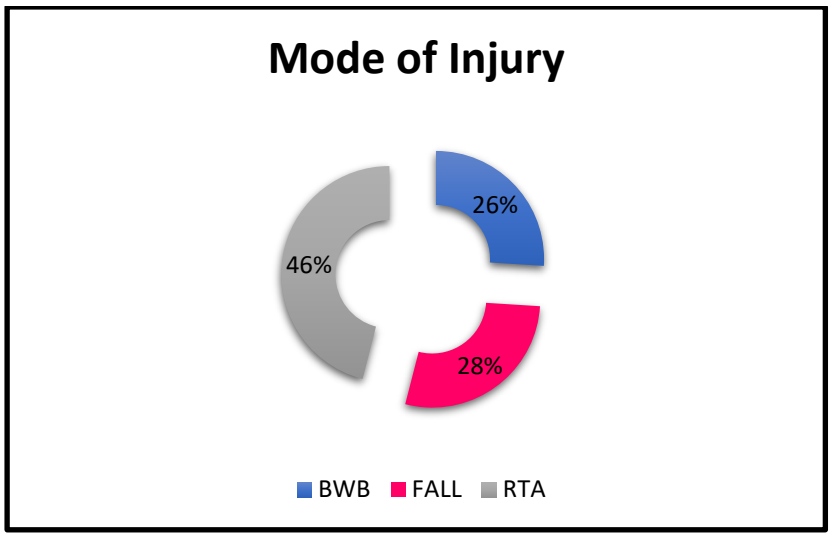


Table -4,5,6-Clinical presentation

Pain

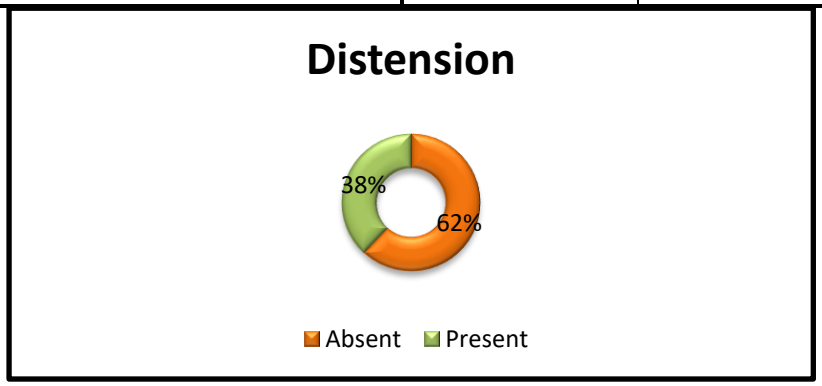
	Frequency	Percent
Present	50	100.0

Distension

		Percent
Absent	31	62.0
Present	19	38.0
Total	50	100.0

Vomiting

	Frequency	Percent
Absent	47	94.0
Present	3	6.0
Total	50	100.0



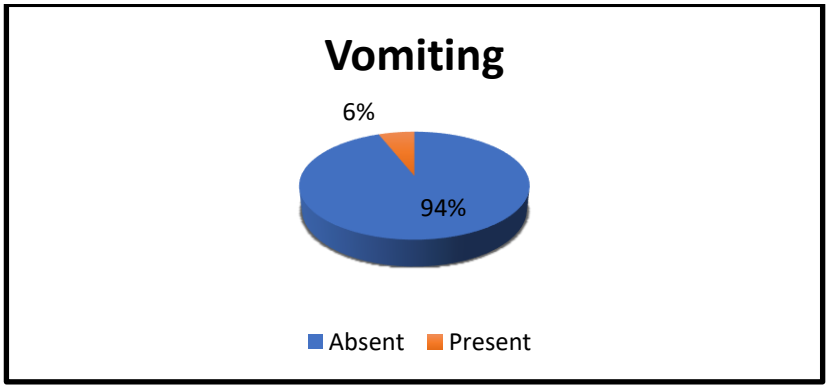


Table-7 LATENT PERIOD

	Frequency	Percent
0 – 4	14	28.0
> 4 – 8	19	38.0
> 8 – 16	16	32.0
> 14 – 24	1	2.0
Total	50	100.0

