Efficacy and safety of two different doses of Dexlansoprazole in assessing fasting gastric residual volume by using ultrasonography in diabetic patients undergoing elective surgery

B.L.D.E (DEEMED TO BE UNIVERSITY)

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I hereby declare that this dissertation entitled "**Efficacy and safety of two different doses of Dexlansoprazole in assessing fasting gastric residual volume by using ultrasonography in diabetic patients undergoing elective surgery** Comparative study" is a bonafide and genuine research work carried out by me under the guidance of DR. SRIDEVI MULIMANI, Professor, Department of anaesthesiology B.L.D.E (DU), Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura

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Dr Devendra kumar verma

ABBREVIATIONS

ASA – Acetylsalicylic Acid (Aspirin) / American Society of Anesthesiologists

ASPDA – Anterior Superior Pancreaticoduodenal Artery

ATP – Adenosine Triphosphate

CHA – Common Hepatic Artery

CSA – Cross-Sectional Area

DKA – Diabetic Ketoacidosis

DL –Dexlansoprazole

DR – D-related (HLA gene)

ECL – Enterochromaffin-like (cells)

ENS – Enteric Nervous System

FBS – Fasting Blood Sugar

GDA – Gastroduodenal Artery

GDM – Gestational Diabetes Mellitus

GIT – Gastrointestinal Tract

GRV – Gastric Residual Volume

HbA1c – Hemoglobin A1c

HCl – Hydrochloric Acid

HHS – Hyperosmolar Hyperglycemic State

HLA – Human Leukocyte Antigen

ICC – Interstitial Cells of Cajal

IDF – International Diabetes Federation

LADA – Latent Autoimmune Diabetes in Adults

LGA – Left Gastric Artery

LGEA – Left Gastroepiploic Artery

MHC – Major Histocompatibility Complex

MODY – Maturity-Onset Diabetes of the Young

PDGFRα – Platelet-Derived Growth Factor Receptor Alpha

PPI – Proton Pump Inhibitor

PSPDA – Posterior Superior Pancreaticoduodenal Artery

RBS – Random Blood Sugar

RGA – Right Gastric Artery

RGEA – Right Gastroepiploic Artery

T1DM – Type 1 Diabetes Mellitus

T2DM – Type 2 Diabetes Mellitus

ABSTRACT

Background: Diabetic patients undergoing elective surgery are at increased risk of pulmonary aspiration due to gastroparesis, which leads to delayed gastric emptying and increased gastric residual volume (GRV). Proton pump inhibitors (PPIs) such as Dexlansoprazole have been shown to reduce gastric acid secretion and volume. However, the optimal dose for effective GRV reduction in this patient population remains uncertain. Aimed to assess the efficacy and safety of two doses of Dexlansoprazole (30 mg and 60 mg) in reducing fasting GRV using ultrasonography in diabetic patients scheduled for elective surgery.

Materials and Methods: A double-blind comparative study was conducted on 184 diabetic patients, randomized into two groups: DL30 (30 mg Dexlansoprazole) and DL60 (60 mg Dexlansoprazole). Gastric volume was measured using ultrasound in supine and right lateral decubitus positions. Additional assessments included glycemic parameters, fasting duration, and postoperative nausea and vomiting. Statistical analysis were performed using SPSS Version 20, with $p<0.05$ considered significant.

Results: The DL60 group showed a significant reduction in GRV (28.32 ± 7.58 mL) compared to the DL30 group (39.72 ± 8.54 mL, $p<0.05$). There was a significantly lower incidence of regurgitation (3.3% vs. 15.2%) and aspiration risk (7.6% vs. 29.3%) in the DL60 group ($p<0.05$). Demographic variables, ASA grade, and glycemic parameters were comparable between the groups.

Conclusion: A higher dose of Dexlansoprazole (60 mg) is more effective and safer in reducing fasting gastric residual volume and minimizing aspiration risk in diabetic patients undergoing elective surgery. These findings suggest that 60 mg Dexlansoprazole should be preferred for preoperative gastric volume reduction in high-risk patients.

Keywords: Dexlansoprazole, Gastric Residual Volume, Diabetic Gastroparesis, Aspiration Risk, Ultrasonography, Elective Surgery

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

The stomach's primary role in the human body is to aid with digesting,acid secretion, enzyme secretion, gastrointestinal motility, and its role as a reservoir are the four main aspects of gastric digestive function. “The stomach's ability to store fluid permits it to expand greatly while only minimally increasing internal pressure. The safety of gastric residual volume in patients with established risk factors, such as those known to cause a delay in stomach emptying or an increase in gastric residual volume, is also uncertain. The reduction of stomach residual volume (GRV) will lower the risk of aspiration, which is the justification for fasting. The predicted level of GRV in a population of fasting patients is unclear. Several earlier investigations that used various measurement methods reported values in the range of 0.4 to 1.2 ml/kg.[1–5]

Due to the concomitant gastroparesis, autonomic neuropathy in a diabetic patient may increase the risk of pulmonary aspiration and predispose the patient to hemodynamic instability during anesthesia. Gastroparesis, a condition characterized by delayed gastric emptying without mechanical obstruction, has a reported incidence of approximately 4.8% in type 1 diabetes and 1% in type 2 diabetes. The impaired gastric motility in these patients can lead to prolonged gastric retention of food, increasing the risk of regurgitation and aspiration, particularly during induction of anesthesia.

The volume, consistency, and nutrient composition of gastric contents play a crucial role in determining the rate of gastric emptying. High-fat and high-fiber meals, as well as hyperglycemia, can further delay gastric emptying, exacerbating the risk in diabetic patients. In addition, autonomic dysfunction may impair the normal physiological responses that facilitate gastric emptying and airway protection, such as lower esophageal sphincter tone and cough reflex, compounding the risk of aspiration pneumonia.

To mitigate this risk, preoperative fasting guidelines provided by the American Society of Anesthesiologists (ASA) are strictly followed to ensure an empty stomach before anesthesia induction. The ASA recommends fasting from clear liquids for at least two hours, from breast milk for four hours, from light meals (such as toast) for six hours, and from heavy meals (including fatty or fried foods) for eight hours before surgery. However, in diabetic patients with gastroparesis, standard fasting durations may not be sufficient, and individualized fasting protocols or the use of prokinetic agents like metoclopramide may be considered." Additionally, in high-risk cases, gastric ultrasound can be utilized as a bedside tool to assess residual gastric volume before surgery.[3]

Acute and chronic illness symptoms of diabetes mellitus significantly increase the likelihood that a patient will require surgical intervention. The metabolic disturbances associated with diabetes, including hyperglycemia,

insulin resistance, and impaired wound healing, can complicate both surgical procedures and postoperative recovery.

Acute complications, such as diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS), can present as medical emergencies requiring urgent surgical management. These conditions are often triggered by infections, cardiovascular events, or uncontrolled diabetes and can lead to severe dehydration, electrolyte imbalances, and altered mental status, all of which need to be stabilized before surgery.Chronic complications of diabetes, including cardiovascular disease, nephropathy, neuropathy, and retinopathy, further increase surgical risks. Cardiovascular involvement, such as coronary artery disease or peripheral vascular disease, predisposes diabetic patients to increased perioperative morbidity and mortality. Diabetic nephropathy can impair drug metabolism and fluid balance, necessitating careful perioperative management. Neuropathy, particularly autonomic dysfunction, may lead to intraoperative hemodynamic instability, gastroparesis, and an increased risk of aspiration. Retinopathy, while not directly affecting surgical outcomes, may require ophthalmologic evaluation before certain procedures.

Pulmonary aspiration of gastric content in patients undergoing general anesthesia is a serious perioperative complication. Gastroparesis despite standard fasting in diabetic patients may increase the aspiration risk.

Pneumonitis has been reported in up to 47% of patients who suffer pulmonary aspiration.[6]

The PPIs are potent blockers of acid secretion from parietal cells and significantly raise gastric pH compared to histamine receptor antagonists i.e Ranitidine, famotidine or antacidsi.e aluminium hydroxide, magnesium carbonate, magnesium hydroxide.[2]Unlike other agents, tolerance does not develop to the acid blocking ability of the PPIs as they are able to consistently provide acid suppression over prolonged (months to years) periods of time. Thus, the PPIs have become the gold standard for treatment of reflux related diseases and not only do they improve efficacy, but they are generally safe, well tolerated pharmacologic agents

Dexlansoprazole a newer generation Proton pump inhibitor (PPI), used in the treatment of hydrochloric acid suppression, are currently among the most commonly used drug. Their popularity stems from the high efficacy in inhibiting gastric acid secretion suppression, The discovery of PPIs radically changed the therapeutic approach to diseases caused by excessive secretion of acidic gastric juices.[4] At present, PPIs are the most widely used and the strongest-acting antisecretive drugs.

Proton pump inhibitor therapy reduces the volume of gastric contents significantly during the first 75 minutes after a meal. PPI therapy, in addition to increasing gastric pH, may reduce the frequency of gastro-oesophageal reflux by decreasing the volume of gastric contents.[3]

Dexlansoprazole having the dual release of the active ingredient in the duodenum and the small intestine makes it possible to achieve two peak concentrations at various times, within two and five hours of administration and longest maintenance of drug concentration in the plasma of all known proton pumpdue to its unique formula, Dexlansoprazole.

Hence the present study wasevaluateof the efficacy and safety of two doses of Dexlansoprazole 30 mg and 60 mg in assessing fasting gastric residual volume by ultrasonography in diabetic patients undergoing elective surgeries in relation to the duration of diabetes, blood sugar levels, HbA1c and fasting duration.

AIMS & OBJECTIVES

Aim:

To evaluate gastric residual volume in fasting diabetic patients with two doses of oral Dexlansoprazole (30mg and 60mg) scheduled for elective surgery.

Objective:

Primary Objective:

- Measuring and calculating the gastric volume in the supine position and right lateral decubitus position with Ultrasonography.

Secondary Objective:

- To determine the minimum safety dose of dexlasnoprazole
- To assess postoperative nausea and vomiting with 24hours

Measuring GRV with respect to

- Duration of diabetes
- Blood sugar level
- HbA1c
- Fasting duration
- Regurgitation & Aspiration.
- Nausea & vomiting.

ANATOMY AND PHYSIOLOGY

Diabetes has often been considered a high-risk state posing a serious challenge to the anaesthesiologist in many aspects. One of the feared complications is pulmonary aspiration as diabetic patients are considered as possible full stomach due to autonomic gastropathy.[7,8]

ANATOMY OF STOMACH

The stomach is a "vital organ that is the most dilated part of the digestive system. The oesophagus comes first, followed by the small intestine. It is a huge, muscular, hollow organ with the ability to hold food. It is divided into four sections: the cardia, fundus, body, and pylorus. The cardia is related to the oesophagus and is where food enters the stomach for the first time. The fundus is a bulbous, dome-shaped, superior part of the stomach that follows the cardia. The fundus is followed by the body, or the major, biggest section of the stomach. The pylorus, which follows the body, conically channels food into the duodenum, or upper section of the small intestine." The stomach is positioned in the upper abdomen of the human body, to the left of the midline. The next step of digestion begins in the stomach after mastication or chewing.

Structure and function[9]

The stomach's main functions include temporarily storing food and facilitating partial chemical and mechanical digestion. "As food enters, the

upper sections (cardia, body, and fundus) relax to accommodate larger volumes, while the lower region contracts rhythmically to break down food and mix it with stomach fluids. These fluids aid in digestion, transforming the food into chyme, which is prepared for further digestion. Mixing waves occur every 20 seconds, increasing in intensity as they move toward the stomach's lower region.

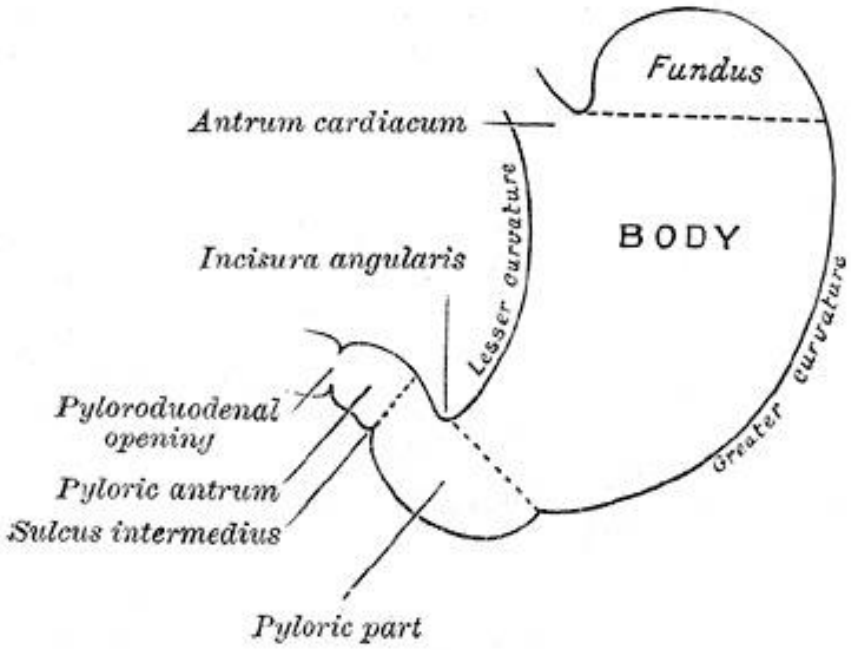


Figure 1: Outline of stomach

With each wave, “the pyloric sphincter admits little amounts of sufficiently liquefied/broken down chyme into the small intestine that the duodenum can manage and control. Stomach juices are liquids generated spontaneously by the fundus section of the stomach for chemical digestion reasons, and they comprise hydrochloric acid (HCl) and the enzyme pepsin. In addition to HCl,

the stomach's parietal cells create intrinsic factor. The intrinsic factor generated at this stage of digestion facilitates vitamin B12 (cobalamin) absorption later in the small intestine." The intrinsic factor's generation is vital since vitamin B12 is required for the creation of red blood cells and neurological processes.

