"STUDY OF EFFICACY, SAFETY & OUTCOME OF LAPROSCOPIC CLOSURE OF DUODENAL PERFORATION"

By
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DISSERTATION SUBMITTED TO THE
SRI DEVARAJ URS ACADEMY OF HIGHER & RESEARCH CENTRE , TAMAKA,KOLAR-563101, KARNATAKA

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
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under the guidance of
Prof. Dr. SREERAMULU P N

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DECLARATION BY THE CANDIDATE

I, hereby declare that this dissertation entitled **"Study Of Efficacy,Safety &Outcome Of Laparoscopic Closure Of Duodenal Perforation "**is a bonafide and genuine research work carried out by me under the guidance of **DR.SREERAMULU P N** M.S, **Professor of General Surgery.**

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ABSTRACT

Need for the study:

Perforated peptic ulcer is a common abdominal disease that is treated by surgery.Approximately 10 – 20 % of patients with peptic ulcer suffer a perforation of stomach or duodenum in which a chemical peritonitis develop initially from gastric & duodenal secretion but in a few hours bacterial contamination superimpose the disease.The disease could be life threatening,early diagnosis & treatment is extremely important[1]. The mortality will increase up if perforation exists more than 24 – 48 hours[2].Usually the only surgical procedure that is necessary is simple closure with or with out omental patch. When repair of perforated duodenal ulcer can be achieved by suture closure,laproscopic approach seems to be appropriate[3].This study has been undertaken for evaluating the efficacy ,safety and outcome of laproscopic surgery for perforated duodenal ulcer

METHODS

. Patients admitted to emergency ward with duodenal ulcer perforation during the period of January 2010 to January2011 at R.L.Jalappa Hospital and Research Centere.Operative duration,analgesics and antibiotics required,postoperative complications and post operative hospital stay were considered as parameters of the study.

RESULTS

A total number of 60 cases were dignosed as peritonitis secondary to duodenal perforation.Out of these 30 underwent laproscopic closure and 30 underwent open method for closure of duodenal perforation.The results of our non-randomised controlled study revealed that laproscopic repair is

associated with lesser antibiotic requirement,lesser analgesic requirement,lesser postoperative complications and postoperative stay .The operative duration was more in laproscopic procedure but did not effect the overall result.

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LIST OF ABBERIVATIONS USED

Ac.	—	Acute
BUN	—	Blood Urea Nitrogen
CO2	—	Carbon Dioxide
CT	—	Computerized Tomography
Cl	-	Chloride Ion
Df	—	
Durat	—	Duration
Fig	—	Figure
H2	—	Histamine
H2O	—	Water
H	—	Hydrogen Ion
LFT	—	Liver function test
L	—	Liver
MRI	—	Magnetic Resonance Imaging
PID	—	Pelvic inflammatory disease
Mins	—	Minutes
Meq	—	Milli Equivalents
RIF	—	Right iliac fossa
PUD	—	Peptic Ulcer Disease
PPU	—	Perforated Peptic Ulcer
Std	—	Standard
Sig	—	

INTRODUCTION

Perforated peptic ulcer is a common abdominal disease that is treated by surgery.Approximately 10- 20 % of patients with peptic ulcer suffer a perforation of stomach or duodenum in which a chemical peritonitis develop initially from gastric & duodenal secretion but in a few hours bacterial contamination superimpose the disease. The disease could be life threatening. Early diagnosis & treatment is extremely important [1].The mortality will increase up if perforation exists more than 24 – 48 hours[2] .Usually the only surgical procedure that is necessary is simple closure with or without omental patch. When repair of perforated duodenal ulcer can be achieved by suture closure,laproscopic approach seems to be appropriate[3] .This study has been undertaken for evaluating the efficacy ,safety and outcome of laparoscopic surgery for perforated duodenal ulcer.

AIMS & OBJECTIVES OF THE STUDY :

To perform the laparoscopic closure of duodenal ulcer perforation in patients with early presentation.

To study the efficacy, safety and outcome of laparoscopic surgery with open procedure.

Review Of Literature

Historical Review

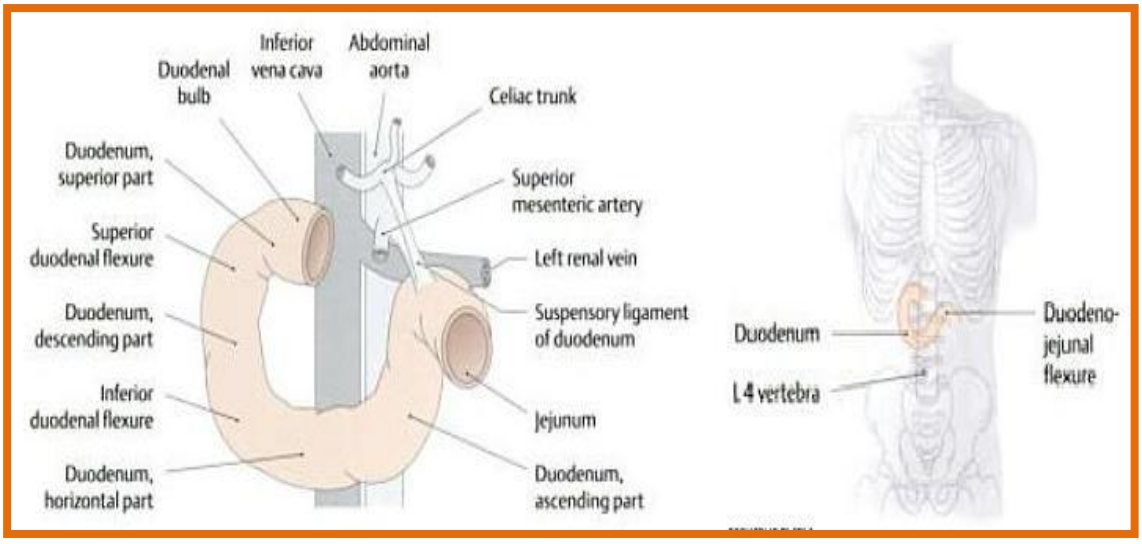
The first description of the notable clinical sign of abdominal guarding and rigidity was given by CELSUS[7].

In 1793 Joseph Penada of Padua described one of the most interesting cases. It was a case of 35 year old male butcher. Penada recorded the symptoms preceding perforation, immediately following it and late signs of generalized peritonitis. Benjamin Travers in 1817 published the first report on patients with acute free perforation of a peptic ulcer. Edward Crisp in 1843 reported 50 patients and described the clinical course of those with peptic ulcer perforation. He also suggested the possibility of operative closure of perforation but the earliest attempt at surgical closure of a perforated ulcer was by Mikulicz in 1884. This was unsuccessful and it was not until 1892 that perforated duodenal ulcer was closed successfully by Kriege. In 1894 Dean of London Hospital reported the first successful operation for perforated duodenal ulcer. He performed simple closure of the perforation. This patient who was 27 year old lady died two months later of intestinal obstruction.Braun in 1897 added posterior gastrojejunostomy in addition to simple closure. In 1902 Keetley advocated immediate gastrectomy.Collon Jones (1929) and Roscoe Graham (1937) emphasized that it is satisfactory to include three interrupted sutures: one at the top, one in the middle and one at the lower end of perforation after bringing up a portion of greater omentum and laying it over the defect. Wangensteen (1935) suggested a conservative treatment with his method of continuous gastric suction. Chemerleins (1951), Heslop et all (1951) and Nermen Taylor (1957) treated series of cases with promising results.Lister Dragested in the Forties was the first to introduce vagotomy in the treatment of peptic ulcer which has still maintained its place. Several surgeons have applied the knowledge of

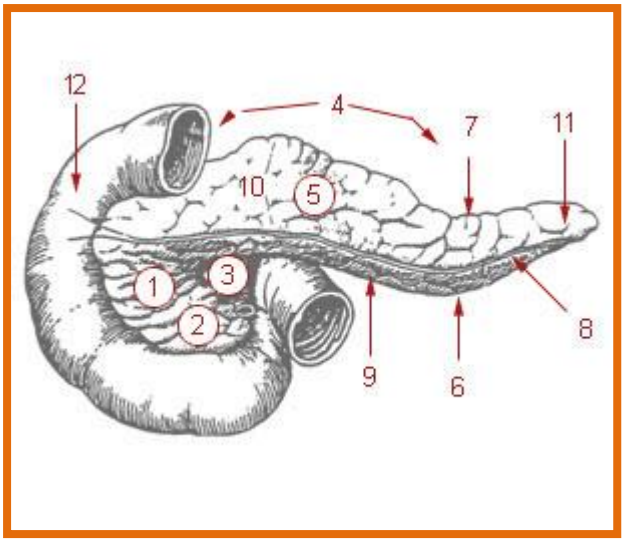
spontaneous sealing of perforation of peptic ulcer. Nasogastric suction was believed to be the key to non surgical treatment. Taylor emphasized the importance of proper positioning of the Nasogastric tube in the stomach. Both Taylor & Seeley in United states employed this nonsurgical method. Herman Taylor reported a mortality of 11 % in 235 cases treated non-surgically. In 1972 James Mc Donough and Foster stated that Plication is a satisfactory operation for most patients with perforated peptic ulcer. Primary definitive operation is indicated in patients liable to suffer continued distress after plication. Pirrondozzi et all (1957) are credited for reappraisal of primary definitive surgery with 1.3% mortality and 42.6% morbidity.Sinha, Tiwari Sharma in 1981 recommended primary definitive surgery with few criteria for selection of patients.Many other surgeons stressed primary definitive surgery e.g. Hamilton (1954), Horbrecht (1962), Wangen (1971) ,Vyavahare and Bhate (1977).Tata and Das (1976) treated perforated duodenal ulcer associated with hemorrhage with partial gastrectomy. S.K. Bannerji 1985, stated that since 90% ulcers on histological picture are chronic, so it is wise go for definitive surgery when patients are fit.Valerio, Hendry and Jones (1985) also suggested that selective definitive surgery has been proved to have acceptable morbidity and mortality. It also protects against the immediate serious complications of reperforation, hemorrhage and outlet obstruction. In the recent years it has been proven that helicobacter pylori infection plays a central role in the genesis of peptic ulcer. In India about 90% of patients with duodenal ulcer have been found to be infected with helicobacter pylori. The incidence of Helicobacter pylori infection in patients with perforated duodenal ulcers is highly variable. Ng et al reported that 50 to 75% of cases of perforated duodenal ulcer, were infected with helicobacter pylori.Sebastian et al showed that 24 out of 29 cases of duodenal ulcer perforation had helicobacter pylori infection[11].

Anatomy of the Duodenum

The duodenum is the shortest, widest and most fixed part of the small intestine. It extends from the pylorus to the duodeno-jejunal flexure. It is curved round the head of the pancreas in the form of the letter "C". The duodenum lies above the level of the umbilicus, opposite to vertebrae Ll, L2 and L3.The duodenum is mostly retroperitoneal and fixed, except at its two ends where it is suspended by folds of peritoneum, and is therefore, mobile. Anteriorly, the duodenum is only partly covered with peritoneum.The duodenum is about ten inches long. (The term "Duodenum" is derived from the Greek word, ddekadaktulos, meaning twelve fingers. The width of twelve fingers is about ten inches). The duodenum is divided into four parts. The first (or superior) part is two inches long. The second (or descending) part is three inches long. The third (or horizontal) part is four inches long. The fourth (or ascending) part is one inch long.



Duodenum And Its Location(fig 1)



Duodenum And Its Parts(Fig 2)

1)superior part – approximately 5 centimeters long

2)descending part – 7 to 10 centimeters long

3)inferior part – 6 to 8 centimeters long

4)ascending part – approximately 5 centimeters long

First Part of Duodenum are as follows:

The first part begins at the pylorus and passes backwards, upwards and to the right to meet the second part at the superior duodenal flexure. Its relations

A. Peritoneal Relations:

The proximal 1 inch is movable. It is attached to the lesser omentum above, and to the greater omentum below.

The distal 1 inch is fixed. It is retroperitoneal. It is covered with peritoneum only on its anterior aspect.

B. Visceral Relations

Anteriorly	:	Quadrate lobe of liver, and gall bladder.
Posteriorly	:	Gastroduodenal artery, bile duct and portal vein.
Superiorly	:	Epiploic foramen
Inferiorly	:	Head and neck of the pancreas.

Second part of the Duodenum

This part is about three inches long. It begins at the superior duodenal flexure, passes downwards to reach the lower border of the third lumbar vertebra, where it curves towards the left (at the inferior duodenal flexure) to become continuous with the third part. Its relations are as follows

A. Peritoneal Relations:

It is retroperitoneal and fixed. Its anterior surface is covered with peritoneum, except near the middle, where it is directly related to the colon.

B. Visceral Relations:

Anteriorly : a) right lobe of the liver ; b) transverse colon ; c) root of the transverse mesocolon; and d) Small intestine.

Posteriorly : a) anterior surface of the right kidney near the medial border b) right renal vessels c) right edge of the inferior vena cava ; and d) right psoas major.

Medially : a) head of the pancreas ; and b) the bile duct.

Laterally : right colic flexure.

The interior of the second part of the duodenum shows the following special features:

The major duodenal papilla is an elevation present posteromedial, 8 to 10 cm distal to the pylorus. The hepatopancreatic ampulla opens at the summit of the papilla.

The minor duodenal papilla is present 6 to 8 cm distal to the pylorus, and presents the opening of the accessory pancreatic duct.

Third Part of the Duodenum

This part is about four inches long. It begins at the inferior duodenal flexure, on the right side of the lower border of the third lumbar vertebra. It passes almost horizontally and slightly upwards in front of the inferior vena cava, and ends by joining the fourth part in front of the abdominal aorta. Its relations are as follows.

A. Peritoneal Relations:

It is retroperitoneal and fixed. Ifs anterior surface is covered with peritoneum, except in the median plane, where it is crossed by the superior mesenteric vessels and by the root of the mesentery.

B. Visceral Relations:

Anteriorly :(a) superior mesenteric vessels; and (b) root of mesentery.

Posteriorly :(a) right ureter; (b) right psoas major;(c) right testicular or ovarian vessels; (d) inferior vena cava; and (e) abdominal aorta with origin of inferior mesenteric artery.

Superiorly :Head of the pancreas with uncinate process

Inferiorly :Coils of jejunum.