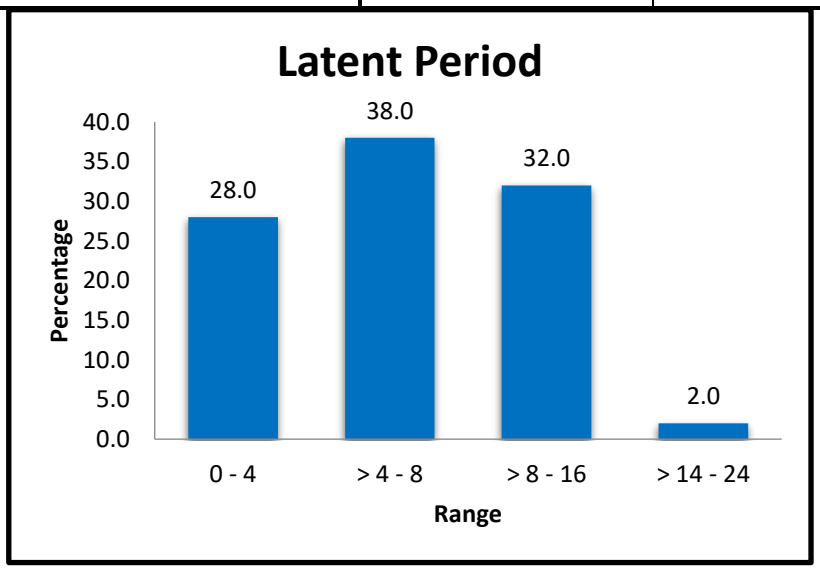


Table – 8,9,10

Tenderness

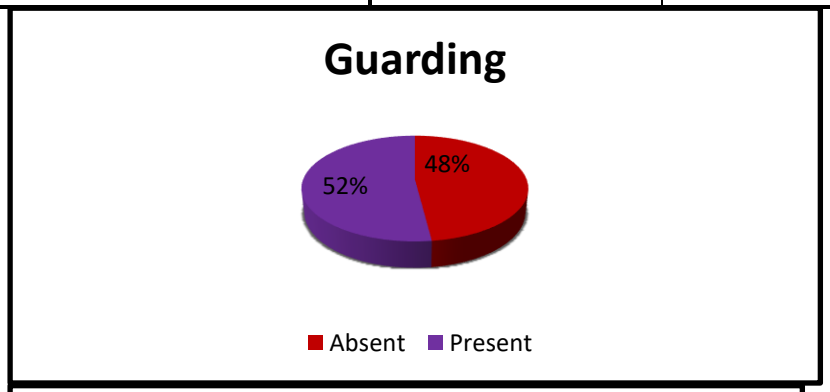
	Frequency	Percent
Present	50	100.0

Guarding

	Frequency	Percent
Absent	24	48.0
Present	26	52.0
Total	50	100.0

Rigidity

	Frequency	Percent
Absent	43	86.0
Present	7	14.0
Total	50	100.0



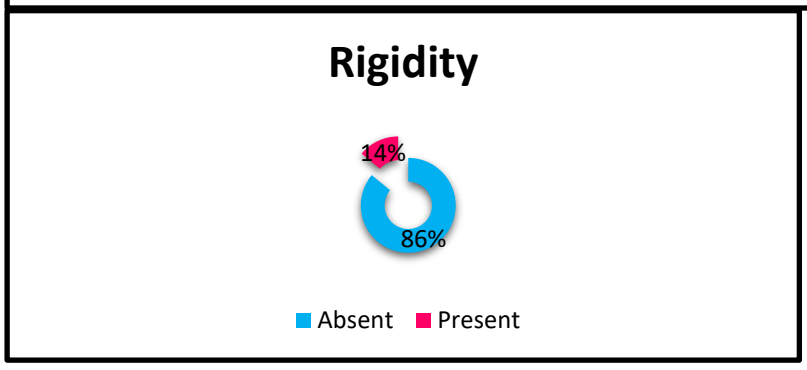


Table-11 shock

	Frequency	Percent
Absent	47	94.0
Present	3	6.0
Total	50	100.0

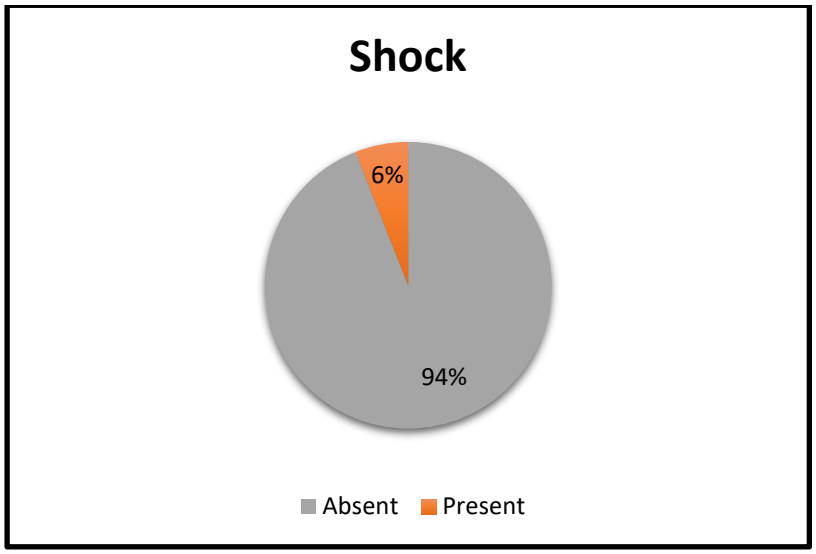


Table – 12

ULTRASONOGRAM

USG

	Frequency	Percent
N	2	4.0
HP	46	92.0
N	2	4.0
Total	50	100.0

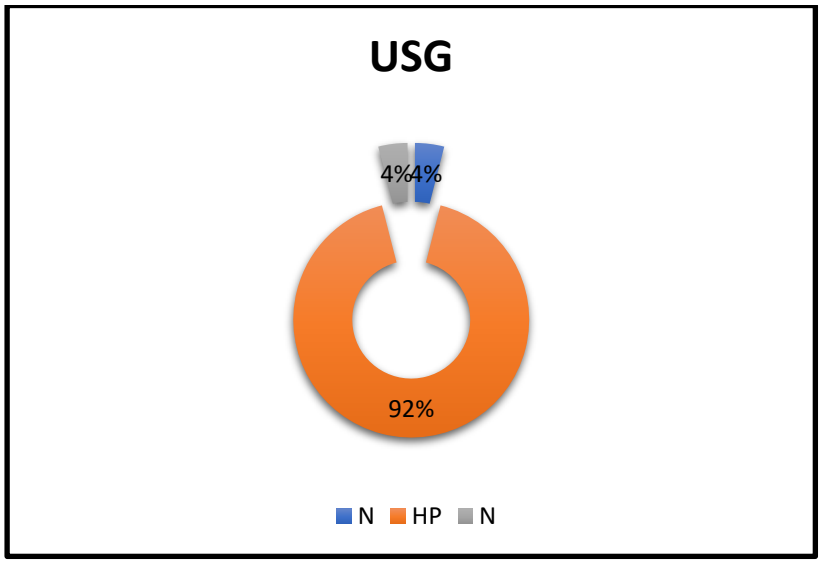


Table – 13

Grade of injury

Grade

	Frequency	Percent
1.0	16	32.0
2.0	15	30.0
3.0	8	16.0
4.0	6	12.0
5.0	5	10.0
Total	50	100.0