On average, the stomach digests food and moves it to the duodenum within 2 to 4 hours, though this process varies based on food type, with carbohydrates and proteins breaking down quickly while fats take longer. "While its primary function is not nutrient absorption, the stomach can absorb certain substances, including water during dehydration, aspirin, amino acids, ethanol, caffeine, and some water-soluble vitamins. Additionally, its acidic environment helps protect the body by eliminating harmful bacteria and microbes that enter through food, reducing the risk of infections and illnesses."[10,11]

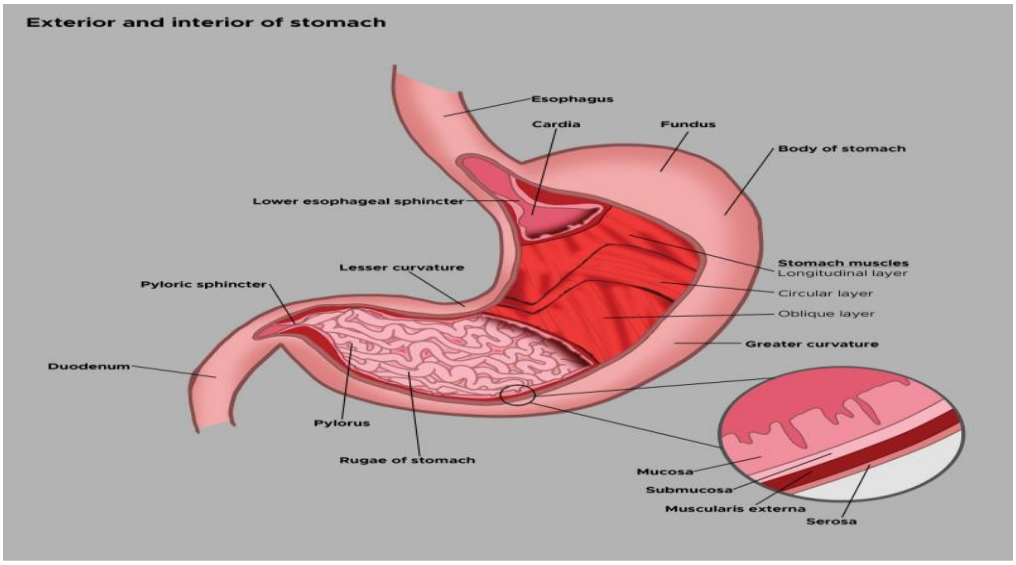


Figure 2: Illustration of the exterior and interior of the stomach

Embryology

The stomach begins to form as the most dilated region of the foregut during the fourth week of development. Because of fast esophageal elongation, “the stomach drops from the level of the C2 vertebrae to the level of the T11 vertebrae by week twelve. By the fifth week of development, one side of the stomach (dorsal wall) develops faster than the other side (ventral wall), causing the stomach to protrude more on one side, giving it its characteristic form. The stomach rotates 90 degrees clockwise about the longitudinal axis during week 7 and then clockwise around the anteroposterior axis during week 8, bringing the pyloric area higher to its ultimate position.”

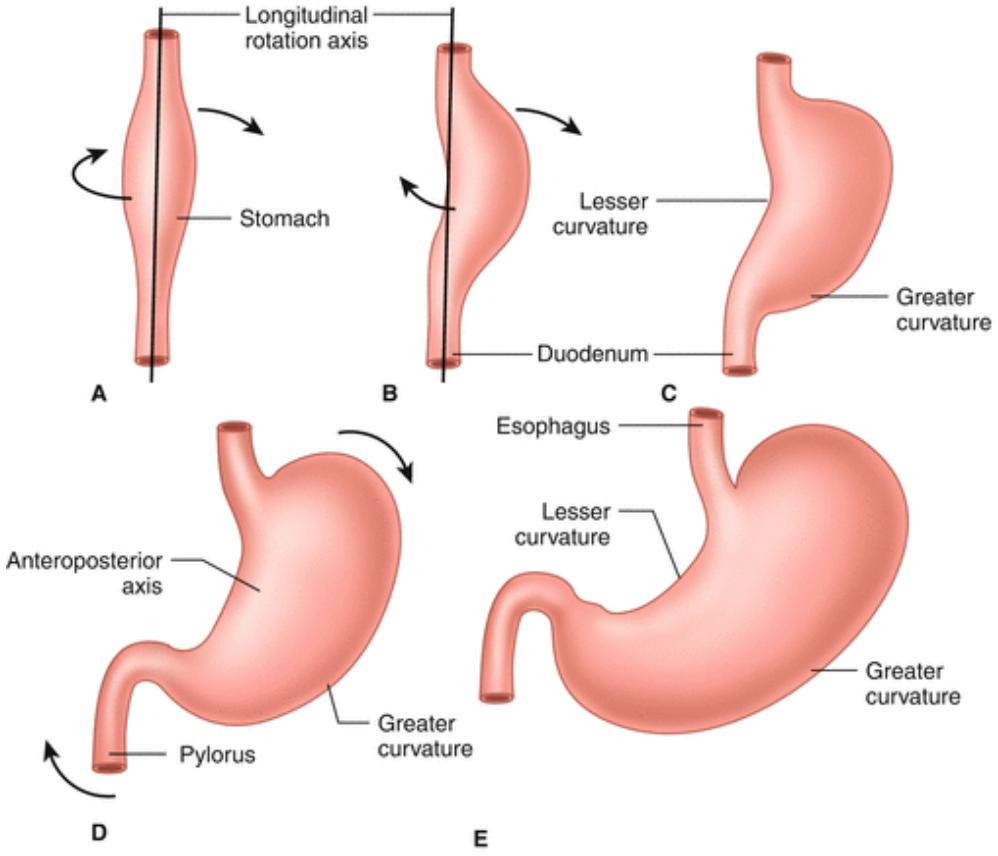


Figure 3: Embryology of the stomach

Blood supply to stomach

The stomach is a highly mobile and distensible organ composed of five different cell types that function at high metabolic rates, supported by multiple muscle layers that facilitate vigorous peristalsis during the second phase of digestion. Its primary arterial blood supply comes from the celiac trunk, which branches anteriorly from the aorta and supplies the common hepatic artery (CHA), splenic artery, and left gastric artery (LGA). The LGA's

descending branch supplies the lesser curvature of the stomach, while its ascending branch provides blood to sections of the esophagus.[9]

The CHA, "which runs superior to the pancreas and to the right, splits off to the gastroduodenal artery (GDA), and the branch that continues from the CHA is the correct hepatic artery. The appropriate hepatic artery then branches into the right gastric artery (RGA). The RGA then goes from right to left across the lesser curved region of the stomach, branching into smaller vessels as it travels through the stomach body to join the network of smaller arteries feeding the stomach that have branched off from the LGA. The GDA branches into the posterior superior pancreatico-duodenal artery (PSPDA), which then branches into the anterior superior pancreatico-duodenal artery (ASPDA), and the right gastro-omental (gastroepiploic) artery (RGEA)."

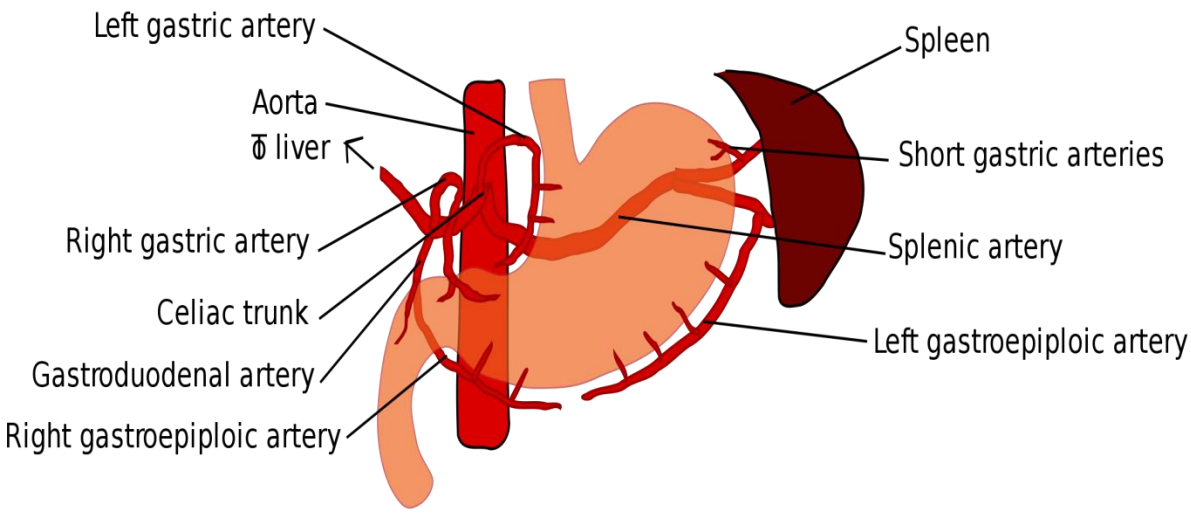


Figure 4: Blood supplyof the stomach

The right gastroepiploic artery (RGEA) crosses and supplies the greater curvature of the stomach from right to left, while the left gastroepiploic artery (LGEA), branching from the splenic artery, serves the same region but moves from left to right. Additionally, three to five minor arteries branch from the splenic artery to nourish the stomach. Venous drainage involves the left gastric (coronary) vein, right gastric vein, and right gastro-omental vein, all of which drain into different sections of the portal vein. The splenic vein is responsible for draining the short gastric veins (vasa brevia) and the left gastro-omental vein.

Lymphatic drain

The lymphatic drainage of the stomach occurs in four stages. Level 1 consists of perigastric lymph nodes, following a drainage pattern that includes the right and left pericardiac nodes, lesser and greater curvature nodes, and the supra- and infra-pyloric nodes. Level 2 involves drainage along the left gastric artery (LGA), common hepatic artery (CHA), celiac axis, splenic hilum, and splenic artery. Level 3 includes nodes within the hepatoduodenal ligament, posterior to the duodenum and pancreatic head, and at the origin of the small bowel mesentery. Finally, Level 4 is characterized by mesocolic and paraaortic lymphatic drainage.

Nerve innervations

The stomach is innervated by the autonomic nervous system via parasympathetic and sympathetic nerves. “The vagus nerve innervates the parasympathetic nervous system via the right posterior and left vagal trunks. Because the stomach rotates during development, the left vagus nerve is anterior and the right vagus nerve is posterior. For innervation of the cardia and fundus, the right vagus nerve branches to the criminal nerve of Grassi. The trunks also follow the stomach's smaller curvature to generate the posterior and anterior gastric nerves of Latarjet, which innervate the body, antrum, and pylorus. From spinal cord segments T6 through T9, sympathetic nerves supply the celiac plexus, including some fibres that convey pain.”

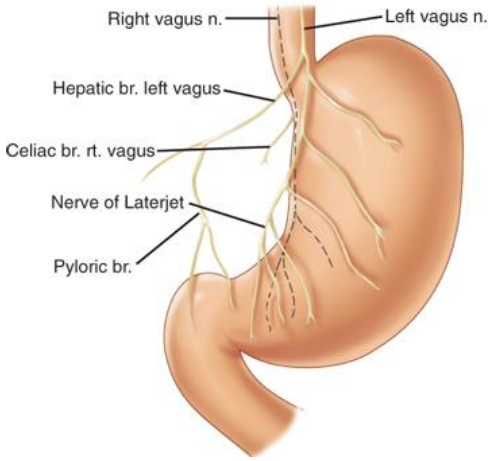


Figure 5: Innervations of stomach

Muscles of stomach

The stomach primarily consists of muscular tissue arranged in three layers—longitudinal, oblique, and circular—forming part of the stomach wall. Before examining its muscular anatomy, it is essential to understand the four major layers of the stomach wall: mucosa, submucosa, muscularis externa, and serosa. The innermost mucosa layer, lined with epithelial tissue, contains gastric glands that secrete stomach juices. The fundus is responsible for producing gastric juices, while the cardia secretes protective mucus via Foveolar cells, shielding the stomach muscles from digestion by gastric secretions, including pepsin from chief cells and hydrochloric acid (HCL) from parietal cells.