Fourth Part of the Duodenum

This part is one inch long. It runs upwards on or immediately to the left of the aorta, up to the upper border of the second lumbar vertebra, where it turns forwards to become continuous with the jejunum at the duodenojejunal flexure. Its relations are as follows:

A. Peritoneal Relations:

It is mostly retroperitoneal, and covered with peritoneum only anteriorly. The terminal part is suspended by the uppermost part of the mesentery, and is mobile.

B. Visceral Relations:

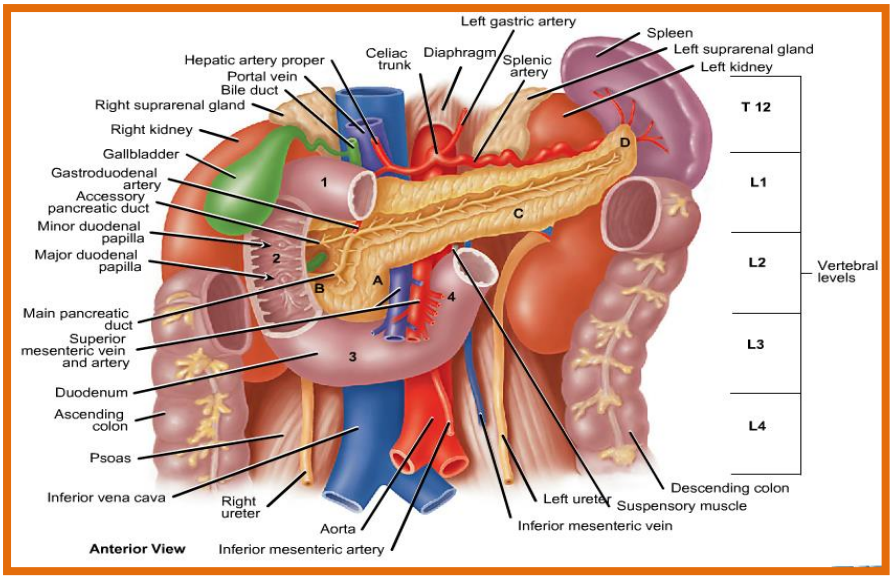
Anteriorly : (a) transverse colon; (b) transverse mesocolon; (c) lesser sac; and (d) stomach.

Posteriorly : (a) left sympathetic chain; (b) left psoas major; (c) left renal vessels; (d) left testicular vessels: and (e) inferior mesenteric vein

To the right : Attachment of the upper part of the root of the mesentery.

To the left : (a) left kidney; and (b) left ureter.

Superiorly : Body of pancreas.



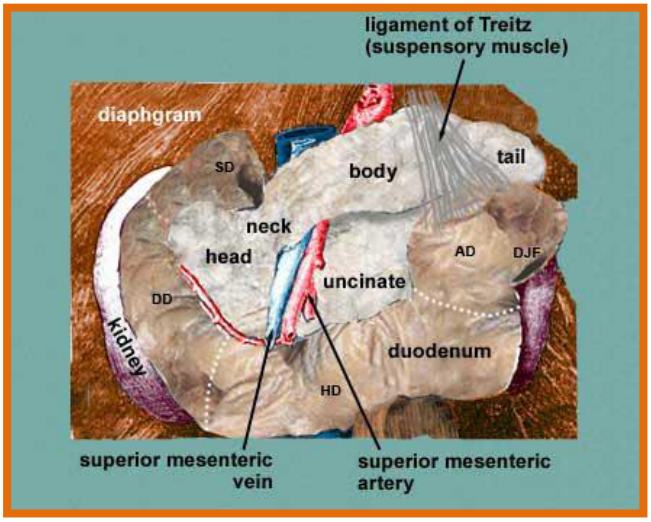
Duodenum And Its Posterior Relations(Fig 3)

Suspensory Muscle of Duodenum

This is a fibromuscular band which suspends and supports the duodeno-jejunal flexure. It arises from the right crus of the diaphragm, close to the right side of the oesophagus, passes downwards behind the pancreas, and is attached to the posterior surface of the duodenojejunal flexure and the third and fourth parts of the duodenum.

It is made up of: (a) striped muscle fibers in its upper part; (b) elastic fibers on its middle part; and (c) plain muscle fibers in its lower part.

Normally its contraction increases the angle of the duodeno-jejunal flexure. Sometimes it is attached only to the flexure, and then its contraction may narrow the angle of the flexure, causing partial obstruction of the gut.[18]



Ligament Of Trietz (Fig 4)

Arterial Supply of Duodenum

The duodenum develops from the foregut and partly from the midgut. The opening of the bile duct into the second part of the duodenum represents the junction of the foregut and the midgut. Above the level of the opening, the duodenum is supplied by the superior pancreaticoduodenal artery, and below it by the inferior pancreaticoduodenal artery.

The first part of the duodenum receives additional supply from (a) the right gastric artery; (b) the supraduodenal artery (of Wilkie) which is usually a branch of the hepatic artery; (c) the retroduodenal branches of the gastroduodenal artery; and (e) some branches from the right gastroepiploic artery[15].

Venous Drainage:

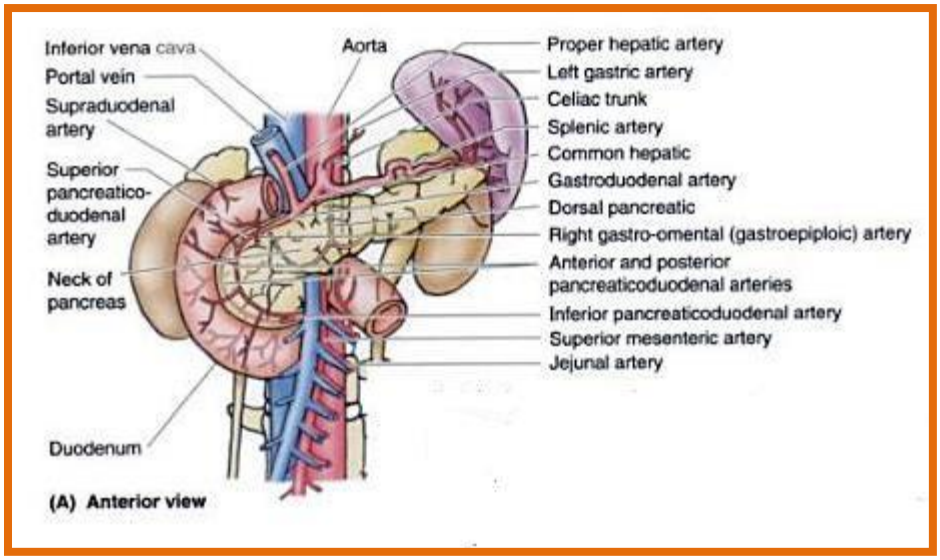
The veins of the duodenum drain into the splenic, superior mesenteric and portal veins.

Lymphatic Drainage:

Most of the lymph vessels from the duodenum end in the pancreaticoduodenal nodes present along the inside of the curve of the duodenum (i.e., at the junction of the pancreas and the duodenum). From here the lymph passes partly to the hepatic nodes, and through them to the coeliac nodes; and partly to the superior mesenteric nodes. Some vessels from the first part of the duodenum drain into the pyloric nodes, and through them to the hepatic nodes. Some vessels drain into the hepatic nodes which drains into the coeliac nodes[17].

Nerve Supply:

Sympathetic nerves from spinal segments T9 and T10, and parasympathetic nerves from the vagus, pass through the coeliac plexus and reach the duodenum along its arteries.



Blood supply Of Duodenum(fig 5)

Physiology Of Gastric Acid Secretion

Characteristic of the Gastric Secretions:

The Stomach in addition to mucus-secreting cells that line the entire surface has two important types of tubular glands: the oxyntic (or gastric) glands and the pyloric glands. The oxyntic (acid-forming) glands secrete hydrochloric acid, pepsinogen, intrinsic factor, and mucus. The pyloric glands secrete mainly mucus for protection of the pyloric mucosa but also some pepsinogen and the hormone gastrin. The oxyntic glands are located on the inside surfaces of the body and fundus of the stomach constituting the proximal 80 per cent of the stomach[18]. The pyloric glands are located in the antral parts of the stomach.

A typical oxyntic gland is composed of three types of cells: (1) the mucous neck cells, which secrete mainly mucus but also some pepsinogen; (2) the peptic (or chief) cells, which secrete large quantities of pepsinogen; and (3) the parietal cells, which secrete hydrochloric acid and intrinsic factor. Secretion of hydrochloric acid by the parietal cells involves special mechanisms as follows.

Basic Mechanism of Hydrochloric Acid Secretion:

The parietal cells contain many large branching intracellular canaliculi. When these cells secrete their acid juice, the membranes of the canaliculi open widely to empty their secretion directly into the lumen of the oxyntic gland. The hydrochloric acid is formed at the villus-like membranes of these canaliculi and are then conducted to the exterior.Different suggestions for the precise mechanism of hydrochloric acid formation have been offered. Chloride ion is actively transported from the cytoplasm of the parietal cell into the lumen of the canaliculus, and sodium ions are actively transported out of the lumen. These two together create a negative potential of - 40 to -70 mill volts in the canaliculus, which in turn causes passive diffusion of positively charged potassium ions and a small number of sodium ions from the cell cytoplasm also into the canaliculus. Thus, in effect, mainly potassium chloride but also much smaller amounts of sodium chloride enter the canaliculus.Water becomes disassociated into hydrogen ions and hydroxyl ions in the cell cytoplasm. The hydrogen ions are then actively secreted into the canaliculus in exchange for potassium ions; this active exchange process is catalyzed by Hydrogen Potassium-ATPase In addition, the sodium ions are actively reabsorbed by a separate sodium pump. Thus most of the potassium and sodium ions that had diffused into the canaliculus are reabsorbed, and hydrogen ions take their place giving a strong solution of hydrochloric acid in the canaliculus, which is then secreted into the lumen of the gland.Water passes into the canaliculus by osmosis because of the secretion of the ions into the canaliculus. Thus, the final secretion entering the canaliculus contains hydrochloric acid in a concentration of 150 to 160 meq/liter, potassium chloride in a concentration of 15 meq/liter ,and a small amount of sodium chloride. Finally, carbon dioxide, either formed during metabolism in the cell or entering the cell from the blood, combines under the influence of carbonic

anhydrase with the hydroxyl ions (formed in step 2 when water was dissociated to form bicarbonate ions). This then diffuses out of the cell into the extracellular fluid in exchange for chloride ion that enter the cell and later will be secreted into the canaliculus. The importance of carbon dioxide in the chemical reactions for formation of hydrochloric acid is demonstrated by the fact the carbonic anhydrase inhibition by the drug acetazolamide diminishes the formation of hydrochloric acid[15,17].

Pyloric Glands-Secretion of Mucus and Gastrin

The pyloric glands are structurally similar to the oxyntic but contain few peptic cells and almost no parietal cells. Instead, they contain mostly mucous cells that are identical with the mucous neck cells of the gastric glands. These cells secrete a small amount of pepsinogen, and an especially large amount of thin mucus that helps to lubricate food movement as well as to protect the stomach wall from digestion by the gastric enzymes.The pyloric glands also secrete the hormone gastrin, which plays a key role in controlling gastric secretion[14,18].

Surface Mucous Cells:

The entire surface of the stomach mucosa between glands has a continuous layer of a different type of mucous cells called "surface mucous cells". They secrete large quantities of a far more viscid mucus that is mainly insoluble and coats the mucosa with a gel layer of mucus often more than 1 millimeter thick, thus providing a major shell of protection for the stomach wall as well as contributing to lubrication of food transport.Another characterstic of this mucus is that it is alkaline. Therefore, the normal underlying stomach wall is not directly exposed to the highly acidic, proteolytic stomach secretion. Even the slightest contact with food or especially any irritation of the mucosa directly stimulates the mucous cells to secrete copious quantities of this thick, alkaline viscid mucus.

Regulation of Gastric Secretion by Nervous and Hormonal Mechanisms:

The basic neurotransmitter or hormones that directly stimulate secretion by the gastric glands are acetylcholine and histamine. All these function by first combining with specific receptors for each on the cells. Then the receptors activate the secretory processes. Acetylcholine excites secretion by all the secretory cell types present in the gastric glands, including secretion of pepsinogen by the peptic cells, hydrochloric acid by the parietal cells, and mucus by the mucus cells. On the other hand, both gastrin and histamine stimulate strongly the secretion of acid by the parietal cells but has little effect in stimulating the other cells.A few other substances also stimulate the gastric secretory cells, such as circulating amino acids, caffeine, and alcohol. The stimulatory effects of these are slight in comparison with acetylcholine, gastrin, and histamine.

Stimulation of Acid Secretion:

Nervous Stimulation *:* About one half of the nerve signals to the stomach that cause gastric secretion originate in the dorsal motor nuclei of the vagi and pass by way of the vagus nerves first to the enteric nervous system of the stomach wall and then to the gastric glands. The other one half of the nervous secretory signals are generated by local reflexes that occur entirely within the wall of the stomach itself in the enteric nervous system. All the secretory nerves release acetylcholine as the neurotransmitter at their endings on the glandular cells, with one exception: for those signals that go to the gastrin-secreting cells in the pyloric glands, an intermediate neuron serves as the final path and secrete gastrin-releasing peptide, which is probably the peptide bombesin, as the neurotransmitter.Nerve stimulation of gastric secretion can be initiated by signals that originate either in the brain, especially in the limbic system, or in the stomach itself. The stomach-initiated signals can activate two types of reflexes: (1) long vasovagal reflexes that are transmitted from the stomach mucosa all the way to

the brain stem and then back to the stomach through the vagus nerves and (2) short reflexes that originate locally and are transmitted entirely through the local enteric nervous system.

The types of stimuli that can initiate the reflexes are (1) distention of the stomach. (2) tactile stimuli on the surface of the stomach mucosa, and (3) chemical stimuli, including especially amino acids and peptides derived from protein foods or acid that has already been secreted by the gastric glands.

Stimulation of Acid Secretion By Gastrin: Both the nerve signals from the vagus nerves and those from the local enteric reflexes, aside from causing direct stimulation of glandular secretion of stomach juices, also cause the mucosa in the stomach antrum to secrete the hormone gastrin. This hormone is secreted by gastrin cells, also called G cells, in the pyloric glands. Gastrin is a large peptide secreted in two forms, a large from called G-34, which contains 34 amino acids, and a smaller form, G-17, which contains 17 amino acids. Although both of these are important, the smaller is more abundant.Gastrin is absorbed into the blood and carried to the oxyntic glands in the body of the stomach; there it stimulates the parietal cells strongly and the peptic cells as well, but to a much less extent. Thus, the important effect is to increase the rate of parietal cell hydrochloric acid secretion, often as much as eight times. In turn, the hydrochloric acid excites still additional enteric reflex activity that not only further increases hydrochloric acid secretion but also stimulates secondarily the secretion of enzymes by the peptic cells to increase two to four times.