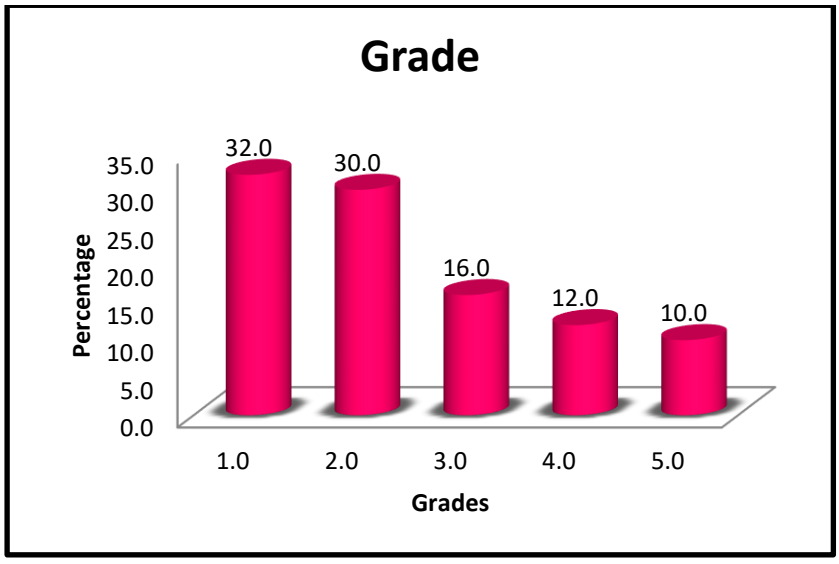


Table – 14

Management

Treatment

	Frequency	Percent
C	47	94.0
O	3	6.0
Total	50	100.0

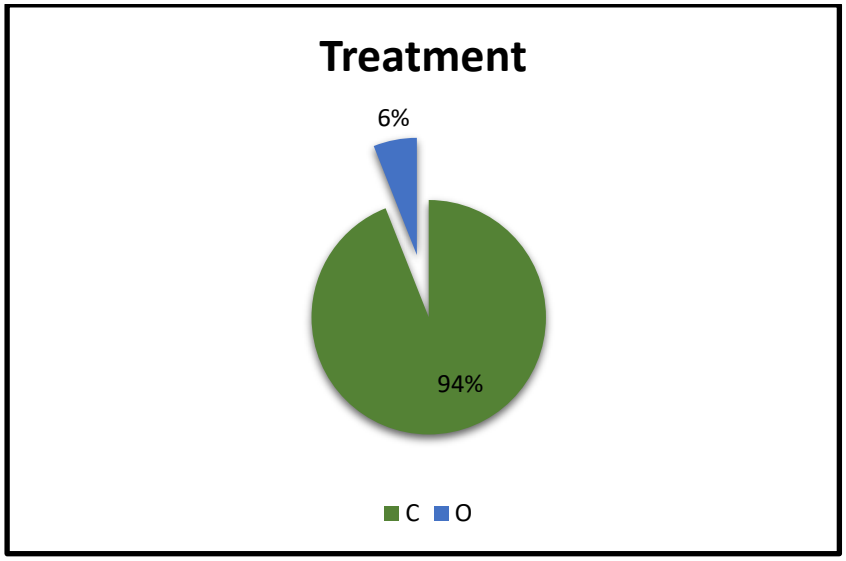


Table-15

Complications

	Frequency	Percent
Liver abscess	1	2.0
Nil	49	98.0
Total	50	100.0

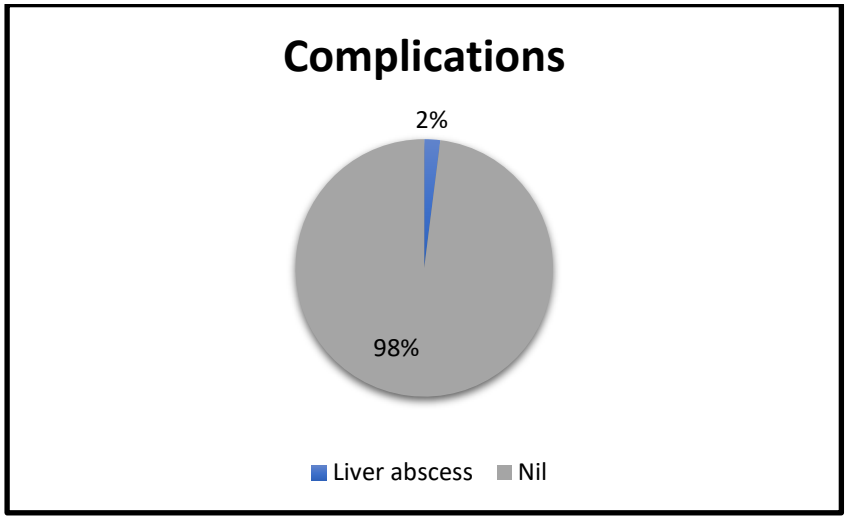


Table- 16

Outcome

	Frequency	Percent
D	3	6.0
I	47	94.0
Total	50	100.0

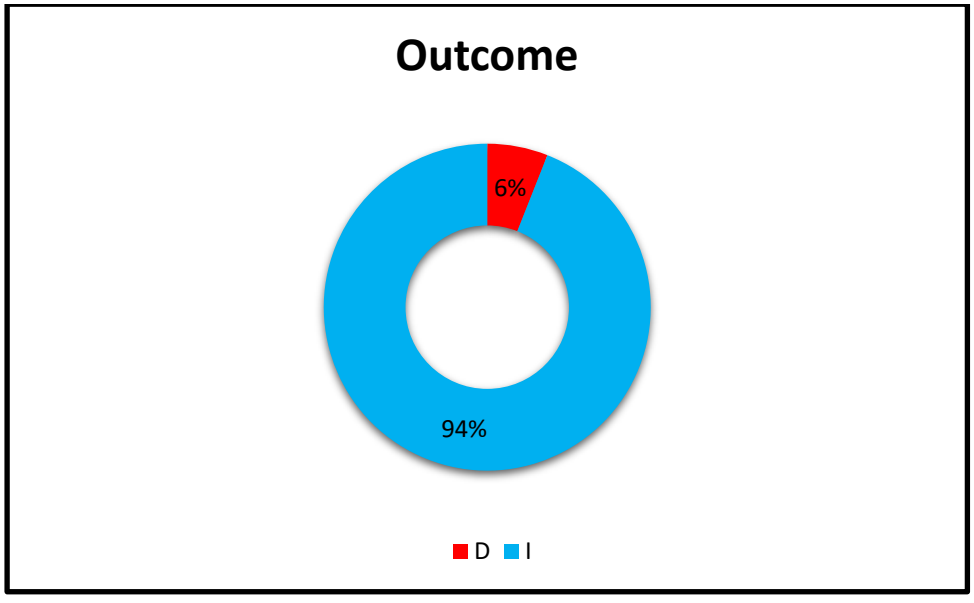


Table -17

Alcoholics

	Frequency	Percent
Absent	35	70.0
Present	15	30.0
Total	50	100.0

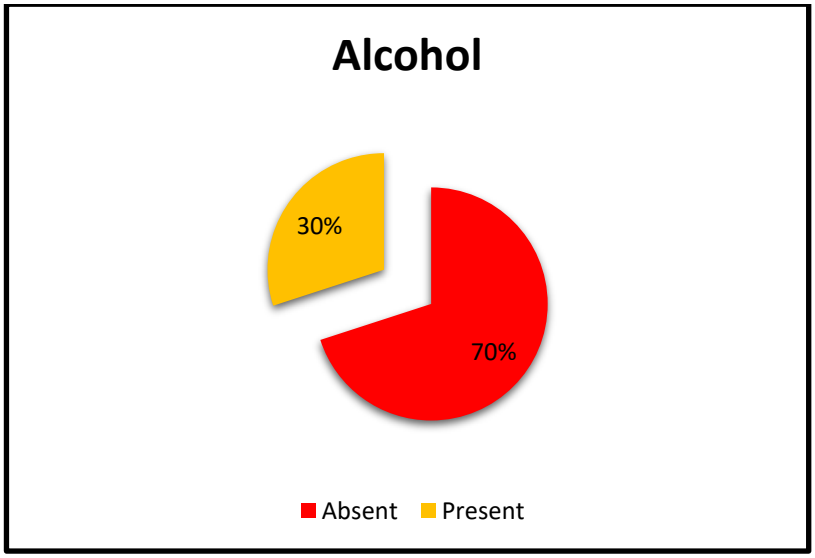


Table -18

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
Age	50	12.0	80.0	37.180	16.5439
latent period	50	2.0	22.0	7.700	4.2390
Pulse	50	82.0	128.0	106.560	10.5447
SBP	50	80.0	130.0	108.600	10.8816
DBP	50	60.0	80.0	69.400	4.2426
Hb%	50	6.0	13.2	9.942	1.5182
Duration of Stay	50	1	18	9.58	3.704
Valid N (listwise)	50				

DISCUSSION

The above mentioned findings were collected from the patients who admitted in govt. Stanley medical college which is a prospective study done between September 2018 - September 2019.

The distribution of gender showing that the males (80%) outnumbered females (20%) . Most common group of age affected are between 21-30 yrs and the less common between 50-60 yrs. The latent period in our study < 16hrs was 98%.

The most common mode of injury was the Road traffic accidents, least with blow with blunt objects. 30% of the alcoholics were affected and more prone to injury in our study.

Majority of the patients presented with pain(100 %).most of them had tenderness over abdomen. Guarding was present in 52% whereas Rigidity in 14%.

6% of them presented with shock , with polytrauma even though resuscitation made could not be saved due to increased latent period.

36% of the patients presen ted with the other associated injuries . Ultrasonography was used in all patients , showing the sensitivitiy of 92% in detecting the hemoperitoneum in our study. X ray , CECT was taken in all the patients in our study.