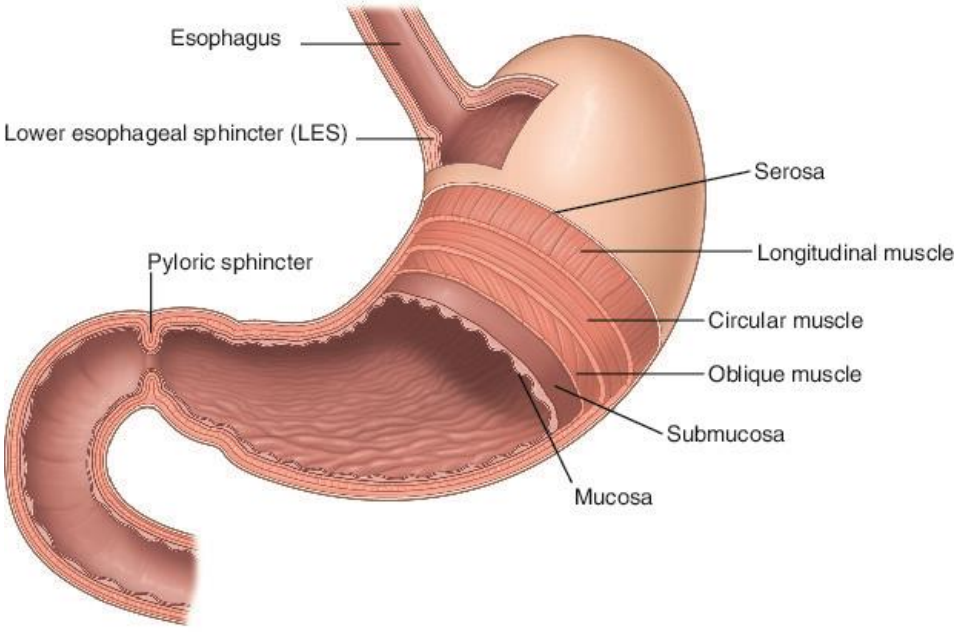


Figure 6: Muscles of stomach structure

The submucosa consists of thick connective tissue containing blood vessels, lymphatic vessels, and nerves, providing structural support to the mucosal layer. It features rugae, accordion-like folds that allow the stomach to expand as food enters. The muscularis externa follows, comprising three sub-layers: the inner oblique layer, unique to the stomach, which facilitates churning and mechanical digestion; the middle circular layer, which surrounds the stomach's longitudinal axis and thickens at the pylorus to form the pyloric sphincter, regulating the passage of food into the duodenum; and the outer longitudinal layer, which aids in peristaltic movement.

The outer longitudinal layer comes next, but between it and the middle circular layer is Auerbach's (myenteric) plexus, which is an area of innervation for the two neighbouring muscle layers. The outer longitudinal layer aids food flow in the direction of the pylorus by shortening the muscles. The last layer, the serosa, is made up of many layers of connective tissue that link to the peritoneum continually.

Physiological variants

Natural physiological changes in stomachs are limited. “The most prevalent variations are linked to the exact position, size, and form, which can be greatly influenced by nutrition. Rugae, for example, may stay swollen if a someone

consistently overeats. However, there are a number of congenital esophageal anomalies, including the following.

- Organ duplication
- Diverticula
- Organ transposition
- Bilocular contractions (horseglass)
- Gastric outlet obstruction

Cellular structure to maintain the function

The stomach wall consists of four main tissue layers: the mucosa, submucosa, muscularis externa, and adventitia/serosa. The mucosal layer is composed of surface epithelium, a connective tissue layer called the lamina propria, and the muscularis mucosa. Gastric pits and glands form as the epithelial layer invaginates into the lamina propria. The stomach glands are lined with specialized cells, including surface mucous cells (foveolar cells), parietal cells, chief cells, and neuroendocrine cells (such as G-cells or ECL-like cells), each contributing to different digestive functions".

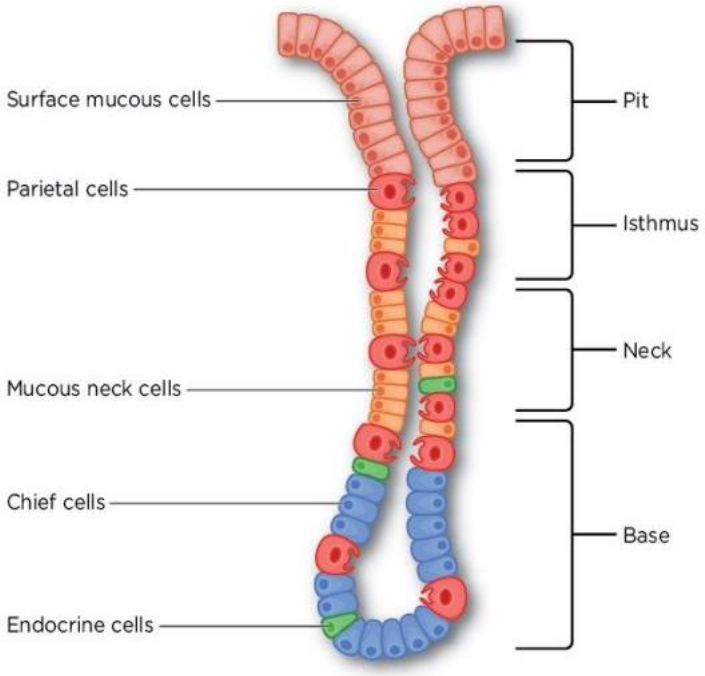


Figure 7: Structure of gastric pit

The surface mucus cells (foveolar cells) are mucus-producing cells that primarily line the gastric mucosa. The secreted mucus acts as a barrier to the corrosive nature of the gastric acid. The rest of the specialized cells are found deep within the gastric glands (i.e., gastric pits).

"Parietal cells are specialized secretory epithelial cells in the stomach, primarily located in the fundus, that produce gastric acid (HCl) and intrinsic factor, a protein essential for vitamin B12 absorption in the terminal ileum. These cells are regulated by three key molecules: acetylcholine (via muscarinic receptors), histamine (via histamine receptors), and gastrin (via gastrin receptors), all of which interact with receptors on the basal side of the cells. This regulation controls the H+/K+ ATPase protein channel on the

lumenal side, which pumps protons into the stomach lumen while absorbing potassium ions. Chloride ions then follow the proton gradient via the K+/Cl- channel, resulting in HCl production.

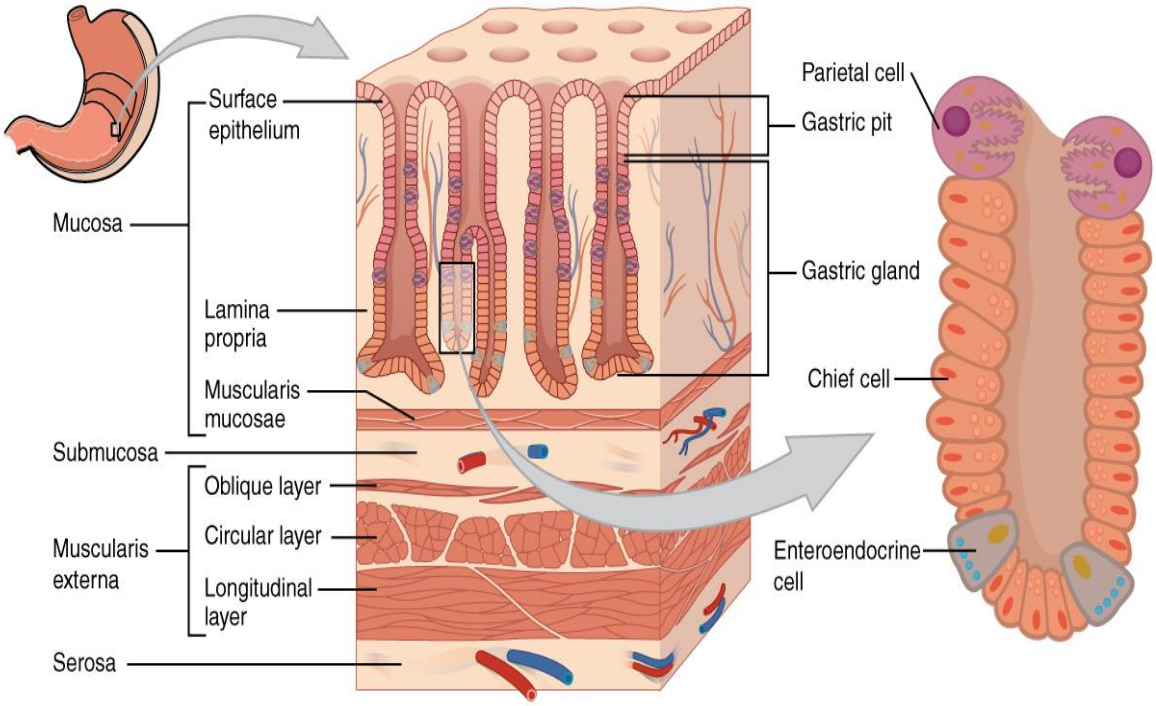


Figure 8: Stomach and gastric epithelial structure

Chief cells, located in the fundus near the base of gastric glands, are specialized secretory cells that release pepsinogen, the inactive precursor of the proteolytic enzyme pepsin. Pepsin is essential for breaking down proteins into smaller polypeptides, but pepsinogen must first be activated by gastric acid produced by parietal cells. This activation mechanism prevents unintended digestion of proteins outside the stomach lumen. Chief cells are

primarily stimulated by parasympathetic cholinergic signals and the hormone gastrin, ensuring efficient protein digestion."

Neuroendocrine cells (also known as enterochromaffin-like cells or G-cells) are present on the stomach mucosa and produce numerous chemicals that help in the formation of gastric acid.

> "When triggered by the hormone gastrin, **ECL-like cells** generate and release histamine, which indirectly boosts HCl synthesis via histamines' direct activities on parietal cells. ECL-like cells are mostly seen in the stomach fundus.
>
> **G-cells** are found in the stomach's pylorus area and create the neuroendocrine hormone gastrin. Gastrin can increase HCl production both indirectly and directly through two ways. The first method involves stimulating ECL-like cells to release Histamine, which subsequently activates Parietal cells. The second method is to stimulate the Parietal cells directly. Both processes boost the activity of the H+/K+ ATPase.
>
> **D-cells** are found in the stomach's pylorus and release an inhibitory substance known as Somatostatin. When the stomach lumen reaches a specific amount of acidity, D cells are activated. Somatostatin then suppresses gastrin release, lowering total stomach acid output."[(12)]

GASTRIC MOTOR FUNCTION

The control of gut motor function is by 3 main levels

- Parasympathetic and sympathetic nervous system
- Smooth muscle cells
- Enteric neurons and interstitial cell of cajal

Autonomic nervous system: "The vagus nerves carry extrinsic neuronal control from parasympathetic circuits to the stomach and upper intestine. Vagal efferents originate in the vagus nerve's dorsal motor nucleus and, to a lesser extent, the nucleus ambiguus and tractus solitarius. They generate distinctive bead-chain-like terminals in the stomach's myenteric plexus but do not directly innervate muscle.[13]The sympathetic supply to the stomach travels through the celiac ganglia from the intermediolateral columns of the spinal cord from T5 to T10 levels. Splanchnic efferents to the stomach have cell bodies in the celiac ganglia; they provide the myenteric ganglia, a few fibres

to the stomach's non-sphincteric muscle, and a dense supply to the pyloric sphincter".

Enteric nervous system: The enteric nervous system (ENS) is an extensive network of ganglionated plexi that integrates extrinsic gastrointestinal motility regulation—via the sympathetic and parasympathetic nervous systems—with sensory afferents in the stomach wall, which respond to luminal stimuli. These neural networks are organized into five layers throughout the gut wall, with the myenteric, deep muscular, and submucosal plexi being the most well-known. The deep muscular plexus consists of interstitial cells of Cajal (ICC), which function as pacemakers for gut wall muscle contractions, alongside fibroblast-like cells expressing platelet-derived growth factor receptor alpha (PDGFRα), another type of pacemaker cell. Additionally, neurons within the myenteric plexus may contain multiple neurotransmitters, contributing to the complex regulation of gastric motility.

Smooth muscle cells regulate gastrointestinal motility through excitable membranes that respond to neurotransmitters such as amines and peptides via neurocrine, endocrine, or paracrine pathways. These transmitters bind to specific receptors on the smooth muscle membrane, influencing contraction. Pacemaker cells, characterized by spontaneous depolarization of the resting membrane potential, generate action potentials that trigger smooth muscle contractions. The stomach muscle consists of three layers—circular, oblique,

and longitudinal—organized along different axes. Functionally, the stomach is divided into two main segments: the fundus and the antrum, with a gastric electrical pacemaker located at the midpoint of the greater curvature. During fasting, the stomach contributes to the migrating motor complex, which helps clear nondigestible solids towards the colon, a process believed to be initiated by the hormone motilin, secreted from the duodenum in animals like dogs.

Pathogenesis of delayed gastric emptying:

There are various disorders that might cause stomach motor dysfunction and, as a result, delayed gastric emptying. Different pathologic conditions may affect each of the stomach regions.

Fundus abnormalities: “A number of illnesses are related with impaired proximal gastric motor function. The accommodation response has been shown to considerably alter the pace of food emptying from the stomach, including the proximal stomach. As a result, greater accommodation is associated with a delay in gastric emptying.”