Role of Histamine in Controlling Gastric Secretion*:* Histamine, an amino acid derivative, also stimulates acid secretion by the parietal cells. A small amount of histamine is formed continually in the gastric mucosa, either in response to acid in the stomach or for other reasons. This amount, acting by itself, causes little acid secretion.

However, whenever acetylcholine and gastrin stimulate the parietal cells at the same time, then even the small normal amounts of histamine greatly enhance acid secretion. We know this to be true becuase when the action of histamine is blocked by an appropriate antihistaminic drug such as cimetidine, neither acetylcholine nor gastrin can then cause significant amounts of acid secretion. Thus, histamine is a necessary cofactor for exciting significant acid secretion.The histamine receptors on the parietal cells are of the H2 type, not of the H1 type. Therefore, only antihistaminic drugs that block the action of histamine H2 receptors are effective in blocking stomach acid secretion. The first important drug of this type was cimetidine, but others are now available

Multiplicative Effect of Acetylcholine, Gastrin and Histamine in Stimulating Acid Secretion:

As no one of the primary stimulators of the acid-secreting parietal cells-acetylcholine, gastrin, or histamine-is effective in causing secretion of more than slight amounts of acid when functioning, alone, it has been postulated that all three of the receptors to these separate transmitter-hormonal substances must be activated simultaneously to give a truly effective stimulus for gastric acid secretion. The histamine seems to be always present under normal conditions in small amounts. Then, when the vagi are stimulated acetycholine is released at the parasympathetic nerve endings, and the gastrin-releasing peptide neurouns stimulated by the vagi cause simultaneous release of gastrin by the gastrin cells. Therefore, all three stimulants become available, and copious amounts of acid are secreted. When food in the stomach elicits enteric reflexes,this too,causes both gastrin and acetylcholine secretion,once again promoting the flow of tremendous quantities of acid.

Feedback Inhibition of Gastric Secretion in Excess Acid : When the acidity of the gastric juices increases to a pH below 3.0, the gastrin mechanism for stimulating gastric secretion becomes blocked. This effect results from two factors. First, greatly enhanced acidity depresses or blocks the secretion of gastrin itself by the G cells. Second, the acid seems to cause an inhibitory nervous reflex that inhibits gastric secretion.This feedback inhibition of the gastric glands plays an important role in protecting the stomach against excessive acidity, which would promote peptic ulceration. In addition to this protective effect, the feed back mechanism is important in maintaining optimum pH for function of the peptic enzymes in the digesting process, which is pH of about 3.0.

Phases of Gastric Secretion:

Gastric Secretion is said to occur in three phases: a cephalic phase, a gastric phase and an intestinal phase. These three phases occur simultaneously.

Cephalic Phase: The cephalic phase of gastric secretion occurs even before food enters the stomach and also while it is being eaten. It results from the sight, smell, thought, or taste of food, and the greater the appetite the more intense is the stimulation. Neurogenic signals that cause the cephalic phase of secretion can originate in the cerebral cortex or in the appetite centers of the amygdala or hypothalamus. They are transmitted through the dorsal motor nuclei of the vagi to the stomach.This phase of secretion normally accounts for about 20 per cent of the gastric secretion associated with eating a meal.

Gastric Phase: Once the food enters the stomach, it excites the long vagovagal reflexes, the local enteric reflexes, and the gastrin mechanism, which in turn cause secretion of gastric juice that continues throughout the several hours that the food remains in the stomach.The gastric phase of secretion accounts for about 70 per cent of

the total gastric secretion associated with eating a meal and therefore accounts for most of the total daily gastric secretion of about 1500 millilitres.

Intestinal Phase : The presence of food in the upper portion of the small intestine, particularly in the duodenum, can cause the stomach to secrete small amounts of gastric juice, probably partly because of the small amounts of gastrin that are also released by the duodenal mucosa in response to distention or chemical stimuli of the same type as those that stimulate the stomach gastrin mechanism. In addition, amino acids absorbed into the blood as well as several other hormones or reflexes play minor roles in causing secretion of gastric juice.

Inhibition of Gastric Secretion by Intestinal Factors : Although chyme stimulates gastric secretion during the intestinal phase of secretion, it paradoxically often inhibits secretion during the gastric phase. This inhibition results from at least two influences.

1.The presence of food in the small intestine initiates an enterogastric reflex, transmitted through the extrinsic sympathetic and vagus nerves, that inhibits stomach secretion. This reflex can be initiated by distention of the small bowel, the presence of acid in the upper intestine, the presence of protein breakdown products, or irritation of the mucosa[14,17,18].

2.The presence of acid, fat, protein breakdown products, hyperosmotic or hypo-osmotic fluids, or any irritating factor in the upper small intestine causes the release of several intestinal hormones. One of these is secretin, which is especially important for control of pancreatic secretion. In addition to having this effect, secretin opposes stomach secretion. Three other hormones-gastric inhibitory peptide, vasoactive intestinal polypeptide, and somatostain-have slight to moderate effects in inhibiting gastric secretion.The functional purpose of the inhibition of gastric secretion by intestinal factors is probably to slow the release of chyme from the stomach when

the small intestine is already filled. In fact, the enterogastric reflex and these inhibitory hormones usually reduce stomach motility at the same time that they reduce gastric secretion.

Secretion During the Inter digestive Period: The stomach secretes a few milliliters of gastric juice per hour during the "interdigestive period", when little or no digestion is occuring anywhere in the gut. The secretion that does occur is almost entirely of the so called nonoxyntic type, meaning that it is composed mainly of mucus that contains little pepsin and almost no acid.

Structure and secretory activity of Duodenum:

The mucosal surface of the duodenum and the small intestine is adapted to provide a huge area for absorption. The mucosa shows finger - like projections of about 1 mm height called villi. The villi are covered by a layer of columnar cells which themselves possess a brush border consisting of microvilli one micrometer long and 0.1 micrometer broad. Each villus in its core contains a lymphatic vessel continuous with the lymphatic plexus of the sub mucosa, smooth muscle fibers, continuous with muscularis mucosae, and an arteriole and a venule with their relavant capillary plexus. The core of the villus also contains a nerve net which has connections with the submucosal (Meissner's) Plexus.Between the villi are the intestinal glands which are also know as the crypts of lieberkuhn. These are simple tubular glands that do not penetrate the muscalris mucosae. They are lined by low columnar epithelium with goblet cells (which secrete mucus), argentaffin cells which synthesize secretin, and 5-hydroxytryptamine (a powerful stimulant of intestinal motility) an large acidophilic Paneth cells. The epithelial and Paneth cells are zymogenic - that is they produce a great variety of enzymes capable of digesting proteins carbohydrates, fats and nucleic acids They also produce enterokinase which activates trypsinogen, forming trypsin.

The duodenum, in addition to these general features of the small intestinal mucosa possesses special submucosal mucous glands which resemble the gastric pyloric glands. Known as Brunner's glands they are tortuous, long, and penetrate the muscularis mucosae. Their ducts empty into the crypts of Lieberkunhn. Numerous in the first part of the duodenum, there are few below the common opening of the bile and pancreatic ducts. They show only a small basal secretion but the ingestion of fatty foods or the injection of secretin induces a large volume of alkaline mucous secretion from them.The crypts of Lieberkuhn, which characterize the whole of the small intestinal length, continuously form the cells which migrate up the villus. These cells secrete the fluid and enzymes which form the succus entericus or intestinal juice. Enterokinase (enteropeptidase) is the most important - it activates trypsinogen. Other enzymes include maltase, invertase, dipeptidase, and alkaline phosphatase. These enzymes are located in the luminal (brush) border of the epithelial cell.About three litres of fluid (similar in electrolyte composition to extracellular fluid with a pH however of 7.60) is secreted during the course of a day. The control of this secretory activity is not nervous nor humoral but is effected by local, mechanical, and chemical stimulation of the intestinal mucosa by the presence of chyme and the food particles which it contains.The bile salts are carried back to the liver and re-excreted in the bile - the so called entero-hepatic circulation.Cholesterol is absorbed directly into lymphatics and recovered therefrom as cholesterol esters.Almost complete absorption of the products of digestion and of other materials (e.g. water, salts, vitamins) normally occurs in the small intestine.

Movements of the Duodenum:

Two different types of contraction occur: a) Rhythmic segmental contractions, b) Peristalsis.

Segmental contractions in the duodenum are of two types (i) eccentric ii) concentric Eccentric movements involve localised contraction of segments of 1-2 cm which occurs in several regions at once or sequentially. They more markedly involve the outer longitudinal muscle layer and seem to subserve a mixing function. Concentric contractions involve the circular muscle layers over a segment longer than 1-2 cms and their end result, as observed radiographically during the sequelae of ingesting a barium meal, seems to be the emptying of the barium sulphate from the duodenum. The frequency of these segmental movements during digestion is highest in the duodenum about 12 mm in man and decreases as the distance from the pylorus increases. The pacemaker cells responsible for the initiation of segmental contractions are located in the second part of the duodenum in the neighbourhood of the point of entry of the bile and pancreatic ducts. The co-ordination of these rhythms in Peristalsis consists of waves of contraction passing along the intestine, preceeded by waves of relaxation. It is usually superimposed upon the rhythmic segmental contractions and the peristaltic contraction increases the local intraluminal pressure which the segmental contraction is itself causing.

PATHOPHYSIOLOGY OF PEPTIC ULCER:

Peptic ulcers are chronic, most often solitary, lesions that occur in any level of the gastrointestinal tract exposed to the aggressive action of acid-peptic juices. They are so common in industrialised nations that, they virtually represent Stigmata of Civilization. Approxiamately 98 to 99% of peptic ulcers occur in either duodenum or the stomach in a ratio of about 4 :1. About 10% to 20% of patients with a gastric ulcer have a concurrent duodenal lesion.

Genetic influences are important in the predisposition to duodenal ulcer. Duodenal ulcers are about three times more common in first - degree relatives of ulcer patients

than in the general population. A 50% concordance for duodenal ulcers has been observed in monozygotic twins as compared with 14% in dizygotic twins. Individuals of blood group 0 are about 37%more likely to develop these lesions than those of other blood groups. Another genetic trait is the capacity to secrete mucopolysaccharide blood group substances into salivary and gastrointestinal secretions. Nonsecretors are 50% more prone to duodenal ulcers than are Secretors. An increased incidence of HLA-B5 antigen has also been identified in white males with duodenal ulcer. An elevated serum level of immunoreactive pepsinogen I has been observed in several kindreds having multiple members with duodenal ulcers. The genetic trait for pepsinogen hypersecretion segregates as an autosomal dominant and is hailed as a marker for a predisposition to duodenal ulcer.Duodenal ulcer is more frequent in patients with alcoholic cirrhosis, chronic renal failure, chronic obstructive pulmonary disease, and hyperparathyroidism. Numerous explanations have been offered to explain these associations, but all tenuous. All that can be said with any certainty is that increased serum calcium levels, whatever the cause, stimulate gastrin secretion and, therefore, acid secretion in the stomach.

Pathogenesis: Though significant advances have been made in the clinical management of peptic ulcer disease, very few inroads have been made on the understanding of its pathogenesis.In general, duodenal ulcer patients have (1) an increased tendency to secrete acid and pepsin, (2) increased responsiveness to stimuli of acid secretion, and (3) more rapid gastric emptying. However, not all these characterstics are present in every patient.

In general, duodenal ulcer patients have a higher mean basal acid output and maximal acid output than do normal controls, and significantly higher levels than are present in patients with gastric ulcers. However, in almost half the patients with duodenal ulcer

the hypersecretion is not very marked, and there is considerable overlap between the duodenal ulcer and normal groups. The acid hypersecretion can be directly correlated with an increased parietal cell mass, on average twice normal.Increased responsiveness to all known stimuli of gastric acid secretion is usually present in patients with duodenal ulcers. This is not merely a function of their greater parietal cell mass, since the maximal secretory output is disproportinate to the basal acid output. The mechanism of this increased responsiveness is unknown. It does not appear to be due to increased vagal tone, since vagotomy reduces the acid secretion in duodenal ulcer patients no more than in those with gastric ulcers.

In the recent years if has been proven that helocobacter pylori infection plays a central role in the genesis of peptic ulcer. In India about 90% of patients with duodenal ulcer have been found to be infected with helocobacter pylori. It is a micro aerophilic gram negative cocci and play an etiological role in peptic ulcers by causing the following gastroduodenal structural and functional changes.Helicobacter - Pylori infection leads to a number of perturbations of gastroduodenal structure and function.

a) Gastric structure

1) Inflammation - Cytokines, oxygen radicals etc.Trafficking of WBC's through mucosa

2)Intestinal metaplasia

3)Destruction of surface cells, parietal cells, endocrine cells

b) Gastric function

1) Increased gastrin release

2)Decreased gastrin processing

3)Possibly an increase in acid secretion

4)Hyper – pepsinogenemia

c) Duodenal structure:

1) Inflammation

2) Reduces surface area

d) Duodenal function :

1) Decreased bicarbonate secretion

2)Local acid secretion

3)Abnormal motor function

There is rapid and essentially complete reversal of histological abnormalities following surgery The incidence of Helicobacter pylori infection in patients with perforated duodenal ulcers is highly variable. Ng et al reported that 50 to 75% of cases of perforated duodenal ulcer, were infected with helicobacter pylori. Sebastian et al showed that 24 out of 29 cases of duodenal ulcer perforation had helicobacter pylori infection.

Morphology :

The gross appearance of a chronic duodenal ulcer is quite characteristic. In at least 80% of cases they are solitary lesions and in about 10 to 20% of patients with gastric ulceration there is a coexistent duodenal ulcer. About 90% of duodenal ulcers occur in the first portion of the duodenum, generally within a few centimeters of the pyloric ring. The anterior wall of the first portion of the duodenum is more often affected than the posterior wall. The classic Duodenal ulcer is a round to oval, sharply punched out defect with relatively straight walls. The mucosal margin may overhang the base slightly, particularly on the upstream portion of the circumference. The margins are usually level with the surrounding mucosa or only slightly elevated. The depth of these ulcers varies from superficial lesions, involving only the mucosa, down to deeply excavated, penetrating ulcers having their base in the muscularis. Penetration of the

entire wall may occur, and occasionally the base of the ulcer may be formed by the adjacent pancreas, omental fat, or adherent liver. The base of all peptic ulcers is smooth and clean, owing to peptic digestion of any exudate. At times, thrombosed or even patent vessels that provided the source of a fatal hemorrhage project into the base. In most peptic ulcers, Underlying scarring causes puckering of the surrounding mucosa, so that the mucosal folds radiate from the crater in spoke like fashion. The gastric mucosa surrounding an ulcer is somewhat edematous and reddened, owing to the almost invariable gastritis.