However CECT forms the superiority than usg, any other investigations in detecting the free fluid and solid organ injury.

The most common grade of liver injury was grade 1 liver laceration(32%), least being grade 5 (10%).Grade 6 was the rarest not visualised a case.

94% of the patients were **conservatively** managed. Only 6 % were taken for laparotomy associated with other organ injuries like spleen. Even grade 5 liver injury had been managed conservatively according to the vitals, Hb , haematocrit were constantly recorded with the serial abdominal examinations. Usg was taken after 1 week .

2% presented later with liver abscess with the one recovered from grade 5 liver laceration. The hospital duration is also increased for the grade 5 liver injury patients.

CONCLUSION

Nonoperative management can be used to successfully manage most blunt hepatic trauma patients and a select group of penetrating hepatic trauma patients. The cornerstone of nonoperative management is hemodynamic stability. An active "blush" on contrastenhanced CT mandates immediate angiography, irrespective of CT grade of injury. Successful embolization of the lesion usually permits continued nonoperative management. Should the patient under observation become hemodynamically unstable or develop peritoneal signs, operative intervention should be undertaken without the slightest hesitation. Grade 5 liver injuries are managed conservatively nowadays

When the liver injury requires operative intervention, four essential maneuvers should be kept in mind: (1) manual compression of the injury, (2) resuscitation, (3) assessment of the injury, and (4) the Pringle maneuver (inflow occlusion). These maneuvers can be lifesaving, even in the hands of those with limited experience in this area.