Postvagotomy dysfunction: Following vagotomy and partial gastric resection, the stomach's accommodation response and phasic contractility in response to

distention are eliminated. This explains why the liquid part of the meal is transferred to the distal stomach and beyond so quickly, whereas solids are delayed in becoming emptied.Fundoplication is one of the most prevalent causes of decreased fundal accommodation; the reduced relaxation may be exacerbated by concurrent vagal damage.Motility disturbances in function dyspepsia: Functional dyspepsia (also known as nonulcer or motility-like dyspepsia) is a condition in which patients experience nausea, early satiety, postprandial fullness, bloating, and discomfort without any clear organic pathology (eg, by upper endoscopy or upper gastrointestinal studies). Indeed, a research from the Gastroparesis Clinical Research Consortium demonstrated the interchangeable symptoms and baseline features of gastroparesis and functional dyspepsia, as well as modification in stomach emptying with time, resulting in criteria that "alter" the diagnosis.

DIABETES MELLITUS:

Type 2 diabetes mellitus a disease with huge global burden whose epidemic is already underway in both developed and developing countries. As per International Diabetes Federation approximately 463 million adults are living with diabetes already.

Diabetes mellitus - an overview

Diabetes Mellitus is defined as a group òf metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The American Diabetes Association Expert Panel recommends a diagnosis òf diabetes mellitus when one òf the three criteria ismet.[14]

Diabetes is a global pandemic. Diabetes prevalence has increased globally as a result òf changing lifestyles and rising obesity. In 2017, the global prevalence òf diabetes was 425 million. According to the International Diabetes Federation (IDF), approximately 10% òf the American population had diabetes in 2015. 7 million òf these people went undiagnosed. The prevalence òf diabetes rises as people get older. Diabetes affects approximately 25% òf the population over the age òf 65.[15]

Types of Diabetes Mellitus

Diabetes mellitus (DM) is classified into three main types based on cause and clinical presentation: type 1 diabetes, type 2 diabetes, and gestational diabetes (GDM). Less common types include monogenic diabetes and secondary diabetes.

Type 1 Diabetes Mellitus (T1DM)

- Accounts for 5-10% of DM cases.
- Caused by autoimmune destruction of insulin-producing beta cells in the pancreas, leading to an absolute insulin deficiency.
- Triggered by a combination of genetic susceptibility and environmental factors.
- Commonly occurs in children and adolescents, but can develop at any age.

Type 2 Diabetes Mellitus (T2DM)

- Accounts for around 90% of DM cases.
- Characterized by insulin resistance, where the body's response to insulin is diminished.
- Initially, the body compensates with increased insulin production, but over time, insulin production decreases.
- Typically seen in individuals over 45, but increasingly in younger people due to rising obesity, inactivity, and high-calorie diets.

Monogenic Diabetes

- Caused by a single genetic mutation in an autosomal dominant gene.
- Includes conditions like neonatal diabetes mellitus and maturity-onset diabetes of the young (MODY).
- Accounts for 1-5% of all diabetes cases.
- MODY typically presents before age 25 and is familial.

Secondary Diabetes

- Results from other diseases affecting the pancreas, hormonal disturbances, or drug use.
- Examples include pancreatitis, Cushing's disease, and corticosteroid use.

Etiologic classification of Diabetes Mellitus

- Type 1 diabetes mellitus
    - Immune mediated
    - Idiopathic
- Type 2 diabetes mellitus

- Other specific types
    - Genetic defects of cell function
        - MODY 1 (Hepatocyte nuclear transcription factor 4)
        - MODY 2 (Glucokinase)
        - MODY 3 (HNF-1)
        - MODY 6 (NeuroD1)
        - Mitochondrial DNA
        - Subunits of ATP-sensitive potassium channel
        - Proinsulin or insulin conversion
- Genetic defects in insulin action
    - Type A insulin resistance
    - Rabson mendenhall syndrome
    - Leprechaunism
    - Lipodystrophy syndromes
- Diseases of exocrine pancreas: Pancreatitis, Neoplasia, Pancreatectomy, Cystic Fibrosis, FibrocalculusPancreatopathy Hemochromatosis, Mutations In carboxyl ester lipase
- Endocrinopathies: Glucagonoma, Cushing`s syndrome, Acromegaly, Pheochromocytoma, Hyperthyroidism, Aldosteronoma and Somatostatinoma,
- Drug or chemical induced

- Infections: Coxsackie virus, Cytomegalovirus, congenital Rubella
- Other genetic syndromes: Down’s syndrome, Klinefelter’s syndrome, Friedreich’s Ataxia. myotonic dystrophy, Turner’s syndrome

Pathophysiology

In Type 1 Diabetes Mellitus (T1DM), the body's immune system attacks and destroys insulin-producing beta cells in the pancreas. This condition has a strong genetic component, with about 40-50% of familial T1DM cases linked to variations in the major histocompatibility complex (MHC) genes, specifically the class II HLA genes DQ and DR4-DQ8, and DR3-DQ2, which are present in 90% of T1DM patients.

Latent autoimmune diabetes of adults (LADA) is a slower-onset form of T1DM that occurs in adulthood. The destruction of beta cells tends to be rapid in children and slower in adults. Autoantibodies against islet cells, insulin, GAD-65, and ZnT8 may be detected in patients but decline over time and are not reliable for diagnosis after the first year. Patients with T1DM are generally not obese and are at higher risk for other autoimmune diseases like Addison's disease, Graves' disease, Hashimoto's thyroiditis, and celiac disease. A subset called idiopathic T1DM, which lacks insulin autoimmunity

and HLA association, is more common in African and Asian populations and often presents with episodic diabetic ketoacidosis (DKA).

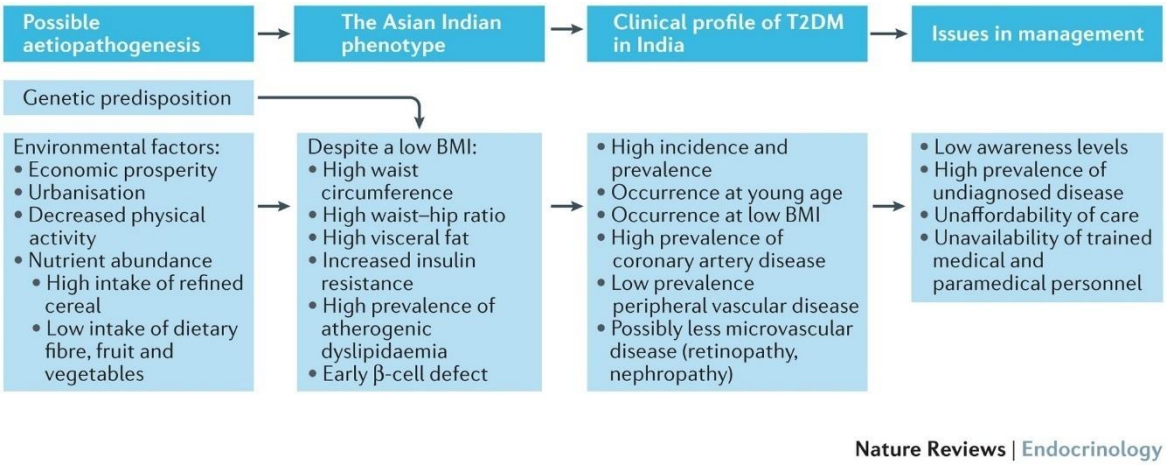


Figure 9: Distinctive features of T2DM in India[24]

Type 2 Diabetes Mellitus (T2DM) is characterized by insulin resistance and beta-cell dysfunction. Initially, increased insulin secretion compensates to maintain normal glucose levels, but over time, this compensation fails, leading to hyperglycemia. Most T2DM patients are obese or have a high percentage of abdominal fat, which promotes insulin resistance through inflammatory mechanisms like increased free fatty acid release and adipokine dysregulation. Additional risk factors for T2DM include lack of physical activity, a history of gestational diabetes, hypertension, and dyslipidemia. Emerging research points to roles for adipokine dysregulation, inflammation, abnormal incretin biology, hyperglucagonemia, increased renal glucose reabsorption, and gut microbiota abnormalities in the development of T2DM.

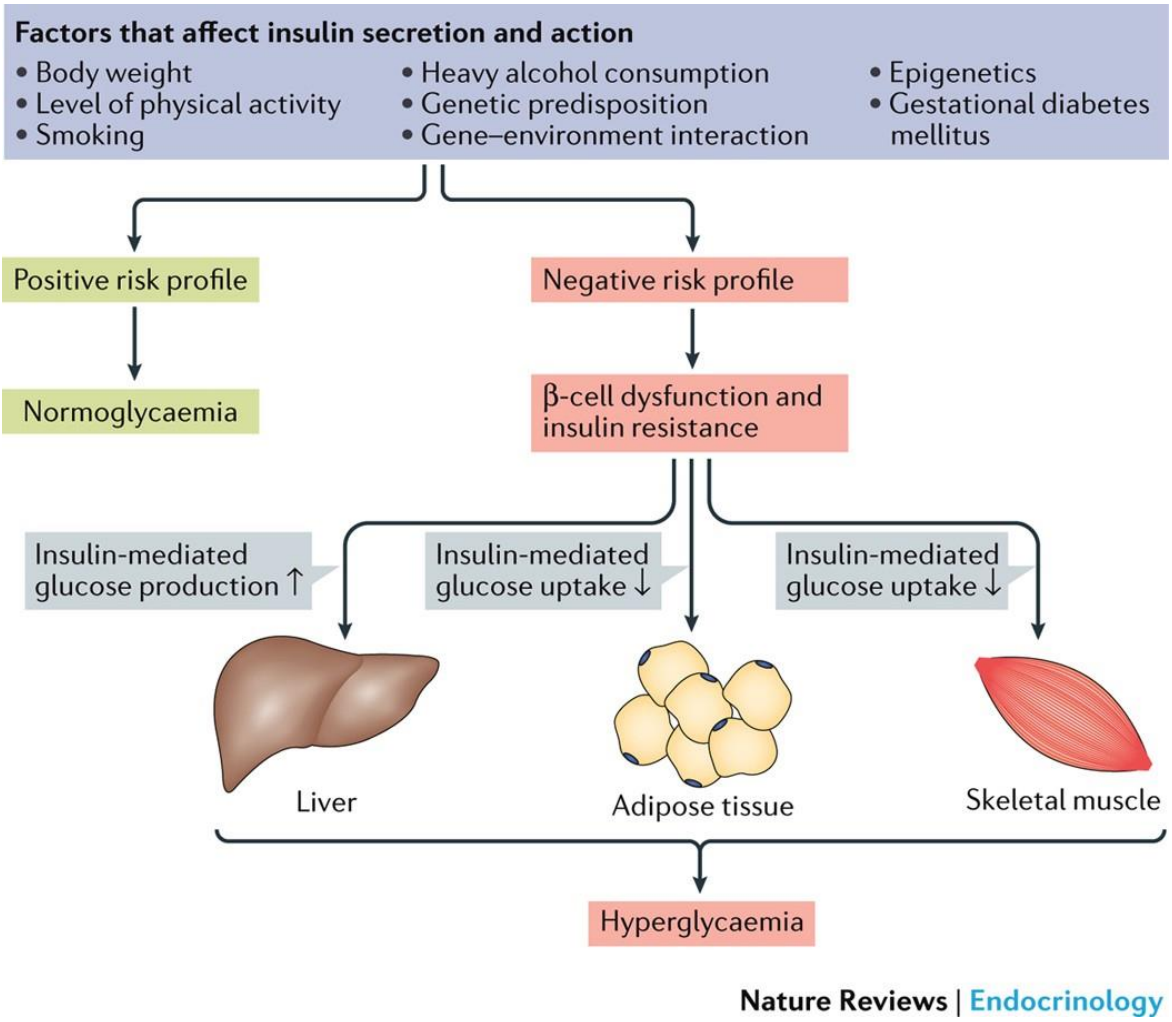


Figure 10: Factors affecting insulin secretion[16]

Genetic Susceptibility in Type 2 Diabetes

Type 2 diabetes is a polygenic disease influenced by numerous genetic factors and their interaction with environmental conditions. Key observations highlighting genetic influence include:

- 39% of patients have at least one parent with the disease.
- Approximately 90% of monozygotic twins with one affected twin eventually both develop the disease.

- First-degree relatives have a 5-10 times higher lifetime risk compared to those without a family history.

Large-scale genome-wide association studies have identified over 1000 genetic signals associated with type 2 diabetes, with polygenic risk scores combining thousands of markers to predict disease risk. Despite these advances, the impact of individual genetic variants is small, and environmental factors still play a crucial role. “The Diabetes Prevention Program showed that lifestyle changes significantly reduce diabetes risk, even among those with high genetic susceptibility.