The histologic appearance varies with the activity, chronicity, and amount of healing. In the stage of active necrosis, four zones are classically demonstrable. (1) The base and margins have superficial thin layer of necrotic fibrinoid debris not visible to the naked eye; (2) beneath this layer is the zone of active nonspecific cellular infiltrate with neutrophills predominating; (3) in the deeper layers, especially in the base of the ulcer, there is active granulation tissue infiltrated with mononuclear leukocytes; and (4) the granulation tissue rests on a more solid fibrous or collagenous scar. The scarring characterstically fans out widely and may extend to the serosal surface. The vessel walls within the scarred area are characterstically thickened by the surrounding inflammation and occassionally are thrombosed. The lamina propria of the mucosa surrounding the gastric ulcer is infiltrated by plasma cells, lymphocytes and a few neutrophils.

Clinical Features:

Perforation of a duodenal ulcer into the general peritoneal cavity is a catastrophe which often occurs with dramatic suddenness, and unless correctly treated progresses in a definite manner with a typical course until the death of the patient about two or three days after the perforation. It is most important to diagnose early and treat promptly,

usually by surgical intervention. Although some cases have been and may be treated successfully without operation.

The signs and symptoms produced by the perforation vary according to the time which has elapsed since the rupture occured. There are three stages in the pathological process which can usually be recognised easily although there is no hard-and-fast limit between the stages[16].

The symptoms of each stage can be enumerated.

Early (within first 2 hours)

1. Great and generalised abdominal pain
2. Anxious countenance.
3. Livid or ashen appearance.
4. Cold extremities.
5. Cold sweating face.
6. Sub normal temperature (95 F or 96 F)
7. Pulse which is small and weak.
8. Shallow respiration.
9. Retching or vomiting (slight)
10. Pain on the top of one or both shoulders.

Intermediate (2-12 hours)

a) Vomiting ceases.

b) Less abdominal pain .

c) normal appearence, face regains normal color.

d) Normal temperature

e) Pulse normal.

f) shallow respiration

g) rigid andtenderabdominalwall

h) Tender pelvic peritoneum.

i) Diminution of liver dullness.

j) Movable dullness in flanks (sometimes)

k) Great pain on movement of body

Late **(after 12 hours)**

a) Vomiting more frequent but still not profuse.

b) Facies of later peritonitis.

c) Abdomen tender and distended.

d) Pulse rapid and small; hypovolemic shock may be present.

e) Temperature either slightly febrile or subnormal

f) Respiration labored and rapid.

Early stage (First two hours)

The initial symptoms are those due to the pain and reaction (probably due to the release ofcatecholamines) consequent on the flooding of the peritoneal cavity with gastric contents. The sudden great stimulation of the innumerable nerve terminations by the irritating fluid escaping from the ruptured viscus may be so severe that the patient may feel faint or fall down in a syncopal attack. The pulse temporarily is small and feeble, the face livid, the extremities cold, temperature about 95 F. The face shows pain and anxiety, and the patient may cry out in his agony.

The site of the initial pain is generally epigastric, but quickly it extends downwards, and in a short time is felt all over the abdomen. This stage may last for but a few minutes or persist for an hour or two. Its length depends to a certain extent upon the size of the perforation and the degree two which the general peritoneal cavity is flooded.

In cases where the perforation is very small and soon sealed up by fibrinous exudate the symptoms of onset are correspondingly less severe.

Intermediate Stage (2-12 Hours)

The intensity of the initial pain subsides, and the patient then looks better and feels more comfortable. The circulatory system recovers to such an extent that the limbs may become warmer, the face normal in colour, and the pulse normal in frequency, and strength, while the thermometer may show no indication either of subnormality or fever. The improvement in symptoms does not imply any stoppage of the pathological process, though the casual observer might easily think that real improvement was taking place. Upon the proper appreciation by the practitioner of this dangerous latent period depends the patients chance of recovery from the disease. It is in this stage that the inexperienced house surgeon thinks he has made a mistake in summoning the surgeon so urgently, and almost apologizes for having brought him up needlessly. But it is at this period that the favourable opportunity for operation passes, nor should there be any difficulty in diagnosis if careful examination is made.No certain guide is to be obtained from the pulse and temperature, for they are frequently normal, nor is the patients own opinion of his condition always to be trusted, for he often expresses himself as feeling much better, and he may even begin to think lightly of his condition. But his attitude and his acts will always belie his words. Relief will be sought by the drawing up of the legs, and if he is asked to turn over in bed the attempt is made cautiously and with evident dread of increasing the pain. If no morphine has been administered there will still be complaint of generalised abdominal pain, though the intensity will not be so great as at first. There are in addition five observations, some or all of which give valuable indication of the serious intra-abdominal mischief. The

abdominal wall is rigid and tender, respiration shallow and of costal type, the pelvic peritoneum is tender, and there may be free fluid and free gas in the peritoneal cavity.

The rigidity of the abdominal wall is an almost constant feature. The muscles are flat and board-like, and even firm pressure cannot make them give way. It takes a fairly deep anaesthesia to cause them to relax. Pressure on any part of the abdominal wall causes pain, and may evoke retching. Tenderness is often greater in the right iliac fossa in the case of ruptured duodenal or pyloric ulcer. The rigid muscles do not move on respiration, and the movement of the diaphragm is also considerably inhibited so that breathing is shallow and of the costal type.

The tenderness of the pelvic peritoneum is a most important sign. This can be determined by a rectal or,in the female, by a vaginal examination. Within a very short time of a perforation the pelvis fills with escaped contents and inflammatory exudate, and, though no lump can be felt, pressure against the pelvic peritoneal pouch through the rectal wall by the inserted finger produces pain which makes the patient wince. However, the tenderness of the pelvic peritoneum is not always present if the case is examined within an hour or two of perforation and if the opening is small or quickly sealed up.

Movable dullness in the flanks due to free fluid in the peritoneal cavity should usually be determinable, but the shifting of the patient necessary to elicit the sign is not always advisable. In doubtful cases it may be of value.

The diminution or absence of liver dullness is the sign produced by free gas in the peritoneal cavity. It is often easily demonstrated but is frequently ambiguous. Percussion over the front of the liver may produce a resonant note even when no free gas is present in the peritoneal cavity, for it may result from distended intestine which is sometimes pushed up in cases of intestinal obstruction or peritonitis from any cause. If

there is no abdominal distension, however, diminution of the liver dullness anteriorly is significant. It is always of significance to obtain resonance on percussion over the liver in the midaxillary line. If in any acute abdominal case distinct resonance is obtained over the liver in the midaxillary line about two or more inches above the costal border, one is certainly dealing with perforation of gastric or duodenal ulcer. However, it is only in the minority of cases that the sign is positive.

It is possible to get great help from a simple radiograph of the diaphragmatic region. By this means small quantities of free gas between the liver and diaphragm may be observed but in 15-20 percent of cases of perforated ulcer, no free gas can be demonstrated.

An additional symptom which may be helpful is the occurence of pain on the top of the shoulder, in the supraspinous fossa, over the acromion, or over the clavicle, that is, the region of distribution of the cutaneous branches of the fourth cervical nerve. This symptom, if present, has to be considered carefully with the other indications, for diaphragmatic pleurisy causes similar pain; but if the pain is felt on both shoulders from the onset of the attack it is suggestive of a perforation of the anterior wall of the stomach causing irritation of the median portion of the diaphragm. In the case of perforation of a pyloric or duodenal ulcer the shoulder pain is usually felt in the right supraspinous fossa.

Late Stage (After 12 hours)

This follows quickly after the previous stage.Locally the extensive peritonitis is clearly shown by increasing distension of the abdomen. Distension of the abdomen is not a sign of a perforated ulcer – it is an indication that peritonitis is advanced, and that the condition has been allowed to proceed too far.

The other effects of extensive peritonitis are increasing and persistent vomiting, gradual increase in rate and depreciation in force and volume of the pulse, and the consequent decrease in temperature of the extremeties and body generally.The abdomen remains tender, but in late peritonitis the rigidly frequently lessens. Finally, as a result of the vomiting and depressed circulation, the face becomes pinched and anxious, the cheeks hollow and the eyes dim and beringed with dark circles, the so - called facies Hippocratica, which is not so much a sign of peritonitis as the mask of death following peritonitis.

Diagnosis & Differential Diagnosis:

During the initial stage it is nearly always possible to say that there is a condition needing surgical intervention, though the exact nature of the catastrophe may be slightly doubtful. Great help is sometimes obtained from a previous history of chronic indigestion or of duodenal pain, coming on about two hours after food. Quite a number of patients, however, give but a recent history of pain after food. This is more common in the case of young people in whom acute pyloric ulcers appear to be not uncommon.If, in one who has been subject to chronic indigestion, sudden collapse and very severe abdominal pain suddenly supervene, and if at the same time the abdominal wall becomes generally rigid,, one is justified in suspecting a perforation of an ulcer. If in addition the pelvic peritoneum is tender, and there is resonance over the lateral aspect of the liver, diagnosis is certain.

In the second stage the general symptoms temporarily improve, but all the local signs remain or become still more definite, so that the careful observer should not be misled.

In the third stage, there is no difficulty in diagnosing that some serious catastrophe within the abdomen has occurred.In some instances in which the diagnosis is in doubt even after a careful history, examination, and plain films of the abdomen, the oral

administration of gastrograffin may demonstrate a perforation or exclude its presence. Such studies are not often necessary, but may be helpful in late cases, especially if the question of sealing of the perforation has arisen.

Differential Diagnosis:

There are three conditions sometimes giving rise to symptoms similar to those of perforated ulcer, which either do not call for operation or in which operative interference is positively contraindicated. They are: a)Severe colic (either biliary or renal)

b)Some cases of pleuropneomonia or pulmonary infarction.

Colic : Biliary and renal colic may cause severe collapse and terrible abdominal pain. The extent of the collapse is not a differential point, since in biliary colic the patient may sometimes appear in extremis, but diagnosis is usually clear on a consideration of the previous history and careful observation of the condition of the abdominal wall, the liver dullness, and the pelvic peritoneum. A clear account given to prior attacks of pain and jaundice, or hematuria and the passing of gravel or a small stone, would serve to indicate the probability of a stone trying to pass down the biliary ducts or ureter respectively.

The radiation of the pain of billiary colic to the subscapular region, and that of renal colic to the testicle, are sufficiently diagnostic. In uretral stone colic the abdominal wall is not usually rigid, and the sufferer may throw himself about or writhe in agony while attempting to gain a more easy position. After perforation of an ulcer the general abdominal rigidity and increase of pain on movement forbid and prevent movement, though occasional exceptions occur. Finally the pelvic peritoneum is not tender nor is there any diminution of liver dullness in biliary or renal colic. If jaundice or hematuria

is observed, diagnosis will not be in doubt. The pain of renal colic is nearly always strictly limited to one side.

Tabes dorsalis : The gastric crises of tabes dorsalis (now rarely seen) may give rise to difficulty in diagnosis, for the intensity of the abdominal pain and the severity of the vomiting may cause extreme collapse. It should be a rule to always test the knee - jerks and the pupillary reactions in every acute abdominal case, for in tabes one or other of these is nearly always abnormal. Persisting rigidity of the abdominal wall is never due to tabes, and tragic misdiagnosis may occur if this point is not remembered[17].

Right sided or bilateral pleuropneumonia or pulmonary infarction :

An acute case of bilateral or right-sided pleuropneumonia or pulmonary infarction will sometimes cause considerable abdominal rigidity, and great epigastric pain, but in such there are usually sufficient signs in the lung to point to the true cause of the condition. The alae nasi will be working and the respiration rate will be distension. With pleauropneumonia there is usually fever and a raised pulse rate. Once more rectal examination and percussion over the lateral aspect of the liver are of importance in diagnosis.

There are six other conditions which are sometimes difficult to distinguish from a perforated gastric or duodenal ulcer and which are more commonly confused with perforated ulcer than the above three conditions. The are.

1. Acute pancreatitis.
2. Acute perforative appendicitis
3. Ruptured ectopic gestation (in women)
4. Acute intestinal obstruction.
5. Postemetic rupture of the esophagus.

To these should be added the rarer conditions of mesenteric thrombosis and dissecting aneurysm. When the diagnosis of acute pancreatitis is quite clear most surgeons now reserve operation until the acute symptoms have subsided and special indications appear.

Acute pancreatitis : This simulates visceral perforation very closely, and before the abdomen is opened is quite often mistaken either for that condition or for intestinal obstruction. In pancreatitis the pain may be even more agonizing, but the abdominal rigidity is not so generalised nor so constant. Cyanosis and slight jaundice are more often seen in pancreatitis, which often occurs in obese subjects.

Acute appendicitis : This should easily be distinguised by consideration of the history, the order of the symptoms, and the local signs. It is infrequent for inflammation of the appendix to cause such acutely severe symptoms as those ushering in a gastric perforation, but in the second stage, a perforated ulcer may be, and often is, misdiagnosed as appendicitis. Especially is the case of a leaking duodenal ulcer, for the escaped contents may trickle down chiefly on the right side of the abdomen and cause pain, particularly in the right side of the abdomen and more commonly in the right iliac fossa. This simulates appendicitis closely, for the sequence - epigastric pain, nausea and vomiting right iliac pain and fever - may be produced just as in inflammation of the appendix. The intensity of the initial collapse may serve to distinguish them and the persistence of tenderness over the duodenal area should help to determine, the condition. In appendicitis the abdominal rigidity is seldom so extensive as in perforated ulcer, and the liver dullness is normal, though in both cases there may be rectal tenderness, In many cases of perforated duodenal ulcer, the patient may complain of pain on the top of the right shoulder or over the right supraspinous fossa : very rarely is shoulder pain felt in appendicitis, and when felt (due to irritative fluid reaching the

diaphragm) the pain would be more likely felt over the acromion or clavicular region. In both cases operation is indicated.