Complex hepatic injuries (grades IV and V) continue to challenge trauma surgeons and tax the resources of trauma centers. Most of these patients are hemodynamically unstable, have multiple associated injuries, require massive blood transfusions, and have a significant mortality rate.

There is general agreement that postobservational scanning in patients with grades I and II injuries contributes little to the clinical management of

asymptomatic patients. In patients with grades III to V injuries, repeat CT scan or ultrasound, showing resolution of the injury, can serve as an invaluable guide in identifying patients for whom critical care monitoring may no longer be necessary. The optimal time frame for follow-up CT scan in these patients, if necessary, is 7 to 10 days after the original injury.

The overall liver-related mortality rate in most large series of nonoperatively managed blunt hepatic injuries is 6%. When blunt hepatic injuries are stratified by severity, it is clear that with the exception of grades IV and V injuries, it is the associated organ injuries, specifically brain and cardiopulmonary injury, which ultimately affect mortality rates. In most large series of blunt hepatic injuries, associated brain injuries account for most (60% to 70%) of the deaths.

Most liver-related fatalities result from complex hepatic trauma (grades IV and V), especially juxtahepatic venous injuries and portal triad injuries, which often result in prohibitively high mortality rates.

Over the past 2 decades, the mortality rate of complex hepatic injuries has decreased, predominantly because of a reduction in deaths from liver hemorrhage. Responsible contributing factors include prolonged inflow occlusion times, hepatotomy with selective vascular ligation, early packing and reexploration, and adjunctive interventional procedures, especially hepatic artery AE. Although surgical managements are applicable, liver injuries are always **managed conservatively** unless there is the deteroriation of patients that needs surgical intervention.