T2DM is characterised by

- Reduced glucose uptake due to Muscle insulin resistance
- Increase in hepatic sensitivity to glucagon
- Increased glucose production due to Hepatic insulin resistance leading
- Increased insulin resistance stimulates the adipocytokine release
- increase in plasma free fatty acids due to Adipocyte insulin resistance
- Progressive beta-cell failure
- Hyperglucagonemia
- Increased renal glucose reabsorption
- Impaired incretin effect (GLP-1 and GIP)

- Brain neurotransmitter dysfunction leading to failure of appetite suppression resulting in weight gain

Clinical features

Clinical features	Type 1 diabetes mellitus	Type 2 diabetes mellitus
Age of diagnosis (years)	Majority <25, but may occur at any age	Typically>25 but incidence is increasing in adolescents, paralleling increasing rates of obesity in children and adolescents¶
Weight	Usually thin, but with obesity epidemic overweight and obesity at diagnosis becoming more common	>90% at least overweight
Autoantibodies	Present	Absent
Insulin dependent	Yes	No
Insulin sensitivity	Normal when controlled	Decreased
Family history of diabetes	Infrequent (5 to 10%)	Frequent (75 to 90%)
Risk of diabetic ketoacidosis	High	Low

Diagnosis

American Diabetes Association Criteria for the diagnosis òf Diabetes Mellitus

1. A1C ≥6.5%. The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.* **OR**
2. FPG ≥126 mg/dL (7 mmol/L). Fasting is defined as no caloric intake for at least 8 hours.* **OR**
3. 2-hour plasma glucose ≥200 mg/dL (11.1 mmol/L) during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.* **OR**
4. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L).

Diabetic Gastroparesis:

Gastroparesis is characterized by a significant delay in gastric emptying in the absence of any structural blockage." A notable complication of poorly managed diabetes, diabetic gastroparesis arises due to impaired coordination and function of the autonomic nervous system, neural pathways, and the stomach's specialized pacemaker cells, known as interstitial cells of Cajal (ICC). Additionally, dysfunction in the smooth muscle cells of the

gastrointestinal tract further contributes to the condition, disrupting normal digestive processes.[17–20]

Elevated blood glucose levels exceeding 200 mg/dL, a hallmark of poorly controlled diabetes, have been linked to diabetic gastroparesis due to neuropathic damage caused by prolonged hyperglycemia. Unlike acute hyperglycemia, which can temporarily slow gastric emptying but often improves with better glycemic management, chronic hyperglycemia-induced neuropathy persists even after glucose levels are controlled.[21–23]

The process of gastric emptying relies on the precise coordination of fundal tone, rhythmic antral contractions, and the simultaneous relaxation of the pylorus and duodenum. This complex mechanism is regulated by interactions between the enteric and autonomic nervous systems, smooth muscle cells, and the stomach's specialized pacemaker cells, known as myenteric interstitial cells of Cajal (ICCs). In diabetes, gastric motor dysfunction can result from multiple factors, including autonomic neuropathy affecting both sympathetic and parasympathetic pathways, enteric neuropathy impacting excitatory and inhibitory neurons, ICC dysfunction, acute fluctuations in blood glucose levels, the use of incretin-based medications, and psychosomatic influences. Consequently, most individuals with diabetes experience disruptions at multiple stages of gastric emptying, manifesting as

impaired postprandial gastric accommodation, weakened antral contractions, and overall dysmotility.

Pathogenesis: Diabetic gastroparesis arises from dysfunction within the autonomic and enteric nervous systems, primarily due to prolonged hyperglycemia or inefficient glucose uptake, which leads to neuronal damage. This damage disrupts myenteric neurotransmission, particularly involving the vagus nerve, impairs inhibitory nitric oxide signaling, and affects the function of smooth muscle and pacemaker cells (interstitial cells of Cajal). As a result, patients experience weakened antral contractions, uncoordinated antro-duodenal motility, and pyloric spasms, collectively contributing to delayed gastric emptying.

In addition to gastric dysmotility, abnormal small bowel motility may also contribute to delayed digestion, likely through mechanisms similar to those affecting the stomach. Some individuals with diabetes may experience altered gastric compliance, either increased or decreased, further influencing the rate of gastric emptying.

Postprandial glucose levels have a direct impact on gastric motility. In diabetic patients with autonomic neuropathy, acute hyperglycemia enhances gastric electrical activity. Conversely, in individuals without neuropathy—both diabetic and healthy—elevated blood glucose levels cause relaxation of

the proximal stomach while suppressing gastric electrical activity. This suppression reduces the frequency, propagation, and strength of antral contractions in both fasting and postprandial states, leading to delayed gastric emptying.

Acute hyperglycemia has also been associated with heightened gastrointestinal sensitivity, potentially explaining the common symptoms of postprandial dyspepsia in diabetic gastroparesis, including early satiety, nausea, vomiting, heartburn, bloating, and abdominal pain.

Furthermore, the rate of gastric emptying plays a crucial role in carbohydrate absorption by regulating the release of glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP). Slower gastric emptying results in prolonged carbohydrate absorption, leading to higher serum glucose levels, which in turn exacerbates gastroparesis symptoms, creating a vicious cycle of delayed digestion and worsening glycemic control.

Diabetic Autonomic Neuropathy

Diabetic autonomic neuropathy (DAN) is a frequently overlooked complication of diabetes, despite its profound impact on both survival and quality of life. As a subset of the peripheral polyneuropathies associated with diabetes, DAN affects the autonomic nervous system (ANS), which regulates vital functions across multiple organ systems, including the cardiovascular, gastrointestinal, genitourinary, sudomotor, and ocular systems. The ANS comprises both the sympathetic and parasympathetic divisions, and DAN typically presents as a widespread disorder that disrupts the function of both. Given that the vagus nerve—responsible for nearly 75% of parasympathetic activity—is among the longest nerves in the body, DAN initially affects longer nerves, leading to extensive dysfunction even in its early stages.[24]

Symptoms of autonomic neuropathy often emerge years after diabetes onset. While signs of autonomic dysfunction are relatively common, they are not always indicative of true neuropathy. However, subclinical autonomic dysfunction can develop within a year of diagnosis in type 2 diabetes and within two years in type 1 diabetes. Among the various forms of DAN, cardiovascular autonomic neuropathy (CAN) is the most clinically significant and well-researched due to its strong association with cardiovascular mortality. Over the past two decades, the introduction of simple, noninvasive

cardiovascular autonomic function tests has facilitated extensive clinical and epidemiological research on CAN. These studies provide compelling evidence for the importance of early detection and ongoing monitoring of autonomic impairment in diabetes management.

GI autonomic neuropathy: Gastrointestinal (GI) symptoms are prevalent among individuals with diabetes and often indicate underlying diabetic GI autonomic neuropathy (DAN). However, while GI symptoms are common in this population, they are not always directly linked to autonomic dysfunction. The manifestations of GI autonomic neuropathy vary widely and are categorized based on the affected section of the GI tract. These include esophageal enteropathy, which involves disordered peristalsis and abnormal lower esophageal sphincter function, and gastroparesis diabeticorum, a non-obstructive impairment of gastric motility characterized by abnormal gastric rhythms (bradygastria or tachygastria) and pylorospasms. Additionally, diabetic GI neuropathy can lead to altered bowel motility, resulting in diarrhea due to bacterial overgrowth or increased secretory activity, as well as constipation caused by dysfunction of intestinal neurons and a diminished gastrocolic reflex. Other manifestations include fecal incontinence due to impaired rectal sensation and abnormal sphincter function, as well as gallbladder atony and enlargement.[24,25]

Esophageal dysfunction in diabetes is primarily attributed to vagal neuropathy, leading to symptoms such as heartburn and difficulty swallowing solids. Studies using radioisotopic techniques have shown that nearly half of patients with long-standing diabetes experience delayed gastric emptying, or gastroparesis. Gastric motility is heavily reliant on vagus nerve function, which is often disrupted in diabetes. While many cases of diabetic gastroparesis are asymptomatic, severe cases can be highly debilitating, presenting with early satiety, anorexia, nausea, vomiting, bloating, and epigastric discomfort. These symptoms may persist for extended periods, ranging from days to months, or occur in cycles.

Diarrhea is reported in approximately 20% of diabetic patients, particularly those with established DAN. It often manifests intermittently but can be severe, with bowel movements occurring up to 20 times per day, frequently presenting as watery stools. Bacterial overgrowth, resulting from intestinal stasis, is a contributing factor, and broad-spectrum antibiotics such as tetracycline or metronidazole may provide symptomatic relief. Constipation, characterized by fewer than three bowel movements per week, may also alternate with episodes of diarrhea. Managing diabetic diarrhea, with or without constipation, should prioritize prokinetic agents rather than constipating medications, which may exacerbate the cycle of irregular bowel movements. Additionally, fecal incontinence, commonly linked to impaired

anal sphincter tone, is prevalent among individuals with diabetes and may either accompany severe, episodic diarrhea or occur as an independent anorectal dysfunction.

DEXLANSOPRAZOLE

Dexlansoprazole is a proton pump inhibitor (PPI) used to treat conditions caused by excess stomach acid, such as erosive esophagitis and gastroesophageal reflux disease (GERD). By reducing gastric acid production, it helps relieve heartburn and prevent esophageal damage. Available by prescription, it is marketed as Dexilant® and Kapidex® in delayed-release capsule form, while Dexilant Solutab® was withdrawn from the US market in 2017.[26]

Structure and pharmacological properties:

Molecular formula - $C_{16}H_{14}F_3N_3O_2S$

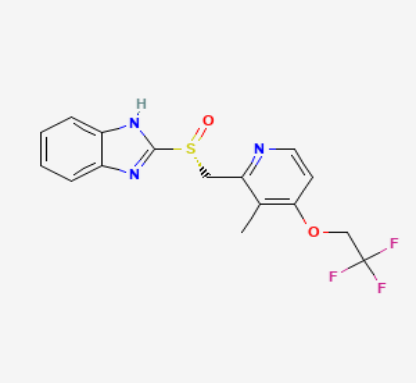


Figure 11: Chemical structure of dexlansoprazole

Molecular weight: 369.4g/mol

Dexlansoprazole is an enantiomer of lansoprazole, specifically designed to provide extended acid suppression through a dual delayed-release formulation.

Dexlansoprazole is a sulfoxide and a member of benzimidazoles. Dexlansoprazole is a new-generation proton pump inhibitor (PPI) used for the management of symptoms associated with gastroesophageal reflux disease (GERD) and erosive esophagitis. Dexlansoprazole is the R-enantiomer of [DB00448], which is composed of a racemic mixture of the R- and S-enantiomers. Compared to the older generation of PPIs (which includes [DB00213], [DB00338], and [DB00448]), dexlansoprazole has a unique pharmacokinetic profile due to its delayed-release and dual-delivery release system: This aims to address some limitations of the older-generation PPIs, such as short plasma half-life and the need for meal-associated dosing. Dexlansoprazole inhibits the final step in gastric acid production by blocking the (H+, K+)-ATPase enzyme.

Dexlansoprazole is the R-isomer of lansoprazole and a substituted benzimidazole prodrug with selective and irreversible proton pump inhibitor

activity. As a weak base, dexlansoprazole accumulates in the acidic environment of the secretory canaliculus of the gastric parietal cell where it is converted to an active sulfenamide form that binds to cysteine sulfhydryl groups on the luminal aspect of the proton pump hydrogen-potassium adenosine triphosphatase (H+/K+ ATPase), thereby inhibiting the pump's activity and the parietal cell secretion of H+ ions into the gastric lumen, the final step in gastric acid production.The R-isomer of lansoprazole that is used to treat severe gastroesophageal reflux disease.

Its pharmaceutical form utilizes a dual-release mechanism, allowing the active ingredient to be released in two phases at different pH levels and time intervals. This results in two peak serum concentrations and an overall serum level that is three times higher than its left-handed enantiomer. Additionally, dexlansoprazole has a slower elimination rate than S-lansoprazole, leading to prolonged acid suppression.[27,28] The dual-release formulation consists of two types of granules in the Dexilant capsule: 25% of the dose is released at a pH of 5.5 in the proximal duodenum, while the remaining 75% is released at a pH of 6.75 in the distal small intestine. This mechanism allows dexlansoprazole to reach peak concentrations at both 1–2 hours and 4–5 hours post-administration, ensuring extended drug retention in circulation and superior proton pump inhibition compared to other PPIs. Unlike traditional PPIs, which require administration 30–60 minutes before a meal to effectively

inhibit active proton pumps, dexlansoprazole's efficacy is independent of meal timing. This flexibility significantly improves patient adherence, as strict timing requirements are a common reason for non-compliance with PPI therapy.[29,30] Studies indicate that only 40% of patients adhere to recommended dosing schedules, leading to suboptimal acid suppression and therapeutic failure. This discrepancy highlights the difference in PPI efficacy between controlled clinical trials, where patients are closely monitored, and real-world clinical practice, where adherence is often inconsistent. Dexlansoprazole addresses this issue by offering an effective, patient-friendly alternative that does not rely on strict dosing schedules.

The efficacy of treatment with dexlansoprazole in clinical trials tends to be higher than in daily practice due to better patient compliance. A key advantage of dexlansoprazole is that its administration—whether before or after breakfast, lunch, dinner, or an evening snack—does not significantly impact its ability to control intragastric pH throughout the day. Studies have shown that the percentage of time during a 24-hour period in which stomach pH remains above 4 is relatively consistent across different meal timing regimens: 71% before breakfast, 74% before lunch, 70% before dinner, and 64% before an evening snack. This highlights the prolonged therapeutic effect of dexlansoprazole compared to single-release PPIs, making it more effective regardless of meal timing.[31,32] The ability of a PPI to suppress hydrochloric

acid secretion is best measured by the duration within a 24-hour period that intragastric pH remains above 4. Maintaining this pH threshold is crucial, as it significantly reduces pepsin activity, which is otherwise activated in an acidic environment and contributes to mucosal damage. By minimizing pepsin's destructive effects, dexlansoprazole promotes the healing of erosions and ulcers in the upper gastrointestinal tract while also alleviating symptoms associated with acid-related disorders.[33,34]

Mechanism of Action

As a PPI, dexlansoprazole inhibits the hydrogen-potassium ATPase (proton pump) in gastric parietal cells, reducing acid secretion. This helps prevent acid reflux and promotes healing in conditions like erosive esophagitis.