Intestinal obstruction : This should not give rise to difficulty save in those cases which come late for diagnosis. Acute strangulation of a coils of small bowel is the most likely type to cause difficulty. In both conditions the onset may be with acute symptoms of collapse, pain and vomiting, and in both there may be evidence of free fluid in the abdomen. But in obstruction the abdominal wall is usually flaccid and quite unlike the rigid boardlike condition in perforated ulcer. In acute obstruction vomiting is almost from the beginning a distinctive feature, and the character of the vomit gradually changes until it is feculent. If, however, a coil of small intestine is acutely strangulated and lies in contact with the anterior abdominal wall, there may be tenderness on pressure and sometimes rigidity of the overlying muscle.

In the late stages of both conditions it may be difficult to distinguish between them, for peritonitis is often a complication of late intestinal obstruction, and the boardlike rigidity accompanying a perforated ulcer tends to diminish somewhat as the distension increases, In such cases the history, and possibly the character, of the vomit may serve to differentiate them.

Ruptured ectopic gestation : Rupture of an ectopic gestation, leading to severe intraperitoneal haemorrarge, may cause syncope and collapse, vomiting, and severe abdominal pain. A history of menstrual irregularity may be obtained, but one must not rely on that for diagnosis. The main points in diagnosis are the blanching of the lips, tongue, nails, and the absecnce of true abdominal rigidity, though the abdomen is generally tender and tumid, especially in the lower part. In both cases there will be some tenderness on digital rectal or vaginal examination. No definite pelvic swelling can be made out in most cases of recent rupture of an ectopic gestation. Free fluid in the

abdomen may be detectable in both conditions and resonance over the front of the liver may be obtainable sometimes with ruptured ectopic gestation (due to intestine pushed up by clots of blood), but resonance over the lateral aspect of the liver is only obtained by a perforated ulcer.

Pain over the clavicles or in the supraspinous fossa in sometimes a complaint in cases of ruptured extrauterine gestation as with perforated ulcer. This is due to diaphragmatic irritation by the clotted blood in the upper abdomen.

Dissecting aneurysm : A dissecting aneurysm of the aorta may cause acute abdominal pain. Collapse and rigidity of the muscles of the abdominal wall, and has on occasion given rise to diagnosis of a perforated peptic ulcer, but the thoracic origin of the pain, the absence of free gas in the peritoneal cavity and the reductionor absence of the pulse in one of the lower limbs should serve to differentiate.

Peritonitis from other causes : Other forms of peritonitis can only be distinguised from that due to a perforated ulcer by considering the history of onset, and by determining the presence or absence of gas on the lateral aspect of the liver. It may be impossible to differentiate from the results of perforation of some other part of the gut. Peritonitis due to rupture of the gallbladder may be accompanied by an icteric tinge in the conjunctivae.

Postemetic rupture of the esophagus : The history is the most important differentiating feature because the pain of this condition almost invariably follows forceful retching and vomiting. Occasionally, after the initial retching, hematemesis may be evident just prior to the development of the pain. The pain is high in the epigastrium and often radiates through to the lower thoracic spine. It is usually aggravated by lying flat, and is relieved somewhat by sitting. Dyspnea is relatively common and crepitus may be detected in the cervical and supraclavicular regions. The

abdomen may appear to be somewhat guarded but true rigidity and rebound tenderness are absent. Evidence of pneumothorax or pleural effusion may be found by physical examination and on the X-ray. The diagnosis of post emetic rupture is relatively simple if it is considered, and it can be confirmed by films while the patient swallows a water soluble dye.

Rupture of an ulcer with formation of localized subphrenic abscess :

When for one reason or another previous adhesions with slow leakage allows time for deposition of fibrin, the escaping gastric contents do not flood the peritoneal cavity. The symptoms are correspondingly modified. The pain may be very great, but the initial collapse is not so dramatic and the abdominal signs will soon localize themselves to the upper segment of the abdomen and lead to the development of a subphrenic abscess containing gas. If such an abscess develops anteriorly, the lcoal signs of intraperitoneal suppuration are very evident, but when the mischief is high up under the diaphragm the signs and symptoms take longer to develop. Irregular temperature rigors, leucocytosis, and dullness at the base of the lung consequent on pleural effusion or basal congestion will lead the observer to diagnose a collection of pus under the diaphragm.

It must be remembered that occassionally a duodenal or pyloric ulcer may perforate, but the perforation may soon be sealed by a fibrin deposit. Such cases give rise to pain, rigidity, and tenderness in the right hypochondrium closely simulating the symptoms of acute cholecystitis, and the condition may clear up without the formation of abscess. On the other hand, such a sealed perforation may become unsealed and cause a recrudescence of the acute symptoms.

Perforation into the lesser sac : Frank perforation of an ulcer into the lesser sac of peritoneum is not common. Usually adhesion takes place to the posterior wall of the sac, and the ulcer simply erodes into the pancreas. When, however, frank perforation does take place, there is initial acute pain in the upper abdomen, but rigidity of the upper abdomen does not result unless the fluid escapes from the foramen of Winslow into the general peritoneal cavity. An abscess in the lesser sac is likely to follow, giving rise to fever and the usual signs of suppuration and formation of one type of subphrenic abscess.

TREATMENT:

Treatment of duodenal ulcer perforation may be classified as

a) Non-operative:

In past studies, comparing operative and non-operative therapy, the former was shown to improve survival following perforation. Nevertheless, in some patients, non-operative treatment may be employed safety. Indications and relative contraindications are shown in the table.

Criteria for Nonoperative Management of Perforated Duodenal Ulcer

Relative indication

a) Sealed ulcer on gastrograffin upper GI series
b) No peritoneal signs
c) Multiple medical illness
d) > 24 hours since the onset of symptoms

e) Relative contraindication

1. Chronic ulcer history
2. Gastric ulcer Peritonitis

3. Diagnostic uncertainty
4. Perforation while on adequate medical therapy

The principle involved is the tendency for perforation to become sealed either by omentum or by adherence to the undersurface of the liver. Thus, if the stomach is kept empty a spontaneous closure may be achieved. The details are:

1) Effective gastric aspiration: For this to be achieved, a radio-opaque tube must be shown (if facilities permit) to be well positioned in the body of the stomach. Aspiration is carried out by hand at half-hourly intervals so that there is no risk of blockage being prolonged for more than 30 minutes. The nurse or other attendant should be instructed to clear the tube by the injection of 5 ml of air before aspiration.

2)Hourly pulse and temperature chart as for the non-operative treatment of an appendix mass.

3)Repeated chest radiography to assess

a)The dimensions of sub diaphragmatic gas.

b)The progress of basal pulmonary complications.

c)The usual attention to fluid balance.

Opiates should not be needed after an initial dose of 10-15 mg morphine intravenously followed by the same amount over 6-8 hours as a continuous infusion. If pain is of sufficient severity after 4-6 hours to indicate a repeat dose, then laparotomy should be considered. Although in early cases there is unlikely to be benefit from antibiotics, in later cases or those with established pulmonary disease antibiotics may be necessary.

Successful treatment by gastric aspiration is associated with gradually decreasing upper abdominal tenderness and rigidity, falling pulse rate and a diminution in pneumoperitoneum. Indicators for operation are the reverse of these. The method is not recommended for routine use, but has an undoubted place in selected situations.

B) Operative:

1)Simple closure:

Simple closure of the perforationcan be performed by interrupted sutures without omentoplasty or (free) omental patch. simple closure of the perforation with a pedicled omentum sutured on top of the repair, respresenting omentoplasty. A pedicled omental plug drawn into the perforation after which the sutures are tied over it and fnally the free omental patch named after Graham. The repair can be tested by either filling the abdomen with warm saline and initiating some air into the nasogastric tube. If no bubbles appear, the perforation has been sealed appropriate. Also dye can be injected through the nasogastric tube[37]. Thorough peritoneal toilet followed is then performed. A drain is not routinely left[38]. The abdominal wound can be infiltrated with bupivacaine 0.25% at the end of the procedure

Omentoplasty or omental patch: necessary or not? Cellan-Jones published an article in 1929 entitled "a rapid method of treatment in perforated duodenal ulcers". Treatment of choice at that time was, after excision of friable edges if indicated, the application of purse string sutures and on top an omental graft[40]. An encountered problem was narrowing of the duodenum. To avoid this, he suggested omentoplasty without primary closing of the defect. His technique consisted of placing 4-6 sutures, selecting a long omental strand passing a fine suture through it.

Different suture technique used for closing perforation-

A. Primary closure by interrupted sutures
B. Primary closure by interrupted sutured covered with pedicled omentoplasty.
C. Cellan-Jones repair: plugging the perforation with pedicled omentoplasty
D. Graham patch: plugging the perforation with free omental plug.

The tip of the strand is then anchored in the region of the perforation and finally the sutures are tied off . It was not until 1937 that Graham published his results with a free omental graft[40]. He placed three sutures with a piece of free omentum laid over these sutures, which are then tied. No attempt is made to actually close the perforation[40]. The omental graft provides the stimulus for fibrin formation. His approach has been the golden standard since[42]. Very often surgeons mention they used a Graham patch, but they actually mean they used the pedicled omental patch described by Cellan-Jones[41]. Schein could not have outlined it any clearer: "Do not stitch the perforation but plug it with viable omentum and patch a perforated ulcer if you can, if you cannot, then you must resect"[11].

Irrigation of the peritoneal cavity Although some surgeons doubt the usefulness of irrigation, nothing has been found in literature supporting this theory. General it is reflected on to be one of the most important parts of the surgery and irrigation with 6-10 litres and even up to 30 litres of warm saline are recommended[23]. However the rational for routinely use of intra-operative peritoneal lavage seems to be more a historical based custom lacking any evidence based support[23,32].

Drainage or not-there seems to be no unanimity of opinion on this topic[37]. In a questionnaire 80% of the responders answered that they would not leave a drain[37]. A drain will not reduce the incidence of intraabdominal fluid collections or abscesses[37]. On the other hand the drain site can become infected (10%) and can cause intestinal obstruction[38]. Often a drain is left as a sentinel. However, in case of suspected leakage a CT- scan will provide all the information needed, probably better than a non-productive drain.

2) Definitive surgery-

Indications for elective surgery are still not defined[26]. The number of elective procedures performed for PUD have declined with more than 70% since the 80's[29]. The results of a questionnaire with 607 responders showed that only 0.3% of the surgeons routinely perform a vagotomy for duodenal ulcer complications and 54.5% mentioned they never include it[29]. Reasons for decline in definitive ulcer surgery are: lower recurrence rate of PUD and PPU because of good results of H.pylori eradication and elimination of NSAID use. Also patients nowadays operated for PPU are older with higher surgical risk which make them less suitable candidates for definitive ulcer surgery. Finally many surgeons practising today have limited experience with definitive ulcer operations[29]. Patients in which definitive ulcer surgery should be considered are those with PPU who are found to be H.pylori negative, or those with recurrent ulcers despite triple therapy[42]. In these patients a parietal cell vagotomy is recommended if necessary combined with anterior linear gastrectomy [40]. This procedure can be safely and relatively easy performed laparoscopically[26]

3)Laparoscopy:

Since the 90's laparoscopic closure of a perforated peptic ulcer has been described. Laparoscopic surgery offers several advantages. First of all a laparoscopic procedure serves as a minimal invasive diagnostic tool[47]. Other benefits from laparoscopic repair are postoperative pain reduction and less consumption of analgesics and a reduction in hospital stay[49]. Also a reduction in wound infections, burst abdomen and incisional hernia due to shorter scars has been noted[49]. Avoiding upper laparotomy might lower the incidence of postoperative ileus and chest infections[23,52]. Drawbacks are a prolonged operating time, higher incidence of re-operations due to leakage at the repair site and a higher incidence of intra-abdominal collection secondary to inadequate lavage[26,42,54]. If the presence of these fluid collections have any clinical relevance is unclear. The higher incidence of leakage might be caused by the diffculty of the laparoscopic suturing procedure. First of all this emphasises the need for a dedicated laparoscopically trained surgeon to perform this procedure[20]. Alternative techniques to

simplify the suturing process have been thought of[41]. Some laparocopic surgeons use omentopexy alone[49]

Suture less techniques have been tried, in which fibrin glue alone or a gelatine sponge has been glued into the ulcer. The downside of this technique is that is only can be used to close small perforations. To overcome this problem a biodegradable patch, that can be cut into any desirable size, has been tested in rats, with good results[51]. Finally, combined laparoscopic-endoscopic repair has been described as well[52].

Postoperative management-

Reviewing literature all patients receive nasogastric probing for at least 48 hrs[23,54]. This however seems to be more "common practice"than evidence based medicine [46]. A recently published Cochrane review concludes that routine nasogastric decompression does not accomplish any of its attended goals and should only be applied in selected cases, which has been supported by other trials as well[50,54]. This also means that oral feeding can be started early, as in colorectal surgery and that waiting for three days, as often is done according to protocol, is unnecessary[54,55].wound infections represent the second most common complication after surgery for PPU. Also the incidence of sepsis is 2.5%. Preoperative intravenous administration of antibiotics has proven to lower the overall infection rate [50]. Although for most surgical procedures a single dose seems to be sufficient, in case of H. Pylori infection triple therapy is recommended consisting of a proton pump inhibitor combined with clarithromycin and amoxicillin

for 14 days [22, 33, 55, 56]. Upper gastrointestinal endoscopy is suggested to be performed after eight weeks to asses healing of the ulcers and to evaluate H.pylori status [55.]

Postoperative complications:(Table A)

Pneumonia	3.6-30%
Woundinfection	10-17%
Urinarytractinfection	1.4-15%
Sutureleak	2-16%
Abscessformation	0-9%
Heart problems (myocardial infarction, heart failure)	5%
Ileus	2-4%
Fistula	0.5-4%
Wounddehiscence	2.5-6%
Biliaryleak	4.9%
Bleeding	0.6%
Re-operation	2-9%
Sepsis	2.5%
Stroke	4%
Death	5-11%

Overview complications after surgery for ppu [13, 16, 19, 20, 42,43, 51-55]

Morbidity rate Mortality rate(tableB)

Boey 0	17.4%	1.5%
Boey 1	30.1%	14.4%
Boey 2	42.1%	32.1%

The postoperative complication most common observed was pneumonia, followed by wound infection. An overview of all Complications and their incidences, based on reviewing literature are listed in table1 [19, 22, 26, 48, 50,56].