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ANNEXURES
PROFORMA

BLUNT INJURY ABDOMEN -LIVER INJURY

Name : IP No.:
Age& Sex : Unit :
Date & Time of Admission :
Date of Discharge :
Date and Time of Injury :
Date and Time of surgery :
Latent Period :
Mode of Injury : RTA
Fall from height
Assault
Bull gore injury
Industrial accidents
Others

Presenting Complaints

1. Pain : Present / Absent
Site
Character
Duration

2. Vomiting : Present / Absent

3. Passed : Urine / Stools / Flatus

4. H/o : Hematuria / Hematochesia / Melena

5. Known H/o : DM / TB/ Epilepsy / Previous surgery / Jaundice

6. Personal R/o : Smoker / Alcoholic / Drug addiction

7. General Examination:
PR
BP
RR
Temp
Level of consciousness

8. Other symptoms
CVS

RS
Musculo skeletal system

9. Assessment of abdomen injuries:
Inspection : Abdominal wall Injury
Discolouration of abdomen wall
Palpation : Distention
Guarding / Rigidity
Tenderness
Renal angle tenderness
Percussion : Free fluid
Liver dullness
Splenic dullness
Renal angle dullness
Auscultation : BS

10. Special Signs
Spleen
Liver
Kidney
Pancreas

11. Associated Injuries:
Head & Neck / ENT
spine and pelvis
Chest
External genitalia
Extremities
Others

12. Investigations
Urine : Albumin
Sugar
Deposits
Blood : Hb% and Haematocrit
Sugar Urea
çreatinine
E1ectrolyte
Amylase
Xray : Chest PA
Abd erect

USG Abdomen and Pelvis

CT Abdomen and pelvis

Diagnostic peritoneal lavage

13. Indication for Laparotomy

14. PreopDiagnosis

15. Operative procedure

Laparotomy findings
Surgical procedure
Blood transfusion

16. Post op. Complications
Fever
Jaundice
Wound infection / dehiscence
Intra peritoneal collections
Ileus
DVT
Others
17. Post mortem Findings if expired:
18. Follow up

PLAGIARISM CERTIFICATE

URKUND

Urkund Analysis Result

Analysed Document: LOKESH THESIS.docx (D57362293)
Submitted: 10/21/2019 12:55:00 PM
Submitted By: lokeshmedico.92@gmail.com
Significance: 17 %

Sources included in the report:

LOKESH THESIS.docx (D57319904)
liver abscess.docx (D42439520)
STUDY ON BLUNT INJURY.docx (D57282218)
FAST AS A POSITIVE PREDICTOR-LIZA1.docx (D56891583)
https://aneskey.com/liver-injury/
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Instances where selected sources appear:

57

CERTIFICATE BY THE ETHICAL COMMITTEE

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01

INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK : STUDY OF CONSERVATIVE MANAGEMENT OF LIVER INJUREY IN BLUNT ABDOMINAL TRAUMA.

PRINCIPAL INVESTIGATOR : DR. A. LOKESHWARAN,
DESIGNATION : PG IN MS GENERAL SURGERY
DEPARTMENT : DEPARTMENT OF GENERAL SURGERY, GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 07.12.2018 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

CONSENT FORM

It has been explained to me in my mother tongue and I completely understand my condition, its related complications and the treatment going to be given. I have been explained in detail regarding this study- "A study of conservative management of liver injury in blunt abdominal trauma". I hereby give

my consent for my treatment and to be a part of the above mentioned study.

DATE:

PLACE:

SIGNATURE OF THE RELATIVE SIGNATURE OF THE PATIENT

S.NO	Name % ip.no	Age & sex	Mode of injury	latent period	Alcohol	pain	Vomiting	Distension	Urinary symptoms	Pulse	BP	Tenderness	Guarding	Rigidity	Bowel sounds	Associated injuries	shock	Hb%	USG	Cect abdomen	Other organinjury	Treatment	Laparotomy findings	Procedure	Complications	Duration of Stay	outcome
1	Chandra (1972122)	55/F	FALL	6	-	+	-	-	-	96	120/70	+	-	-	+	Contusion L hip	-	11	HP	Grade 1 liver laceration	-	C	-	-	-	6	I
2	Padmanabham(1865213)	28/M	FALL	4	-	+	-	+	-	110	110/60	+	+	-	-	-	-	9.6	HP	Grade 2 liver laceration	-	C	-	-	-	8	I
3	Dinesh(1865237)	26/M	RTA	2	+	+	-	-	-	84	110/70	+	-	-	+	-	-	11.3	N	Grade 1 liver laceration	-	C	-	-	-	6	I
4	Arumugam(1764670)	37/M	RTA	8	-	+	-	+	-	114	80/60	+	+	+	-	-	+	8	HP	Grade 5 liver laceration	Grade 2 splenic laceration	O	grade 5 liver laceration and grade 2 splenic laceration	L lobe hepatectomy and splenectomy	-	1	D
5	Manimaran (1868421)	18/M	BWB	4	+	+	-	-	-	98	110/80	+	-	-	+	Contusion L forearm	-	12	HP	Grade 2 liver laceration	-	C	-	-	-	8	I
6	Sharif (1765323)	56/M	FALL	3	-	+	-	-	-	108	100/70	+	+	-	-	-	-	11.8	HP	Grade 3 liver laceration	-	C	-	-	-	12	I
7	Panchavaram(1782443)	42/F	BWB	12	+	+	+	+	-	110	100/70	+	+	-	-	-	-	10.3	HP	Grade 2 liver laceration	-	C	-	-	-	10	I
8	Manikandan(1742326)	32/M	RTA	6	-	+	-	-	-	86	110/70	+	-	-	+	Contusion R hip	-	9.8	HP	Grade 1 liver laceration	-	C	-	-	-	8	I
9	Karthik(173345)	16/M	FALL	14	-	+	+	-	-	102	120/70	+	+	-	-	-	-	9.6	HP	Grade 2 liver laceration	-	C	-	-	-	7	I
10	Muthusamy(172354)	47/M	BWB	5	+	+	-	+	-	108	100/70	+	+	-	-	-	-	9	HP	Grade 3 liver laceration	-	C	-	-	-	12	I
11	Sivagam(176532)	34/F	RTA	9	-	+	-	-	-	120	110/70	+	+	-	-	-	-	9	HP	Grade 4 liver laceration	-	C	-	-	-	8	I
12	Suresh Babu(176234)	40/M	RTA	3	-	+	-	-	-	102	110/70	+	-	-	+	-	-	11.4	HP	Grade 2 liver laceration	-	C	-	-	-	10	I
13	Velmurugan(186540)	22/M	RTA	5	+	+	-	-	-	128	100/60	+	+	+	-	Contusion L leg	-	8	HP	Grade 5 liver laceration	-	C	-	-	-	18	I
14	Ajith(188823)	17/M	FALL	14	-	+	+	+	-	116	100/70	+	+	-	-	-	-	10.6	HP	Grade 3 liver laceration	-	C	-	-	-	12	I
15	Arun(198762)	25/M	BWB	8	-	+	-	-	-	104	110/70	+	-	-	+	Contusion R forehead	-	11.3	HP	Grade 1 liver laceration	-	C	-	-	-	8	I
16	Sivanesan(177541)	20/M	RTA	12	+	+	-	+	-	114	100/70	+	+	-	-	-	-	9	HP	Grade 2 liver laceration	-	C	-	-	-	10	I
17	Gopal (187623)	30/M	RTA	6	-	+	-	-	-	92	110/70	+	-	-	+	-	-	13.2	HP	Grade 1 liver laceration	-	C	-	-	-	8	I
18	Ethumalai(182331)	50/M	BWB	10	+	+	-	+	-	116	100/70	+	+	+	-	-	-	8.4	HP	Grade 4 liver laceration	-	C	-	-	-	14	I
19	Srinivasan(176116)	33/M	FALL	14	+	+	-	+	-	106	110/70	+	+	-	+	-	-	10.6	HP	Grade 1 liver laceration	-	C	-	-	-	7	I
20	Sathish(186999)	12/M	FALL	4	-	+	-	-	-	102	120/70	+	-	-	+	-	-	11.6	N	Grade 1 liver laceration	-	C	-	-	-	9	I
21	Senthil(175523)	48/M	RTA	5	-	+	-	-	-	126	80/60	+	+	+	-	Contusion L leg	+	6	HP	Grade 5 liver laceration	-	O	grade 5 liver laceration predominntly R lobe	R lobe hepatectomy	-	1	D
22	Rajesh(178994)	20/M	RTA	8	-	+	-	-	-	114	110/70	+	-	-	-	-	-	11	HP	Grade 1 liver laceration	-	C	-	-	-	8	I
23	Balaji(177732)	45/M	BWB	7	+	+	-	-	-	106	100/70	+	+	-	-	-	-	8	HP	Grade 1 liver laceration	-	C	-	-	-	7	I