Dexlansoprazole suppresses gastric acid secretion by blocking the final step of acid production. It inhibits the H/K ATPase at the secretory surface of the gastric parietal cell, which is involved in the secretion of hydrochloric acid. H/K ATPase is a proton pump responsible for hydrolyzing ATP and exchanging H+ ions from the cytoplasm for K+ ions in the secretory canaliculus: this action results in hydrochloric acid secretion into the gastric lumen.

Pharmacokinetics

Dexlansoprazole utilizes a dual delayed-release mechanism, allowing a prolonged duration of action. It is primarily metabolized in the liver and eliminated through urine and feces. Due to its extended release, it provides sustained acid suppression throughout the day.

- **Erosive Esophagitis (EE):**
    - Adults and children (≥12 years): 60 mg once daily for up to 8 weeks. Maintenance therapy: 30 mg once daily for up to 6 months.
    - Not recommended for children under 2 years due to potential heart-related risks.
- **GERD:**
    - Adults and children (≥12 years): 30 mg once daily for 4 weeks.

Side Effects

Common side effects include diarrhea, nausea, headache, abdominal pain, and bloating. Serious adverse effects may include kidney issues (acute interstitial nephritis), hypomagnesemia, vitamin B12 deficiency, bone

fractures, and increased risk of lupus or skin reactions like Stevens-Johnson syndrome. Long-term use may also contribute to fundic gland polyps.

Drug Interactions

Dexlansoprazole interacts with several medications:

- **Contraindicated:**Rilpivirine (due to severe interactions).
- **Use with caution:** Amphetamines, atazanavir, ketoconazole, methotrexate, and warfarin, among others.
- **Food and Lifestyle Interactions:** Alcohol and cranberry may increase side effects.

Toxicity

Overuse or prolonged use of dexlansoprazole may lead to severe electrolyte imbalances, kidney issues, and bone fractures. Hypomagnesemia, seizures, and cardiac arrhythmias may occur in extreme cases. Proper dosage adherence and periodic monitoring are advised to prevent complications.

A study by Sharma S et al. (2018) evaluated the "effectiveness of standard fasting guidelines using gastric ultrasound and found that 28.04% of 246 patients had high residual gastric volume, with no significant correlation between fasting duration and gastric volume (P = 0.47). However, a linear relationship was observed between increasing BMI and residual gastric volume (P < 0.0001), and patients with GERD had a 2.3 times higher risk. Among CKD patients, 30% had large residual stomach capacity, though no cases of aspiration were reported. The study concluded that risk factors had a greater impact on residual gastric volume than fasting duration, suggesting that while current fasting guidelines are adequate for healthy individuals, they may be insufficient for those with risk factors. In such cases, preoperative gastric volume assessment via ultrasound serves as a valuable screening tool."[35]

A study by Ohashi Y et al. (2018) examined "preoperative gastric residual volume (GRV) in fasted patients and found no significant association between 'at risk' GRV and factors such as obesity, diabetes mellitus, gastroesophageal reflux disease, or opiate use, though the study lacked sufficient power to rule out their effects. Despite adherence to fasting guidelines, a small number of patients still had GRVs posing a pulmonary aspiration risk, highlighting the need for anaesthetists to consider this

background incidence when planning anaesthetic procedures. While further research is necessary, preoperative bedside gastric ultrasonography may be a useful tool for identifying patients with 'at risk' GRVs."[36]

An observational study by Sharma G et al. (2018) evaluated "preoperative gastric content and volume using bedside ultrasound in adult patients and found that despite fasting for 10 to 15 hours, six out of 100 patients had solid stomach contents, and 16 had >1.5 ml/kg of clear liquids. Patients with diabetes and chronic kidney disease (CKD) showed a significant increase in cross-sectional area (CSA) in both supine and right lateral decubitus positions, while higher BMI was associated with increased estimated gastric capacity." The findings suggest that fasting beyond 6-10 hours does not guarantee an empty stomach, and individuals with comorbidities such as diabetes, obesity, and CKD are more likely to retain hazardous gastric contents.[37]

A study by Sabry R et al. (2019)"assessed gastric residual volume in fasting diabetic patients using gastric ultrasound and found that, compared to the control group, diabetic patients had a larger median antral cross-sectional area and a higher estimated residual gastric volume. Additionally, the diabetes group had a greater aspirated volume through the nasogastric tube, with a strong correlation between ultrasound-measured residual volume and aspirated gastric contents." Even after fasting for eight hours before elective

surgery, patients with long-standing diabetes exhibited significantly higher residual stomach capacity than healthy controls.[1]

In a study conducted by Cihang HH et al., (2019) to assess the "clinical efficacy of dexlansoprazole and esomeprazole after 24hr on demand treatment. The study found that both dexlansoprazole and esomeprazole provided similar symptom relief for GERD over 24 weeks, as indicated by lower GERDQ scores compared to baseline. Key clinical outcomes, including symptom relapse rate, sustained healing of erosive esophagitis, treatment failure rate, and medication usage, were comparable between the two groups. However, patients in the esomeprazole group experienced more days with reflux symptoms than those in the dexlansoprazole group (P=0.008)." Additionally, dexlansoprazole demonstrated a sustained improvement in GERDQ scores during the on-demand period, whereas esomeprazole did not. These findings suggest that while both medications are effective, dexlansoprazole may offer more consistent symptom control over time.[38]

A prospective cohort study by Zhou L et al. (2019) evaluated the use of point-of-care ultrasound to measure gastric content in type 2 diabetes patients undergoing elective surgery. "Among 52 diabetic and 50 non-diabetic individuals, the prevalence of a full stomach was significantly higher in diabetic patients (48.1%) compared to non-diabetics (8%) (P = 0.000), with rates of 44.0% after a 2-hour fast following clear liquids and 51.9% after a 6-

hour fast following a light meal. The average gastric emptying time in diabetic individuals was 146.50 ± 40.91 minutes for clear liquids and 426.50 ± 45.25 minutes for a light meal. Additionally, diabetes-related eye conditions were identified as an independent risk factor for a full stomach (OR = 4.83, P = 0.010). These findings suggest that despite adhering to standard preoperative fasting guidelines, nearly half of type 2 diabetes patients have a full stomach, highlighting the importance of preoperative ultrasonography for risk assessment in this population."[39]

An observational study by Garg H et al. (2020)"assessed fasting gastric volume using ultrasound in diabetic and non-diabetic patients undergoing elective surgeries. In the supine position, the craniocaudal (CC) and anteroposterior (AP) diameters were smaller in the control group compared to the diabetic group. In the right lateral decubitus (RLD) position, the CC and AP diameters were 2.28 ± 0.57 cm and 1.24 ± 0.42 cm in the control group, respectively, versus 2.54 ± 0.56 cm and 1.82 ± 0.56 cm in diabetics. The cross-sectional area (CSA) was significantly larger in diabetics (2.57 ± 1.19 cm^2 supine, 3.73 ± 1.61 cm^2 RLD) compared to controls (1.41 ± 0.55 cm^2 supine, 2.30 ± 1.18 cm^2 RLD) (P = 0.001). Gastric volume (GV) was also higher in diabetics (9.15 ± 25.70 ml) compared to controls (4.20 ± 22.26 ml). These findings suggest that diabetic patients have a larger antral CSA and

gastric volume than non-diabetic individuals, as measured by gastric ultrasonography."[2]

A study by Khalil AM et al. (2021) assessed gastric residual volume using ultrasound in fasting obese patients. "While antral cross-sectional area (CSA) differed between groups, the predicted gastric volume remained below 1.5 mL/kg in all participants across both positions. The aspiration risk was low, with 98% of patients showing an empty antrum or minimal fluid in the right lateral position (RLP), and only 2% exhibiting a distended antrum in both positions. Despite obese individuals having larger CSA values than those with normal weight, the low gastric residual volume and aspiration risk suggest that an 8-hour fasting period before elective surgery is sufficient for both groups."[40]

In a comparative study by Harmagatti A et al. (2022),"ultrasound-guided residual gastric volume measurement was assessed in diabetic and non-diabetic patients scheduled for elective surgery. Despite differences in cross-sectional area (CSA) and gastric volume (GV) between the two groups, both had low gastric residual volume (<1.5 mL/kg). The gastric tube aspirate was significantly higher in diabetic patients (1.24 ± 1.46 mL) compared to non-diabetic patients (0.3 ± 0.78 mL). Long-standing diabetes was associated with larger gastric residual volume and antral CSA, but further research is needed

to determine the clinical significance of these findings before specific recommendations can be made for diabetic individuals."[41]

In a study by Rajeswari L et al. (2022)"assessing gastric residual volume using ultrasound in fasting diabetic and non-diabetic adults undergoing elective surgery, diabetic individuals had significantly higher mean antral CSA and estimated gastric residual volume (GRV) in both right lateral and semi-sitting positions. The gastric antrum appeared empty in a greater proportion of non-diabetic patients, while diabetics had a larger mean volume of stomach aspirate. These findings suggest that current fasting guidelines for elective surgery may be inadequate for individuals with long-standing diabetes, highlighting the need for point-of-care ultrasound as an effective screening tool to assess aspiration risk and optimize anesthetic management."[42]

A study conducted by Cunha DD et al., (2022) evaluated the use of "gastric ultrasonography in assessing and quantifying gastric content in fasting diabetic and non-diabetic patients. The mean age of the diabetic group was 49.3 ±16.4 years, while the non-diabetic group had a mean age of 49.4 ±16.8 years. Ultrasonographic findings revealed that 75% of participants, regardless of their fasting status, had Grade 1 gastric content (up to 100 ml). Statistical significance was assessed using a threshold of P 0.05, with no significant correlation found between age and ultrasound results. However, a strong

association was observed between BMI and both gastric content and volume ($P<0.01$). In current clinical practice, NPO status is determined based on patient history, which may be unreliable. This inaccuracy poses a higher risk of aspiration, particularly in individuals with delayed gastric emptying. Implementing gastric ultrasonography as a preoperative screening tool before anesthesia induction and surgical procedures could help reduce unnecessary perioperative complications."[43]

In a study conducted by Chaitra T et al., (2023) to assess the "residual gastric volume using ultrasonography in adults. This study highlights the importance of preoperative gastric ultrasound in assessing gastric residual volume (GRV) to guide perioperative airway management. Despite adherence to fasting guidelines, some patients exhibited significant residual volumes (>1.5 ml/kg), indicating a potential risk for aspiration. Notably, 97 and 118 patients were observed to have distended stomachs in the supine and right lateral decubitus positions, respectively. In terms of GRV classification, 336 patients had a safe volume, while 60 were classified as having a low risk of aspiration (<1.5 ml/kg), and 13 were deemed at high risk (>1.5 ml/kg). Interestingly, some patients who fasted beyond 10 hours still had GRV exceeding 1.5 ml/kg, reinforcing the need for individualized preoperative assessment rather than reliance on fasting duration alone.The study also examined the effects of premedication on GRV, revealing that patients who

received histamine blockers had significantly higher antral cross-sectional areas and GRV compared to those premedicated with proton pump inhibitors (PPIs), suggesting that PPIs are more effective in reducing gastric volume. Additionally, an increase in BMI was significantly associated with an increase in antral CSA in both supine and right lateral decubitus positions. A strong correlation was also observed between type 2 diabetes and elevated GRV, suggesting that diabetic patients may require more rigorous preoperative evaluation.In conclusion, preoperative gastric ultrasound should be considered for patients with high BMI and diabetes to ensure safer airway management strategies." The study underscores that fasting duration alone should not be the sole determinant of aspiration risk, and the use of PPIs over histamine blockers may provide better gastric volume control in surgical patients.[44]

MATERIAL & METHOD

Source of data

This study was carried out in the Department of Anaesthesiology, B.L.D.E. (DU)

Shri. B.M. Patil Medical College,Hospital and Research center,Vijayapura. Karnataka

Method of collection of data:

Study Design: A Double-blinded comparative study.

Study Period: One and half year from April 2023 to October 2024

Sample size:

- The anticipated Mean±SD of reflux symptoms in esomeprazole group 37.3±37.8 and in dexlansoprazole group 53.9±54.2,the required minimum sample size is 92 per group (i.e. a total sample size of 184, assuming equal group sizes)to achieve a power of 80% and a level of significance of 5% (two sided),for detecting a true difference in means between two groups.[45]

$$N = 2\left[\frac{\left(Z_{\propto} + z_{\beta}\right) * S}{d}\right]^2$$

$Z_{\propto}$ Level of significance=95%

Z_{β}--power of the study=80%

d=clinically significant difference between two parameters

SD= Common standard deviation

Randomization:

The study population were assigned using computerized random number table in to two groups.

Group DL30 – This group of patients given with 30mg of Dexlansoprazole.

Group DL60 - This group of patients given with 60mg of Dexlansoprazole.

Study population:

This study was done in diabetic patients undergoing various selective surgical procedures.