Risk factors influencing outcome Mortality after surgery for perforated peptic ulcer is

between 6-10% [20]. There are four main factors which can increase this mortality rate even up to 100%. These are age > 60 years, delayed treatment (>24hrs), shock at admission (systolic BP < 100 mmHg) and concomitant diseases[25,55]. Also gastric ulcers are associated with a two- to threefold increased mortality risk . Boey's score, which is a score based on scoring factors as shock on admission, confounding medical illness, and prolonged perforation, has been found to be a useful tool in predicting outcome (table 2) [29, 45, 56].Perforated peptic ulcer in the elderly Mortality rate after surgery for PPU is three to five times higher in the elderly up to 50% [56]. This can be explained by the occurrence of concomitant medical diseases but also by difficulties making the right diagnosis resulting into delay > 24 hrs[60]. In case of a perforated gastric ulcer or recurrent PUD ,(hemi)gastrectomy with vagotomy might be indicated, but overall simple closure is a safe procedure and there seem to be no need for definitive surgery in this group of patients since ulcer recurrence is only 14%[62, 63].

Conclusion Surgery for perforated peptic ulcer still is a subject of debate despite more than an era of published expertise. Reviewing different policies regarding for instance the indication for conservative treatment, sense or no sense of drains, the need for omentoplasty or not, performing the procedure laparoscopically and the need for definitive ulcer surgery, might contribute to establishing consensus.

Techniques of closure

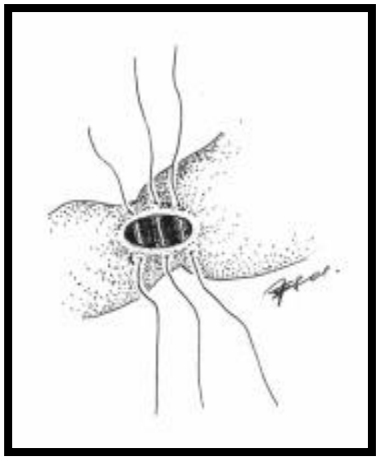
SIMPLE CLOSURE TECHNIQUE

Standard technique

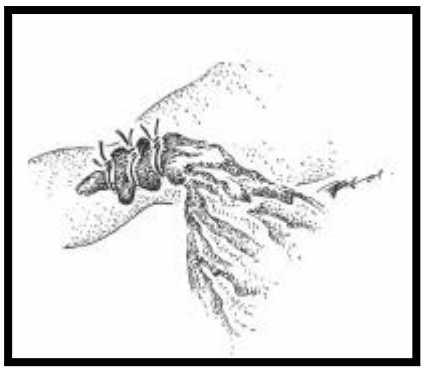
After the patient is placed in supine position on the operating table, the abdomen is prepped and draped in a standard fashion. Transverse epigastric or subcostal incisions have been described. However, an upper midline incision is the preferred route to enter the peritoneal cavity in this setting. In addition to providing good surgical exposure, an upper midline incision also permits extension inferiorly if a perforated ulcer is not found and the remainder of the bowel needs to be inspected and/or manipulated.

Suctioning of the gastrointestinal spillage and of any fibrinous exudates is quickly performed and the attention is turned to inspection of the duodenum and visualization of the perforation. This can be usually found on the anterior wall of the duodenum, in proximity to the duodenal bulb. If the perforation is not apparent, mobilization of the duodenum along with inspection of the stomach and jejunum should be achieved next. After the bowel perforation was identified, sponges can be used to flank the duodenum to prevent further spillage of gastroduodenal contents. Materials commonly used for repair are nonabsorbable sutures like silk or monofilament absorbable sutures like polydioxanone. The suture can be either a 2-0 or 3-0 on a small half-circle swaged needle.

In the original description of the technique, the full-thickness bites were placed approximately 0.5 cm away from the edges of the perforation from one margin to the other. A theoretical hazard with the full-thickness bites is passing the needle through the posterior duodenal wall. Commonly, 3-4 sutures are placed perpendicularly between the edges of the perforation and are laid out on each side of the duodenum (see the image below).



Initial step of the repair with placement of sutures through the wall defect left untied for securement of the omentum. A patch of omentum is brought without tension and positioned over the perforation, and the sutures are successively tied from the superior to the inferior aspect across the omental patch to anchor the omental graft in place (see the images below)



Final repair as seen from anterior with omentum secured in place on to the defect itself.

Final repair of the defect as seen from above.

An important feature of a sturdy repair is reliant on the tying technique. The applied tension to the sutures should be strong enough to stabilize the omentum in place but loose enough to preserve the omental blood supply. Strangulation of the omental patch due to increased tension on the knots is associated with a failure of the repair and continued postoperative leakage. In the classical repair, the sutures are not passed through the omentum but only tied around it. Another variation is to use seromuscular sutures rather than full-thickness bites on the duodenum.

After surgical repair has been accomplished, some surgeons have described performing a leak test to allow detection of technical errors. This can be accomplished either with endoscopic insufflation of air or instillation of methylene blue proximal to the perforation with manual distal compression of the duodenum. However, others argue that this is unnecessary since the repair need not be initially completely occlusive against hydrostatic pressure. These surgeons argue that the goal of the repair is to secure the omentum across the perforation, allowing it to subsequently adhere to the inflamed serosa and thus seal the perforation.

The peritoneal cavity is then irrigated with 10 liters of warm saline solution to remove further contamination. Particular care is taken to irrigate the suprahepatic and infrahepatic recesses, the lesser sac, the paracolic gutters, and pelvis. Optionally, drainage of the areas close to the perforation can be attempted if a concern about possible leakage from the ulceration exists. For this purpose, a Jackson-Pratt drain is sometimes placed in the paraduodenal area or infrahepatic space. The purported advantages of such drainage include the early detection of a postoperative leak and provision of controlled drainage using the closed suction drain if a leak does occur. However, drainage should be selective because routine placement of drains has been found to be associated with significant morbidity and infection with no changes in

incidence of postoperative intra-abdominal abscesses.[5]

The abdomen is then closed in usual fashion with running or interrupted sutures of polypropylene or polydioxanone. If substantial edema of the bowel causes tension on the fascial edges upon attempted closure, then the abdomen may be managed open by various techniques, including a vacuum-assisted closure, the Wittmann patch, or other options.

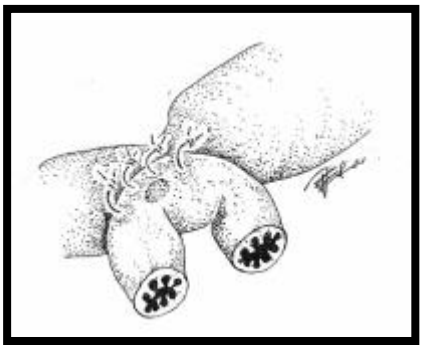
Technique variations

One variation of the classical technique used by some surgeons is the modified omental patch. After sutures are placed between the edges of the perforation in a standard fashion, they are tied in an attempt to close the wall defect. Without cutting the sutures, a segment of omentum is then brought on top of the closed perforation and tied knots and the same sutures are used to tie down the omental patch over the already approximated perforation. Opponents of this modified technique express concern regarding the seal obtained from the omentum when suture knots are interposed between the duodenal serosa and the omental patch. At the same time, the apposition of omentum is not as broad as with the original omental patch. However, no definitive conclusions can be drawn from literature regarding the differences in morbidity or mortality associated with each of these techniques.

Another variation of the standard technique is the use of sero-muscular suture placement (Lembert) instead of full-thickness bites. This is accomplished without entering the duodenal lumen, and these sutures theoretically have a lower risk of passing the needle through the posterior wall, minimizing the risk of obstruction. Seromuscular sutures are generally used during laparoscopic repair of perforated duodenal ulcers.

In patients in whom the omentum is not available because of previous surgery, necrosis,

or anatomy, a variant technique is that of the Thal patch, in which a loop of jejunum is used to patch the perforation (see the image below). In this case, seromuscular sutures are used to attach the serosal side of a loop of jejunum across the ulcer defect.



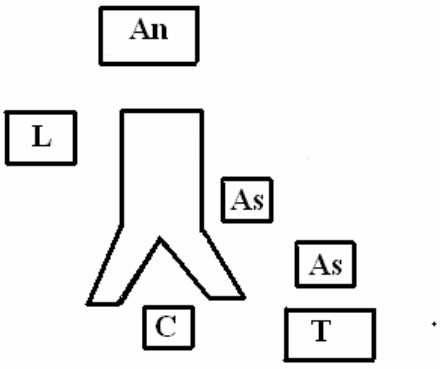
Thal patch: loop of small bowel covering the defect.

LAPROSCOPIC SURGICAL TECHIQUE:

Patient position

At the beginning of the procedure the patient is placed in supine position with legs straight and spread out. The patient position is changed several times during procedure: in steep anti-Trendelenburg position during suture and in lateral decubitus and Trendelenburg position during peritoneal lavage.

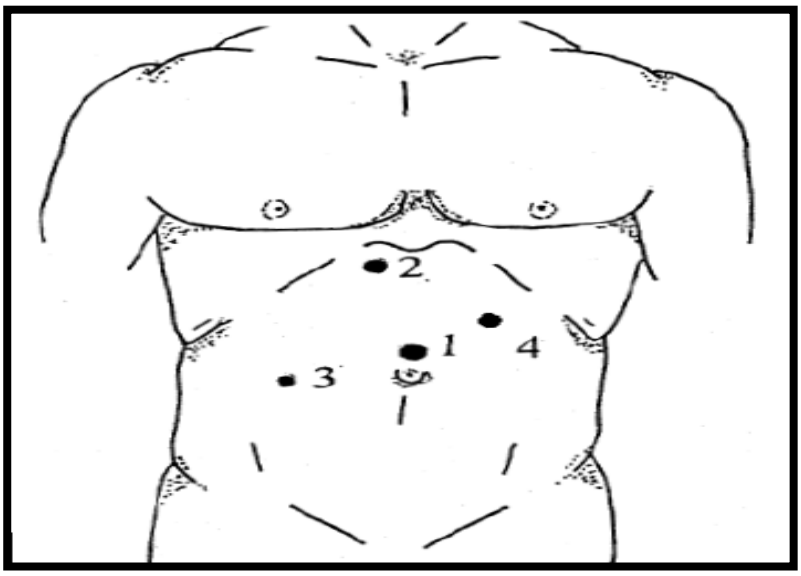
(Fig.6) Patient, team and equipment position.



An-anaesthetic unit; L-laparoscopic unit;

C- surgeon; As- assistant; T-operative table

(Fig.7Trocarspositions)



Team position

The surgical team is placed as for laparoscopic cholecystectomy. The surgeon stands between patient's legs and the assistant to the patient's left (Fig.6). This position is changed during peritoneal lavage with the surgeon to the left of the patient and assistant between patient's legs.

Equipement position

The laparoscopic unit is placed on the patient's left side toward the shoulder. The instrument table is placed at patient's legs.

Trocar position

The position and size of the trocars used vary from one center to the other. The standard technique utilizes four trocars (Fig.7). An optical trocar of 10 to 12 mm is introduced in the periumbilical region. One operating trocar of 5 mm is placed in the inferior aspect of the right upper quadrant on the anterior axillary line for the atraumatic grasper. A 5 or 10/11mm trocar is placed in the left flank. generally at umbilicus level on the midclavicular line for the needle holder which should be perpendicular to the

pyloroduodenal axis. A fourth trocar of 5 mm is placed in the epigastric region and accomodates one or several means of liver and viscera retraction.Some surgeons place the trocars in the same position as for laparoscopic cholecystectomy (French position).In obese patients the position of the trocars needs to be adapted to the morphology of the patients that is to move the trocars closer to the operative region.A three trocar technique can be used, the liver being retracted with the help of a percutaneous suture that suspends the round ligament toward the upper left side of the abdomen.The instruments are similar to those used in most laparoscopic procedures. A 0°laparoscope is commonly used, but a 30° laparoscope may be useful to see better a perforated ulcer placed on the superior surface of the duodenum. The other instruments necessary for this operation are: 2 atraumatic graspers, needle holder, suction-irrigation device, scissors. A liver retractor may be preferred my some surgeons instead of a grasper.Endotracheal anaesthesia is generally used. Close anesthetic monitoring must be done for such a patient and intravenous antibiotic therapy should be done before insufflation. A H2 receptor antagonist or a proton pump inhibitor injection is also advisable.

Fig.8 Perforated duodenal peptic ulcer identified through laparoscopy

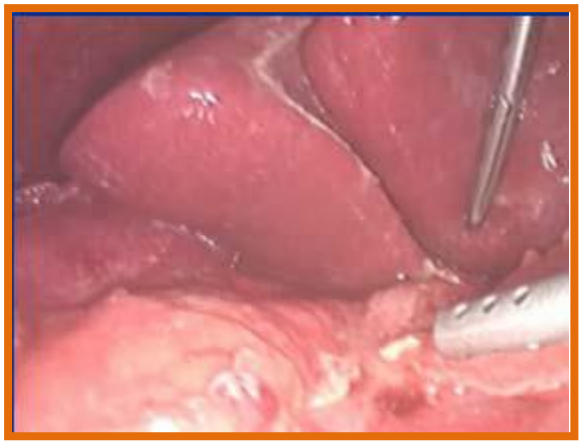
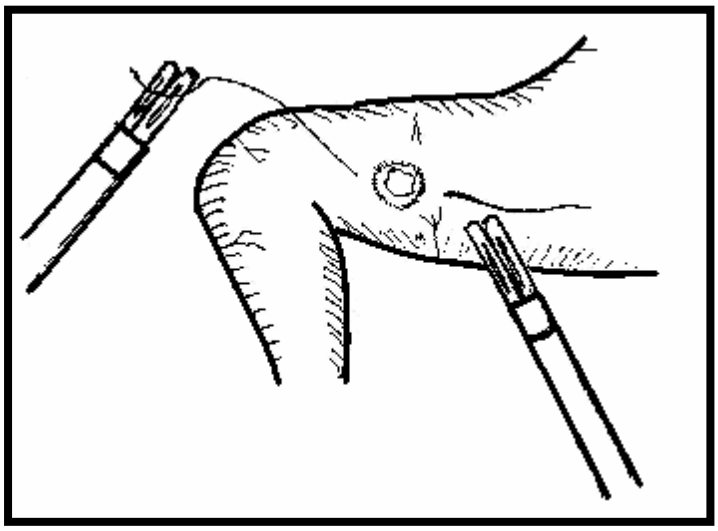


Fig 9 Suture of the perforation using standard stitches



TECHNIQUE:

The Veress needle or an open technique can be used. The abdomen is entered through a small incision just above the umbilicus. A CO2 intraabdominal pressure between 8 and 12mmHg is usually sufficient to realize enough room to work properly.The optic is inserted through the 10-12 mm trocar placed in the supraombilical position. Once the diagnosis is confirmed the other three ports are placed as mentioned above. Bacteriological samples are done and sends immediately to the laboratory.The abdomen is explored to identify the perforation and to assess the magnitude of peritonitis. The gallbladder, which usually adheres to the perforation, is retracted by the surgeon's left instrument and moved upwards. The gallbladder is passed to the assistant using the instrument placed in the sub xiphoid port. Once the liver is retracted the exposed area is carefully checked and the perforation is usually clearly identified as a small hole on the anterior aspect of the first portion of the duodenum (Fig. 3).Next step is cleaning the abdomen. The whole abdomen must be irrigated and aspirated with warm saline solution. Each quadrant is cleaned methodically, starting at the right upper quadrant, going to the left, moving down to the left lower quadrant, and then finally over to the right. The tilt of the operating table should be adapted as necessary.