24	shahin(176221)	20/F	RTA	9	-	+	-	+	-	110	110/70	+	+	-	-	Contusion R Wrist	-	10	HP	Grade 2 liver laceration	-	C	-	-	-	10	I
25	Manoj Kumar(177980)	21/M	RTA	6	-	+	-	-	-	122	100/70	+	-	-	-	-	-	8	HP	Grade 5 liver laceration	-	C	-	-	-	18	I
26	Patchaiyammal(182321)	31/F	FALL	14	-	+	-	-	-	106	120/70	+	-	-	-	Contusion L leg	-	10.4	HP	Grade 1 liver laceration	-	C	-	-	-	8	I
27	Raja(172321)	28/M	FALL	10	+	+	-	+	-	116	100/70	+	-	-	-	-	-	9.8	HP	Grade 3 liver laceration	-	C	-	-	-	12	I
28	Hasin(177214)	64/F	RTA	6	-	+	-	-	-	92	110/70	+	-	-	-	-	-	10.2	HP	Grade 1 liver laceration	-	C	-	-	-	7	I
29	kaleem(176267)	32/M	BWB	8	-	+	-	-	-	102	120/70	+	-	-	-	-	-	11.7	HP	Grade 2 liver laceration	-	C	-	-	-	9	I
30	Jahir Hussain(177012)	25/M	BWB	4	+	+	-	-	-	110	130/60	+	-	-	-	Contusion R forearm	-	8	HP	Grade 4 liver laceration	-	C	-	-	Liver abscess	16	I
31	Ganesh(176111)	17/M	RTA	9	-	+	-	+	-	108	110/70	+	+	-	-	-	-	10	HP	Grade 3 liver laceration	-	C	-	-	-	12	I
32	Madhan kumar(172877)	39/M	FALL	8	-	+	-	-	-	98	110/70	+	-	-	-	-	-	12	N	Grade 1 liver laceration	-	C	-	-	-	9	I
33	NagoorMeeran(176002)	30/M	RTA	6	+	+	-	+	-	94	120/70	+	-	-	-	-	-	11	HP	Grade 2 liver laceration	-	C	-	-	-	10	I
34	Ajay(173221)	45/M	RTA	14	-	+	-	+	-	124	100/70	+	+	+	-	-	-	8	HP	Grade 5 liver laceration	-	C	-	-	-	18	I
35	Vijay(172218)	23/M	FALL	12	-	+	-	-	-	110	110/70	+	-	-	-	-	-	12	HP	Grade 1 liver laceration	-	C	-	-	-	10	I
36	Kannan(176590)	32/M	RTA	16	-	+	-	-	-	104	120/70	+	-	-	-	-	-	10.8	HP	Grade 2 liver laceration	-	C	-	-	-	10	I
37	Ganapathy(171132)	80/M	RTA	2	+	+	-	+	-	112	100/70	+	+	-	-	Contusion R Hip	-	9.2	HP	Grade 3 liver laceration	-	C	-	-	-	12	I
38	Sethu(172241)	45/M	RTA	8	-	+	-	-	-	96	120/70	+	-	-	-	-	-	11.3	HP	Grade 1 liver laceration	-	C	-	-	-	8	I
39	Parvathi(173123)	52/F	BWB	12	-	+	-	+	-	110	100/70	+	+	-	-	-	-	9.2	HP	Grade 2 liver laceration	-	C	-	-	-	9	I
40	Ravi(1765521)	37/M	RTA	7	-	+	-	-	-	112	110/70	+	+	-	-	-	-	8.5	HP	Grade 4 liver laceration	-	C	-	-	-	12	I
41	Ahmed Basha(1768842)	66/M	FALL	10	+	+	-	-	-	94	130/70	+	-	-	+	-	-	11.5	N	Grade 1 liver laceration	-	C	-	-	-	9	I
42	Sivakumar(1763241)	43/M	FALL	4	-	+	-	-	-	108	110/70	+	+	-	-	Contusion L Hip	-	10.2	HP	Grade 3 liver laceration	-	C	-	-	-	12	I
43	Anandhavalli(1734213)	73/F	BWB	5	-	+	-	-	-	100	120/70	+	+	-	-	-	-	11.9	HP	Grade 2 liver laceration	-	C	-	-	-	10	I
44	Parveen(1792314)	65/F	FALL	4	-	+	-	-	-	122	90/60	+	+	+	-	-	+	7	HP	Grade 4 liver laceration	Grade 3 splenic laceration	O	grade 4 laceration liver , grade 3 splenic laceration	R lobe hepatectomy , splenectomy	-	1	D
45	Muthura(1726780)	58/M	BWB	3	-	+	-	-	-	82	130/70	+	-	-	+	-	-	10	HP	Grade 1 liver laceration	-	C	-	-	-	7	I
46	Mani(1734251)	30/M	RTA	4	+	+	-	+	-	104	110/70	+	+	-	-	Contusion R Hip	-	9.1	HP	Grade 3 liver laceration	-	C	-	-	-	10	I
47	Janani(1823166)	23/F	RTA	6	-	+	-	-	-	98	120/80	+	-	-	-	-	-	10.6	HP	Grade 2 liver laceration	-	C	-	-	-	8	I
48	Hariharan(1782091)	27/M	BWB	4	-	+	-	+	-	120	100/70	+	+	-	-	-	-	8.7	HP	Grade 4 liver laceration	-	C	-	-	-	16	I
49	Suresh(1820180)	32/M	RTA	22	-	+	-	+	-	110	110/70	+	+	+	-	-	-	9	HP	Grade 2 liver laceration	-	C	-	-	-	10	I
50	Ragavan(1813452)	68/M	BWB	3	-	+	-	+	-	102	100/80	+	-	-	-	Contusion L leg	-	8.5	HP	Grade 2 liver laceration	-	C	-	-	-	8	I