Inclusion criteria:

- Patients aged between 18-80 years.
- Diabetic patients admitted for elective surgeries
- ASA I ,ASA II & ASA III

Exclusion criteria:

- BMI > 30 kg/m2
- Co-existing autoimmune diseases
- Patients with history of gastric surgeries.

- Patients unable to position in right lateral decubitus position.
- Pregnant women
- H/O Allergy to drugs

Methodology:

Pre-anaesthetic evaluation:

The Pre-anaesthetic evaluation included the following:

History:

History of underlying medical illness, previous history of surgery, anaesthetic exposure, and hospitalization was elicited.

Physical examination:

- The general condition of the patient.
- Vital signs-heart rate, blood pressure, respiratory rate.
- Height and weight.
- Examination of the respiratory system, cardiovascular system, central nervous system, and vertebral system.
- Airway assessment by Mallampati grading.
- The procedure was explained to the patient and patient attenders.

- **Investigations/interventions**

  Routine investigations include CBC, FBS, ECG, Chest

  Xray, HIV, HbsAg, Urine routine, HbA1c, UKB.

Gastric volume was measured using the formula described by perlas et al.

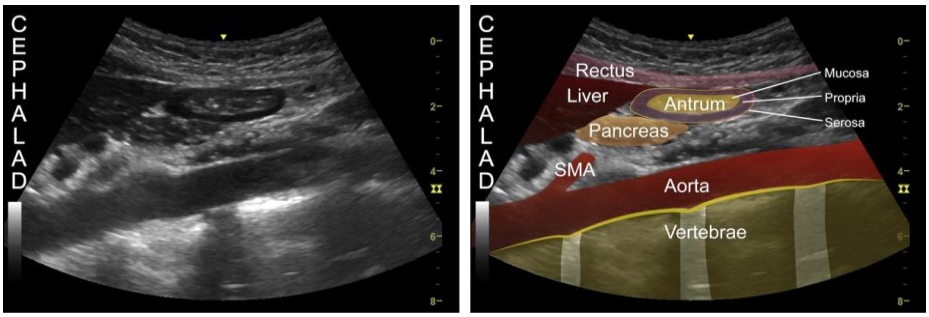
- Volume = 27.0 +14.6 x right lateral CSA -1.28 x age.

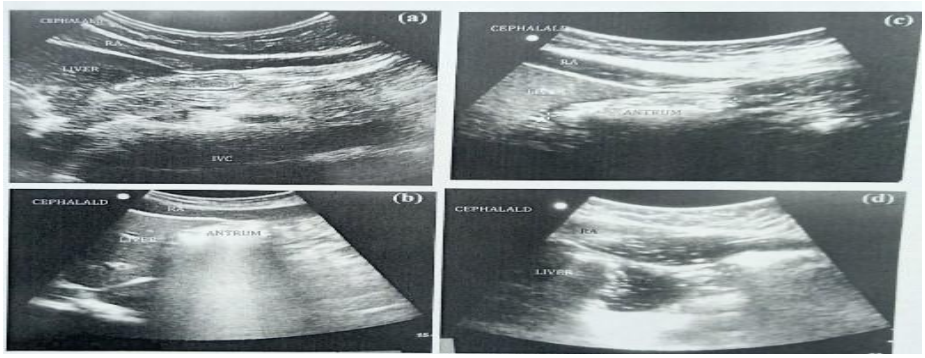
Procedure:

- " Pre anaesthetic evaluation was done in the ward.
- Patients were kept NPO (nill per oral) for more than 8 hrs overnight fasting.
- Patients were selected for the study based on the inclusion and exclusion criteria.
- The procedure was explained to the patient, and informed consent was taken.
- The patient was given oral Dexlansoprazole 30 mg or 60 mg by randomization two hours before shifting to preoperative room and they were unaware of dosage of the study drug.
- Sonosite M Turbo portable ultrasound machine was used.
- A portable curve array low-frequency abdominal probe (2–5 MHz) with abdominal pre-set was used.
- Typical antrum visualization is best found in a parasagittal plane, with the left lobe of the liver anteriorly and the head or body of the pancreas posteriorly seen as a reference point.
- The examination was performed as follows: the patients were first scanned in supine position with the head of the bed elevated to 45° (semi-

recumbent position—SRD-), followed by right lateral decubitus (RLD) with a head of the bed elevated to 45 (RLD), for greatest sensitivity.(38)

- The transducer was placed in a sagittal plane in the epigastric region. The antrum has a characteristic multi-layered wall. As a consensus, measurements were performed when the plane scanned above the large abdominal vessels (aorta or inferior vena cava).(38)
- To calculate the CSA with the two-diameter method (TDM), three still images of the antrum were obtained at rest (between peristaltic contractions) in both SRD and RLD.
- The cross-sectional area (CSA) of the antrum in the RLD is determined based on the formula for an ellipse, using two perpendicular diameters of the antrum, from serosa to serosa: the craniocaudal (CC) and anteroposterior (AP) diameters: The numerical average of the 3 measurements were recorded. Based on antral CSA, total gastric fluid volume was predicted in each patient using a previously reported mathematical model developed by Perlas et al. (where right-lateral CSA is the antral CSA measured in the RLD).(38)
- Volume = 27.0 +14.6 x right lateral CSA -1.28 x age.
- We also use of the free tracing method formula (FTM), which consists of measuring the antral area using the ultrasound unit's free tracing caliper. For the FTM, three measurements were recorded.

- The person who performed the scanning technique was blinded regarding the study drug.
- Then confirm the dosage of the Dexlansoprazole taken by the patient and was noted accordingly.
- 



a. Sonoanatomy of empty gastric antrum, Antrum appears flat or bulls eye shaped structure, RA(rectus abdominis)
b. Sonoanatomy of distended antrum, in early stage,solids resembles ground glass appearance, the posterior wall of antrum is obscured, RA(rectus abdominis)
c. Sonoanatomy of distended antrum, in late –stage,solids appear heterogenous and hyperechoic (rectus abdominis)
d. Distended antrum with clear fluids and gas having /hypoechoic appearance (starry night).

STATISTICAL ANALYSIS

The data obtained was entered in a Microsoft Excel sheet, and statistical analyses was Performed using a statistical package for the social sciences (SPSS) (Version 20). Results were presented as Mean, SD, counts and percentages, and diagrams. For normally distributed continuous variables between the two groups was compared using an independent sample t-test. For not normally distributed variables, the Mann-Whitney U test is used. Categorical variables between the two groups are compared using the Chi-square test/Fisher exact test. $p<0.05$ was considered statistically significant. All statistics are performed two-tailed."

RESULTS

Present study included total of 184 patients fulfilling inclusion criteria, who were divided into two groups as; Group DL30 – receiving 30mg of Dexlansoprazole and Group DL60 receiving 60mg of Dexlansoprazole.

Table 1: Mean age comparison between the groups

Age in yrs	DL 30	DL 60	p-value
	Mean ± SD	Mean ± SD	
	47.8 ± 6.8	49.2 ± 5.2	0.63
The mean age between the group were comparable with no significant difference noted.			

The mean age in DL 30 was 47.8yrs and 49.2yrs in DL60.

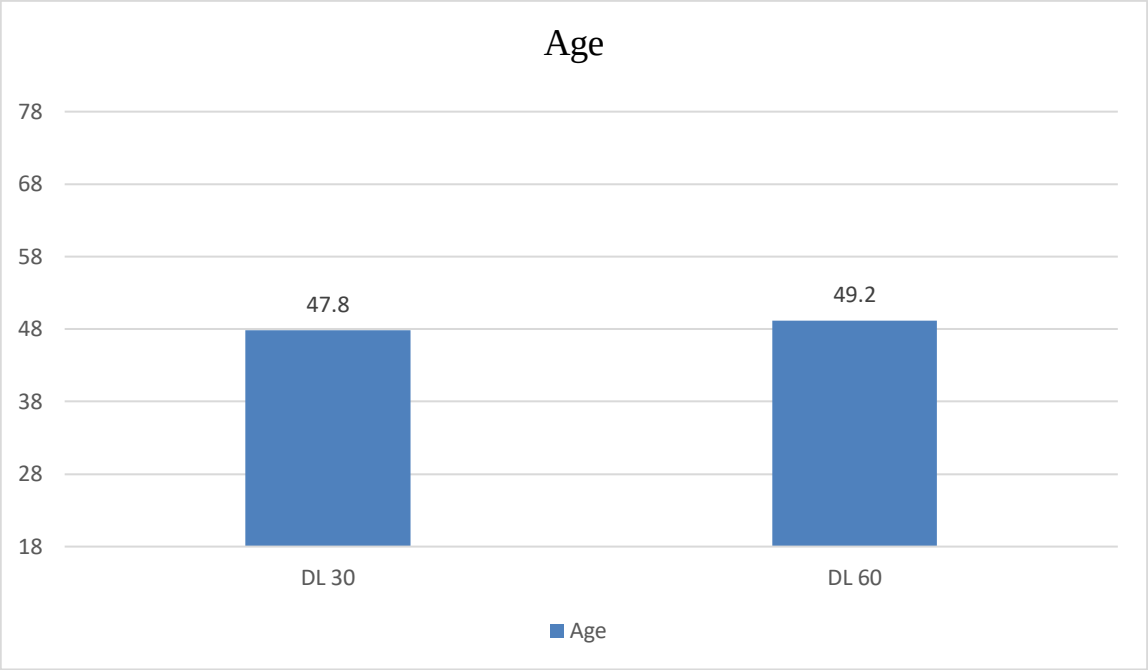


Figure 12: Mean age comparison between the groups

Table 2: Gender distribution between the groups

Gender		DL 30	DL 60	Chi-square
		Count (N %)	Count (N %)	(p-value)
	Female	22 (23.9%)	27 (29.3%)	0.69 (0.40)
	Male	70 (76.1%)	65 (70.7%)	

The gender distribution between the group was comparable with no significant difference noted.

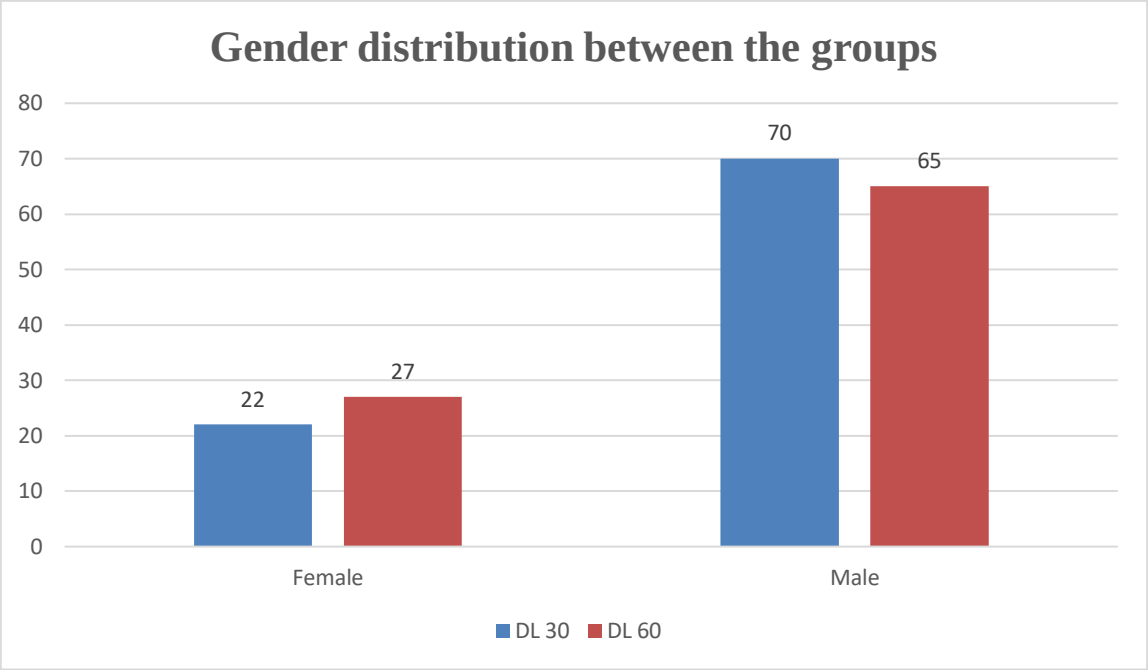


Figure 13: Gender distribution between the groups

Table 3: Comparison of ASA grade between the groups

		DL 30	DL 60	Chi-square
		Count (N %)	Count (N %)	(p-value)
ASA	1.0	4 (4.3%)	3 (3.3%)	0.152 (0.92)
	2.0	55 (59.8%)	56 (60.9%)	
	3.0	33 (35.9%)	33 (5.9%)	

The ASA grade were comparable between the groups, with no significant difference noted.