Special attention should be given to the rectovesical (-uterine) pouch and to the intestinal loops.Fibrous membranes are removed as much as possible, since they might contain bacteria.Once the abdominal cavity is clean the attention is returned to the perforation. Two techniques are generally employed to treat the perforation.

1. The most common technique is suturing the perforation using standard stitches (Fig.4). Biopsy of a duodenal ulcer is not necessary. However, for a gastric ulcer, samples of the gastric wall at the level of the perforation should be taken and sent for histological examination. Suturing is realized with 2/0 or 3/0 slowly absorbable or non absorbable sutures.Interrupted sutures are used and usually two or three stitches are placed in a transversal manner over the perforation focused on the pyloroduodenal axis in case of duodenal ulcer.Once the perforation is sealed, a small fragment of the greater omentum can be fixed over the suture line using the upper thread which was left loose after making the knot. Some surgeons prefer to use instead of omental patch fibrin glue which is spread over the suture. When is difficult to approximate the edges of the ulcer, as is the case with chronic callous ulcers,woven sutures of bigger caliber (0 or 1) must be used in order to avoid cutting the gastroduodenal wall.

2. Closure of the perforation with an omental patch (Graham patch). A floppy piece of greater omentum flap is mobilized. The assistant holds the patch of the omentum just over the perforation and the surgeon sutures it to the edges of the perforation with several interrupted sutures.

3. Alternative options to seal the perforation may include the use of biological glue and sponge plug as a plasty with the round ligament.The peritoneal lavage is continued after the suture. Warm saline solution is used until the returned liquid is clear. About 4 to 6 liters of saline are generally used, but sometimes as much as 10 liters are necessary to clean the abdomen .Routine drainage of the peritoneal cavity is

performed using silicone drains (from 12to 18 French). Depending on the severity of peritonitis, 1 to 3 drains are placed: one drain in the subhepatic region coming out via the trocar site situated on the right flank, another drain at the level of the rectovesical pouch coming out via the trocar site situated on the left flank and a left subphrenic drain coming out via the epigastric trocar site .Before ending the operation the abdomen must be examined for any possible bowel injury or haemorrhage.Trocars are removed one after the other and hemostasis of the trocar sites is checked.The telescope is removed leaving the gas valve of umbilical port open to let out all the gas.The musculo-aponeurotic plane is closed only at the level of the 10/11mm trocar sites. The skin is closed using staples or sutures.

Postoperative management:-The patient may have slight pain initially but usually resolves with mild pain killers.

Intravenous H2 receptor antagonists or proton pump inhibitors are given intravenously and then orally once infusions are stopped. Intravenous antibiotic therapy is maintained depending on the severity of the peritonitis and at least until a culture of the peritoneal fluid taken during the procedure is obtained. If the culture is negative intravenous antibiotic therapy is discontinued after 72 hours. However, if the culture is positive, intravenous antibiotic therapy is continued for 10 days first and then orally after return of bowel function and food intake.The aims of antibiotic therapy are to combat peritonitis and *Helicobacter pylorus*.

The nasogastric tube is removed once peristalsis resumes and a clamping test is successful. Food intake is then restored. Drains are removed once the effluent is less than 100mL per day.When suturing is difficult or bowel function is resumed late, the gastric tube can be left in place longer. Water-soluble gastroesophageal contrast (GastrografinTM) examination is then performed to check the integrity of the closure

and ensure the absence of pyloroduodenal stenosis.Control gastroscopy is performed usually 4 to 6 weeks after the operation. Patient may be discharged 3 days after operation if every things goes well.

Complications-Suture leak represents one of the most frequent postoperative complications with a rate averaging from 5 to 16[66]. Other commonly reported complications are pneumonia,which is most likely related to pneumoperitoneum, prolonged dynamic ileus, intraabdominal abscess formation, external fistula, and hemorrhage.Stenosis at the pyloroduodenal level, notably in the presence of callous chronic ulcer could be a late complication. If needed, an endoscopy performed during the procedure makes it possible to rule out this complication.

Conversion:-The conversion rate varies from 0% up to 60%. Inadequate ulcer localization and large ulcer size are commonly reported reason for conversion[72,77]. Other reported reasons for conversions are infiltration and fragility of ulcer edges, perforation associated with bleeding, cardiovascular instability induced by pneumoperitoneum, peripancreatic infiltration, prolonged perforation >24 hours, inadequate instruments, abscess, postoperative adhesions.

MATERIALS AND METHODS

Patients admitted to emergency ward with duodenal ulcer perforation during the period of January 2010 to January2011 at R.L.Jalappa Hospital and Research Center.60 such patients were included in the study.

30 patients were taken up fo laproscopic closure of perforation and 30 patients were taken for laparotomy and closure of perforation(open procedure).

All patients were followed up for 6 months after surgery.

Parameters Used In The Study

Operative duration

Analgesics and antibiotics requirement

Post operative hospital stay

Local and systemic complications

OBSERVATIONS AND RESULTS

This prospective study compromised of 60 patients with duodenal ulcer perforation who were admitted in R. L.Jalappa hospital and research centere attached to sri devraj urs medical college,Tamaka, Kolar and underwent laproscopic and open closure of duodenal perforation from January 2010 to January 2011.

All the cases underwent detailed preoperative assement.The decision to operate either laproscopically or open was taken by the senior operating surgeon considering inclusion and exclusion criteria.Their preoperative ,intraoperative and postoperative findings and complications were meticulously recorded as per protocol.The findings were tabulated and following observations were made.

OPERATIVE TECHNIQUE

A pneumoperitoneum was created using Hassan open technique . Insufflation pressure was maintained below 11 mm Hg to minimize the risk of transperitoneal translocation of bacteria and endotoxemia. Four ports were inserted: the upper trocar was placed in the subxiphoid area and used for irrigation and suction and/or retraction of the liver. An umbilical port was used for the camera and the 2 remaining working ports were placed on each side of the camera port in a triangulated fashion. The surgeon stands left of the patient, with an assistant on each side. The gallbladder, which usually covers the perforation, was retracted upward and held by the assistant. Inflammatory adhesions were divided and suctioned. The exposed area was examined and the perforation identified. For the purpose of the study, the tip of the suction-irrigation tube (5 mm) was used to measure the size of the perforation. The next step was careful and thorough irrigation and suction of all intra-abdominal fluid, requiring about 10 L of isotonic sodium chloride solution. Each quadrant was cleaned methodically, starting at the right upper quadrant and moving in a clockwise fashion. Special attention was given

to the vesicorectal pouch retracting the sigmoid colon and accessing all loculated pelvic spillage. Fibrinous membranes on the small bowel were removed as much as possible without damaging the serosal surfaces.

The perforation was closed using the classic omental patch technique with the omental patch inserted between 2 or 3 stitches with nonabsorbable sutures before tying the knots intracorporeally. This method was preferred to the technique of suturing the perforation closed and buttressing the repair with an omental patch. Decision to convert to an open approach was dictated by the patient's intolerance to carbon dioxide insufflation and consequent hemodynamic instability, and the inability to obtain appropriate laparoscopic closure due to the size of the perforation or the friability of the ulcer edges and perforation at other sites. Pelvic and subhepatic drains were placed at the end of the procedure, and the fascia was closed in all ports. The open repair was conducted through a midline incision and followed the same technical guidelines.

Plain Erect X-ray Abdomen

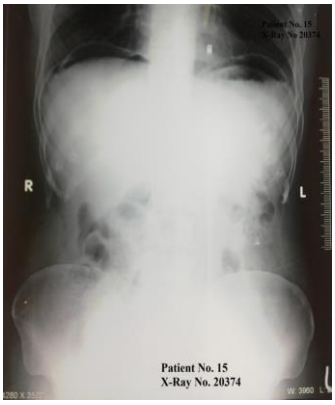


Fig 10: Erect X-ray abdomen showing gas under the diaphragm

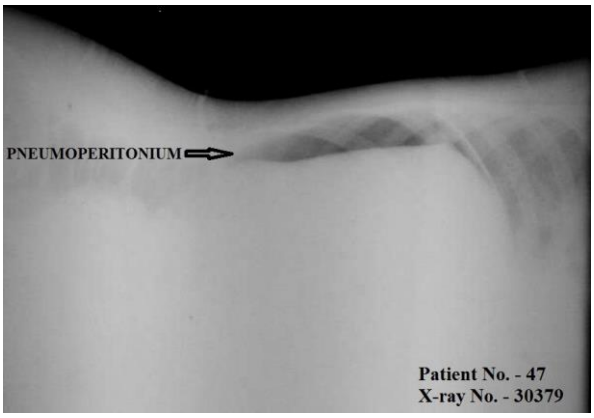
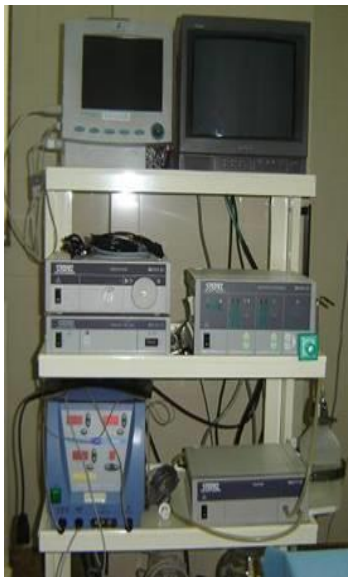
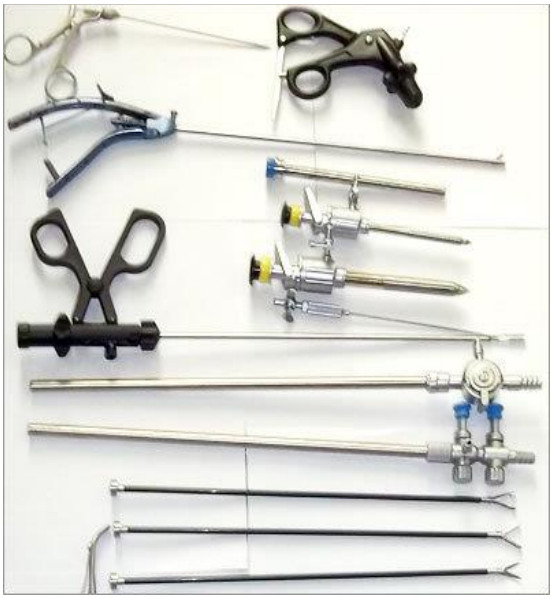


Fig :11 Lateral decubitus X-ray abdomen showing pneumoperitoniu

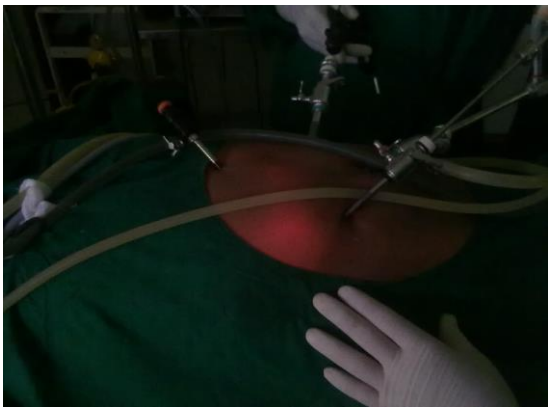
Laprscopic Trolley(fig12)

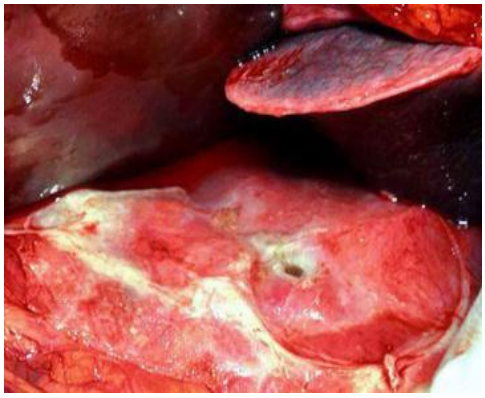


Laproscopic Instruments and camera(fig13)

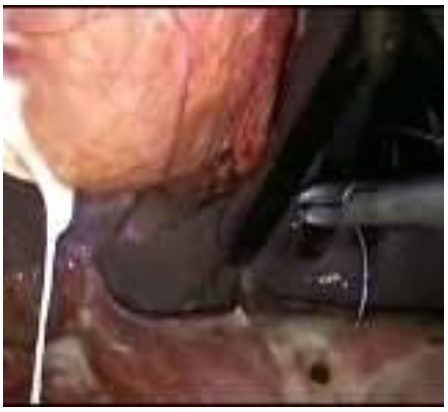


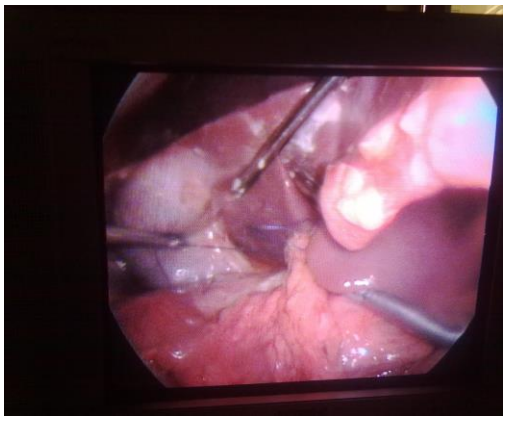
Laprroscopic Placement Of Ports &Creating pnuemoperitoneum(fig14)



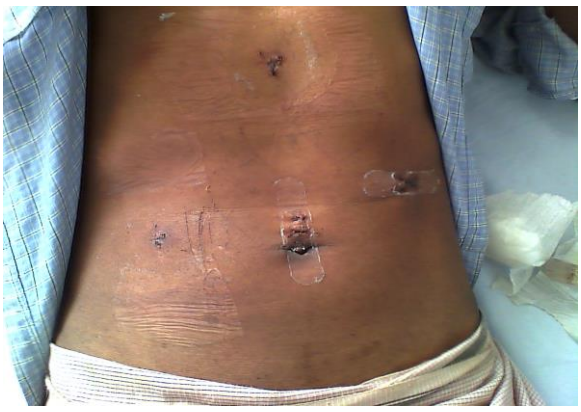


Duodenal Ulcer Perforation(D1)(fig 15)





Perforation Closed By Placing Omentum(fig16



Post operative photo at the time of discharge(fig17)

Table1

Sex distrubution

Gender	No of Cases	%
Male	49	80
Female	12	20
Total	61	100

Table2

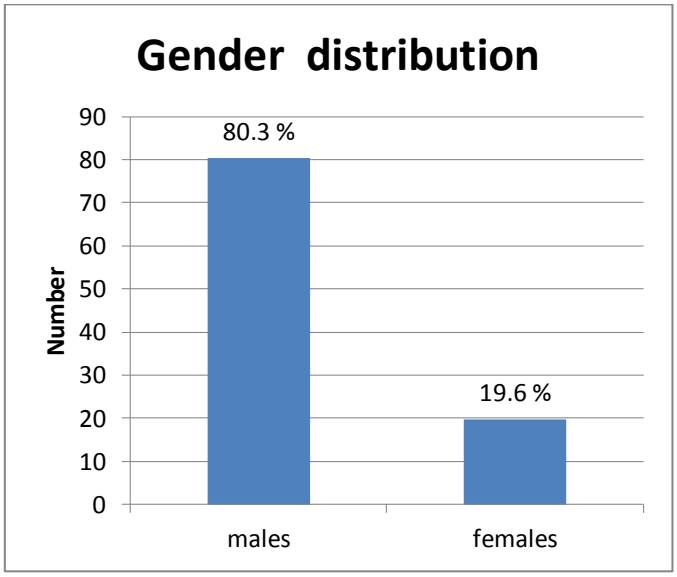
Age distribution of study groups

Age in years	No of Cases	%
21-40	23	37
41-60	35	57
61-80	3	4

There was a male preponderance in the study group with 80% of patints being males. Ther was preponderance of cases in 4^{th},5^{th} and 6^{th} decades of life in the study for accounting nearly 57% of the cases.The mean age group patients in the study was 50 years.

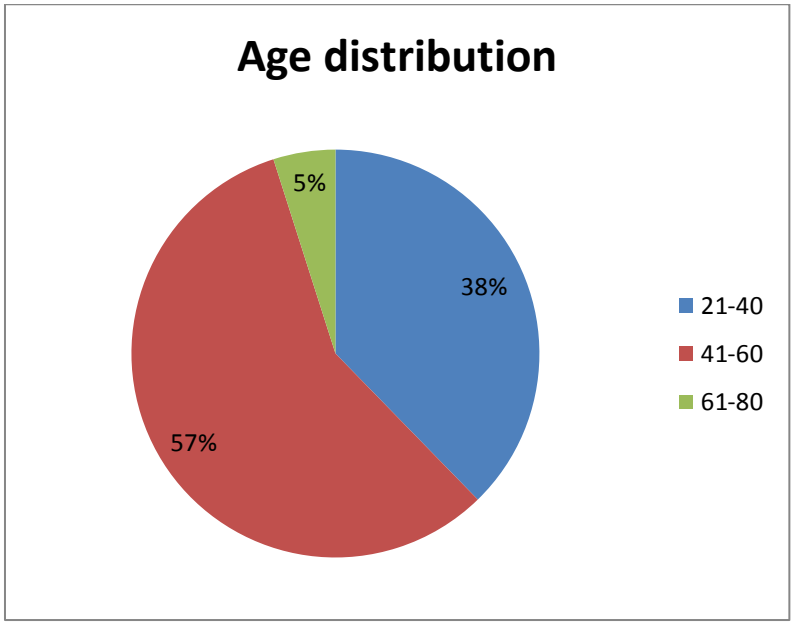
Graph1

Gender distrubution



Graph 2

Age distrubution



Duration Of Surgery(MINS) table3

DURATION								
Independent Samples t Test								
	type of surgery	N	Mean	Std. Deviation	t	df	Sig. (2-tailed)	
durat(MIN)	Open	31	56.45	5.94	-3.13	59	0.003	
	Laproscopic	30	62.17	8.17				

The mean duration of surgery in our study for open procedure was 56 mins compared to laproscopic procedure which was 62 mins which was statistically significant showing lesser time for open procedure(table3).

Number Of Antibiotics(Days)(table4)

ANTIBIOTICS								
Independent Samples t Test								
	type of surgery	N	Mean	Std. Deviation	T	Df	Sig. (2-tailed)	
anti(DAYS)	Open	31	5.26	1.37	3.66	59	0.001	
	Laproscopic	30	4.03	1.25				

The mean number of days of antibiotics used in open group was 5 days compared to 4 days in laproscopic group which was statistically significant.The number of days used in laproscopic group was less(table4)

Analgesics to acess post op pain(days)(table5)

ANALGESICS								
Independent Samples t Test								
	type of surgery	N	Mean	Std. Deviation	T	df	Sig. (2-tailed)	
anal(DAYS)	1	31	6.58	1.20	5.06	59	0.001	
	2	30	4.87	1.43				

The mean number of usage of antibiotics in open group was 7 days and in laproscopic group was 5 days which was statistically significant.there was less requirement of analgesics in laproscopic group(table5).

Post Operative Stay(Days)(table6)

POST OP STAY								
Independent Samples t Test								
	type of surgery	N	Mean	Std. Deviation	T	Df	Sig. (2-tailed)	
stay post(DAYS)	Open	31	7.94	1.53	4.80	59	0.001	
	Laproscopic	30	6.17	1.34				

In our study the mean number of hospital stay for open procedure was 8 days compared to 6 days in laproscopic group which was statistically significant.Number of postoperative stay was less in laproscopic group

POSTOPERATIVECOMPICATIONS(TABLE7)

Post op complications	Open(%)	Laproscopic(%)
Wound infection	3(9%)	1(3%)
Burst abdomen	1(3%)	0
Leakage from perforated site		0
Mortality	2(6%)	0

There was wound infection in 3(9%) persons in open group and 1(3%) in laproscopic group.ther was 1(3%)patient with burst abdomen in open group and 2(6%) mortality in open group.this was not there in laproscopy group.there was no leakage from perforation in either of the groups.

STATISTICAL METHODS

All data are expressed as median and interquartile range unless otherwise stated. Comparison between the 2 groups was made using non parametrical methods . Comparisons between categorical data were made using independent samples T test. The 2 groups were categorized based on type of procedure done . patients in the laparoscopy group who were converted to an open procedure remained in the open group for the statistical comparisons. $P<.05$ was considered statistically significant.

DISCUSSION

There was no difference in age, weight, duration of symptoms, and time to surgery between 2 groups.Often it is mentioned that age of patients presenting with PPU is increasing,due to better antiulcer treatment and also due to more usage of NSAIDS and aspirin usage in elderly population.the results in table1 shows 57%of the population was among the age group of 40-60 and mean age being 52 years which is correlating with literature.

The mean operating time of the laparoscopic patch repair was significantly longer than the open procedure (mean52.45 minutes vs 62.17 minutes ; *P*=.001).our study correlates with the study done by So jb Ku[106,114] ,katakhouda[84] and Bertleff [51,95]. A disadvantage of the laparoscopic approach is a longer operating time[65,82,89,90,93], but this had no effect on the overall results. Some authors were able to shorten the operating time by using sutureless techniques to close the perforation with the use of a gelatin plug and application of fibrin sealant (Tisseel; Baxter Immuno, Deerfield, Ill).[56] We think that this method is elegant and can be added to the surgical armamentarium.

Study	Operating time		P value
	open	lap	
SO JB,Ku(1996)	65	80	0.02
Katkhouda(1999)	63	106	0.003
Bertleff(2009)	50	75	0.02
Siu(2002)	52.3	42	0.03
Sreeramulu et al	52.6	62.7	0.003

3 (9.6) patients in the laparoscopy group underwent conversion to an open procedure. The reasons for conversion were large perforations (diameter >10 mm) in one patient precluding safe laparoscopic closure ,but by using an omental patch this might not necessarily have to be a reason to convert anymore.the other two patients had ileal perforations[56]. Shock at admission and comorbid diseases are other reasons more conversion rate[51]. In analyzing our results compared to other studies, we found 2 clinical parameters that should be excluded for safe laparoscopic repair of perforated ulcers—shock and symptom duration more than 24 hours. Patients who presented with evidence of shock and were treated laparoscopically had a high conversion rate and a significantly worse postoperative course than patients without shock on admission. In this clinical situation, a laparoscopic approach that has a high likelihood of failure should not be attempted, and the patient should have an expeditious open repair. Patients with symptoms for more than 24 hours represent another group where laparoscopy may be attempted with a high risk of conversion to an open procedure. These risk factors are consistent with the operative risk factors in perforated duodenal ulcers defined by Vaidya et al[67], Siu et al and Lunevicius et al[50,58].

Study	Conversion%
Palanivelu (2007)	0
Vaidya(2009)	10.5
Lunevicius(2005)	23.3
Siu (2002)	14.2
Sreeramulu et al	9.6

The best parameters to compare two different surgical techniques are morbidity and mortality.PPU is still associated with high morbidity and mortality, with main problems caused by wound infection,sepsis,leakage at the repair site,and pulmonary problems.In our study high morbidity and mortality was seen in open group which is consistent with the studies shown below.The postoperative complications are summarized in (table 7)[103]. There was significant difference in morbidity between the laparoscopy and open surgery groups (1 of 30 patients vs 6 of 31 patients). Two mortality occurred in open group.

	Woundinfection%		Burst abdomen%		leak%		Mortality	
	open	lap	open	lap	open	lap	open	lap
Bertleff(2009)	6.1	0	0	0	0	0	8.1	3.8
Siu(2002)	0	0	0	0	0	0		
Sreeramulu et al	9	3	6	0	0	0	3	0

The opiate analgesic requirements were significantly less in the laparoscopy group compared with the open surgery group (3 doses [range, 2-7] vs 9 doses [range, 7-11]; *P*=.002). Time to return to a normal diet was also significantly shorter in the laparoscopy group (3 days [range, 1-4] vs 5 days [range, 4-7]; *P*<.001). This was also reflected in the median length of hospital stay that was significantly shorter in the laparoscopy group (3 days [range, 3-7]) compared with the open surgery group (8 days [range, 6-10]) (*P*=.003).

Study	Analgesics(Days)		Hospital stay(Days)	
	open	lap	open	lap
Lau(1996)	3.5	1.5	5	4
Bertleff(2009)	1	1	8	6.5
Sreeramulu et al	7	5	8	6

A follow-up endoscopy at 6 months was performed in 5 patients from the laparoscopy group and in 7 patients from the open surgery group.Rest of the patients did not turn up for followup endoscopy. No recurrent ulcer was documented in any patient in either group.

Conclusion

To conclude Laproscopic closure of early presentation of duodenal ulcer perforation proves to be efficient.

It is safe, with lesser requirement of analgesics,shorter hospital stay.

Its proves to have lesser morbidity and mortality as compared to open closure if presented early.

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PROFORMA OF THE CASE SHEET

Study Of Efficacy, Safety & Outcome Of Laparoscopic Closure Of Duodenal Ulcer Perforation

Case No.

Particulars of the Patients

Name;

Age;

Sex;

Occupation;

Address;

Ward ;

IP No.;

Unit;

Date of Admission;

Date of Discharge;

Complaints

1) Pain

-Time of onset

-Mode of onset

-Site of pain

-Migration of pain

-Character of pain

-Relation to vomiting

-Relation to food intake

2) Vomiting

-Onse

-Duration

-Frequenc

-Amoun

-Character of onse

-Content

4) Bowels

-Last evacuation

-Constipated / Normal

-History of passing worm

4) Distension

-Duration

-Location upper / lower abdomen

-Relation to pain

5) Fever

-Duration

-Nature ; Continuous / Intermittent /Remittent

-Relation to pain

-Whether associated with Chills and Rigors

Previous history

- Of similar complaints

-Haematemesis

-Treatment of peptic ulcer

-Ingestion of drugs

Personal History

-Diet

-Appetite

-Smoking

-Alcohol

-Bowel Habits

-Menstrual History

Family History

-Peptic Ulcer

-Diabetes

-Hyper tension

General Examination

-Appearance

-Attitude

-Build and nourishment

-Level of Consciousness

-Temperature

-Pulse

-Blood Pressure

-Respiration

Local Examination

Inspection

- Contours of the abdomen

- Normal

- Distension

- Uniform

- Upper

-Lower

- Visible peristalsis

- Skin

- Umbilicus

- Operation Scars

- Hernial Orifices

- Genitalia

Palpation

-Temperature

-Tenderness

-Localized

-Diffuse

-Rebound

-Muscular rigidity

-Localized

-Generalized

-Mass

-Liver

-Spleen

-Abdominal girth

Percussion

-obliteration of liver dullness

-shifting dullness

Auscultation

-Bowel sounds

-Present

-Frequency

-Character

-Absent

Other relevant examinations

-Per rectal

-Empty

-Loaded

-Bleeding

-Mass felt

-Per vaginal

Other systems

-Respiratory system

-Cardiovascular system

-Central Nervous system

INVESTIGATIONS

Hematological

-Complete blood count

-Blood grouping

-Random blood sugar

-Renal function test

-Serum electrolytes

-Widal

Urine Routine

Diagnostic tapping of peritoneal fluid

Pre operative treatment

-Antibiotics

-Other drugs

-Intravenous Fluids

-Gastric aspiration

Pre medication and Anaesthesia

Operative details

Antibiotics Requiered

Analgesics Required

Post Operative Complications

Postoperative Hospital Stay

Key To Master Chart

S No-Serial Number

Gender

1-Male

2-Female

Type Of Perforation

1-Open

2-Laparoscopic

Durat-Duration

Mins-Minutes

Anal-Analgesics

Anti-Antibiotics

Stay post-post operative hospital stay

Wnd Inf-wound Infection

Burst Abd-Burst Abdomen