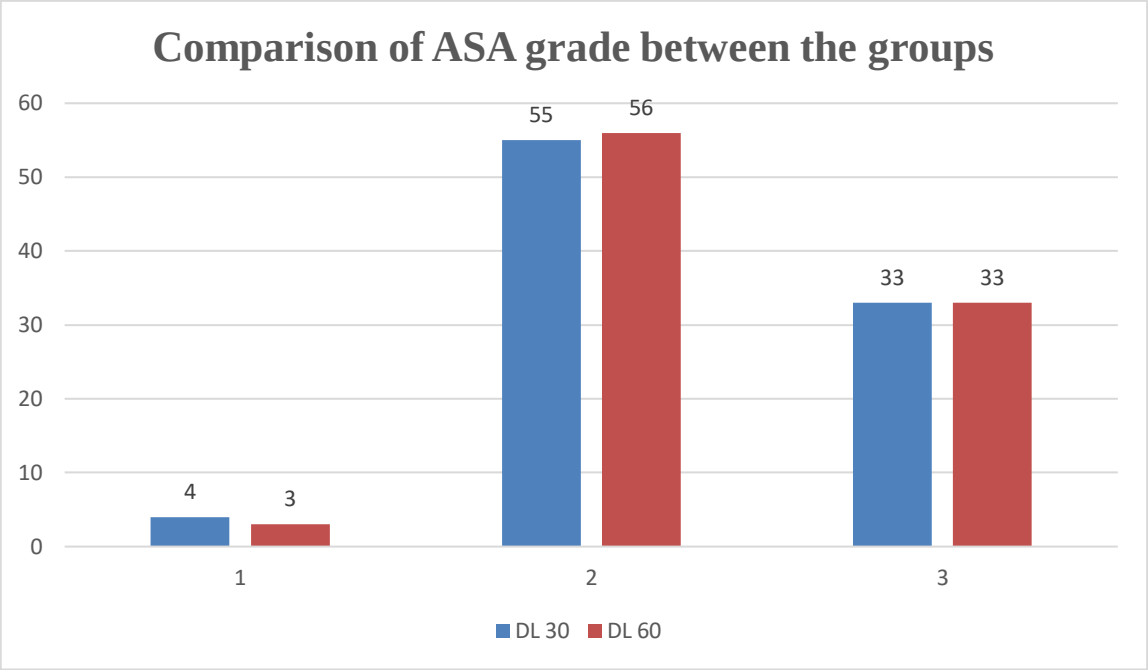


Figure 14: Comparison of ASA grade between the groups

Table 4: Comparison of physical characteristics between the groups

	DL 30	DL 60	p-value
	Mean ± SD	Mean ± SD	
Weight kg	67.9 ± 4.3	68.3 ± 3.9	0.63
Height cm	164.5 ± 3.3	166.4 ± 3.9	0.72
BMI Kg/M^2	25.13 ± 1.68	26.41 ± 1.24	0.51

The mean physical characters were found to be comparable between the groups, with no significant difference noted.

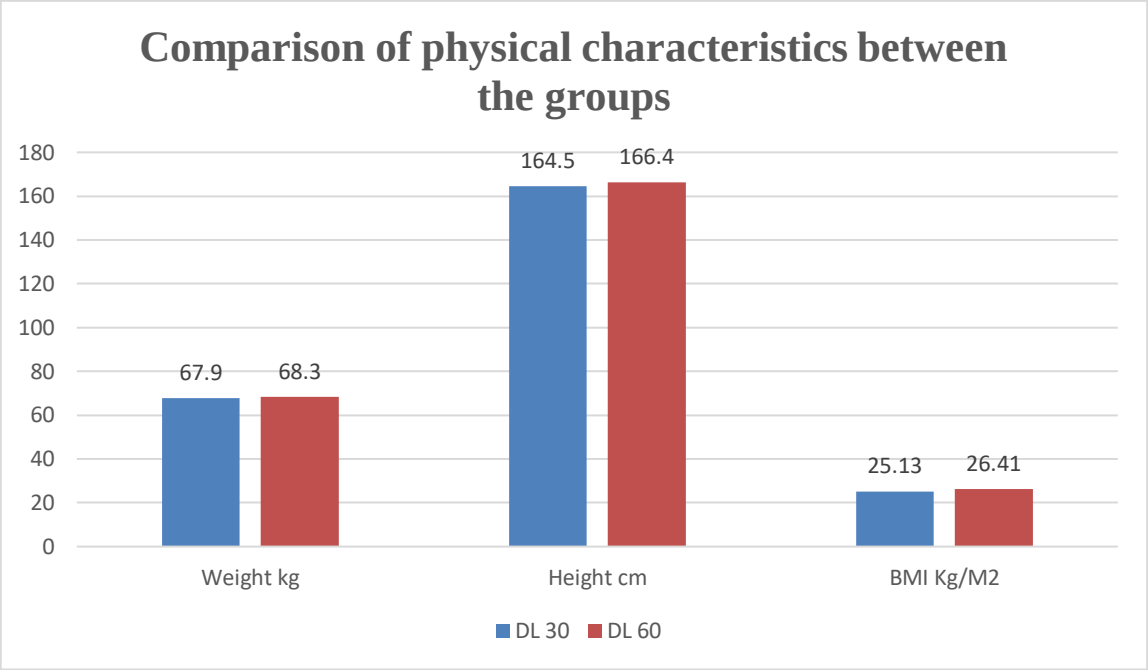


Figure 15: Comparison of physical characteristics between the groups

Table 5: Comparison of the glycemic status paramters between the groups

	DL 30	DL 60	p-value
	Mean ± SD	Mean ± SD	
Fasting Duration (in hours)	8.6 ± .9	8.1 ± 1.2	0.65
FBS (mg/dl)	107.0 ± 9.5	102.3 ± 8.6	0.71
RBS (mg/dl)	197.9 ± 19.7	206.31 ± 22.1	0.54
HbA1C(%)	8.6 ± 1.9	8.2 ± 1.65	0.68

The mean difference of glycemic parameters were found to be comparable with no significant difference noted.

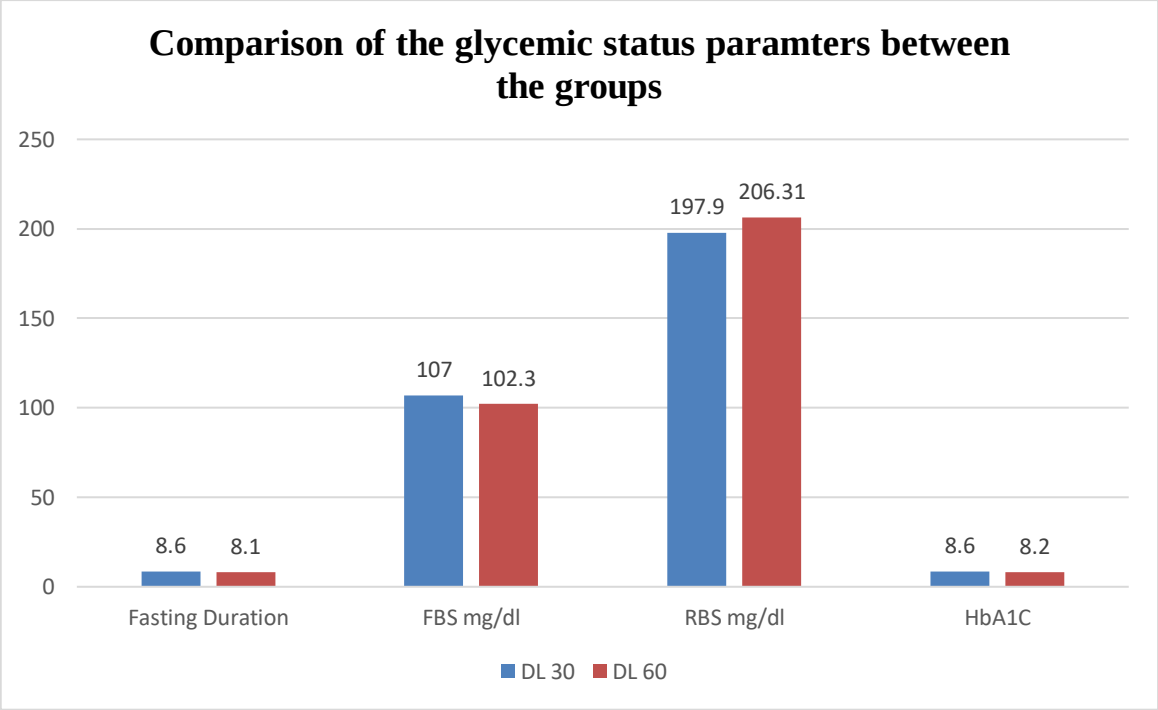


Figure 16: Comparison of the glycemic status paramters between the groups

Table 6: Showing the diameter of craniocaudal, Anteroposterior and cross sectional area of antrum measurements between the groups

	DL 30	DL 60	p-value
	Mean ± SD	Mean ± SD	
CC Diameter [supine position]	2.87 ± 0.16	2.81 ± 0.13	0.63
CC Diameter [in Right lateral position]	3.00 ± 0.18	2.84 ± 0.26	0.67

AP Diameter [supine position]	1.98 ± 0.23	1.84 ± 0.28	0.52
AP Diameter [Right lateral position]	2.15 ± 0.19	2.21 ± 0.24	0.71
CSA(cm^2) [supine position]	4.45 ± 0.62	4.62 ± 0.66	0.48
CSA(cm^2) [Right lateral position]	5.07 ± 0.71	5.3 ± 0.65	0.32

On assessment of the measurement, there is no significant difference noted between the groups. The measurement of craniocaudal diameters, anteroposterior diameter and cross sectional area

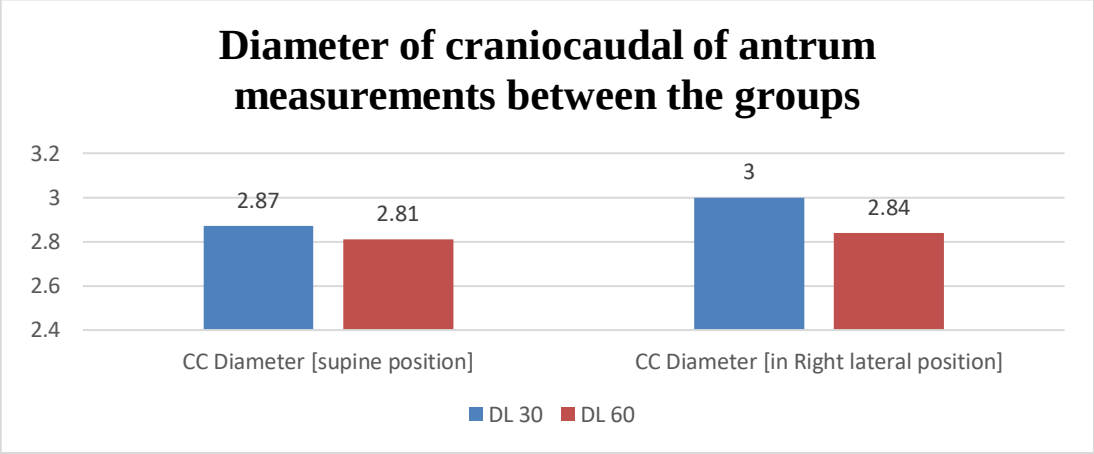


Figure 17: Diameter of craniocaudal of antrum measurements between the groups

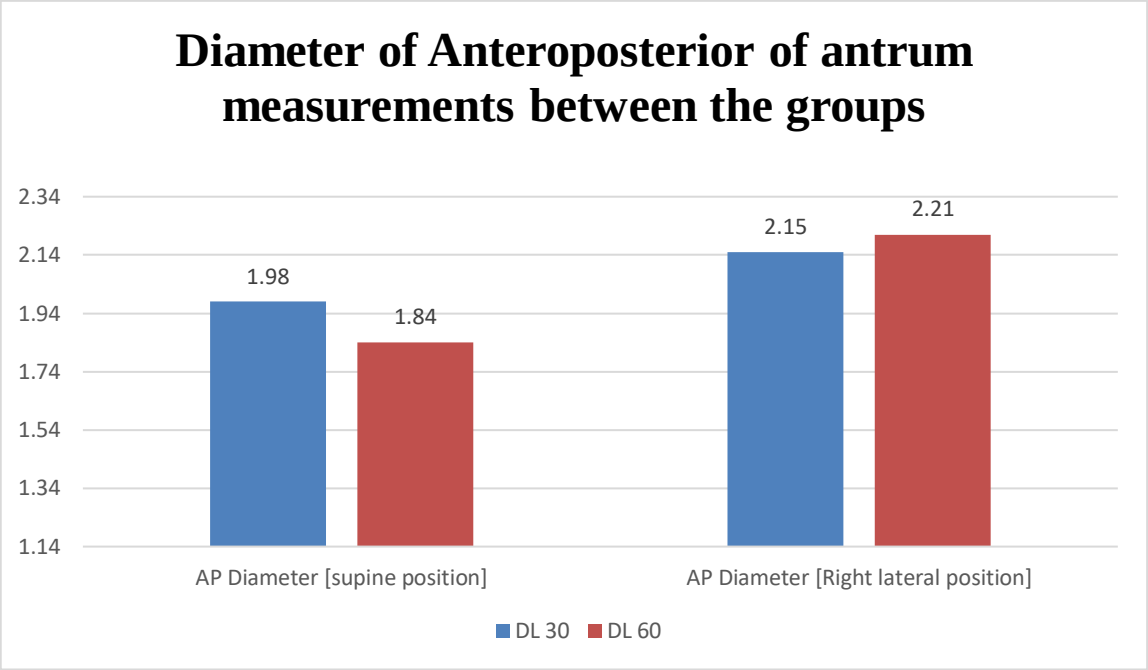


Figure 18: Diameter of Anteroposterior of antrum measurements between the groups

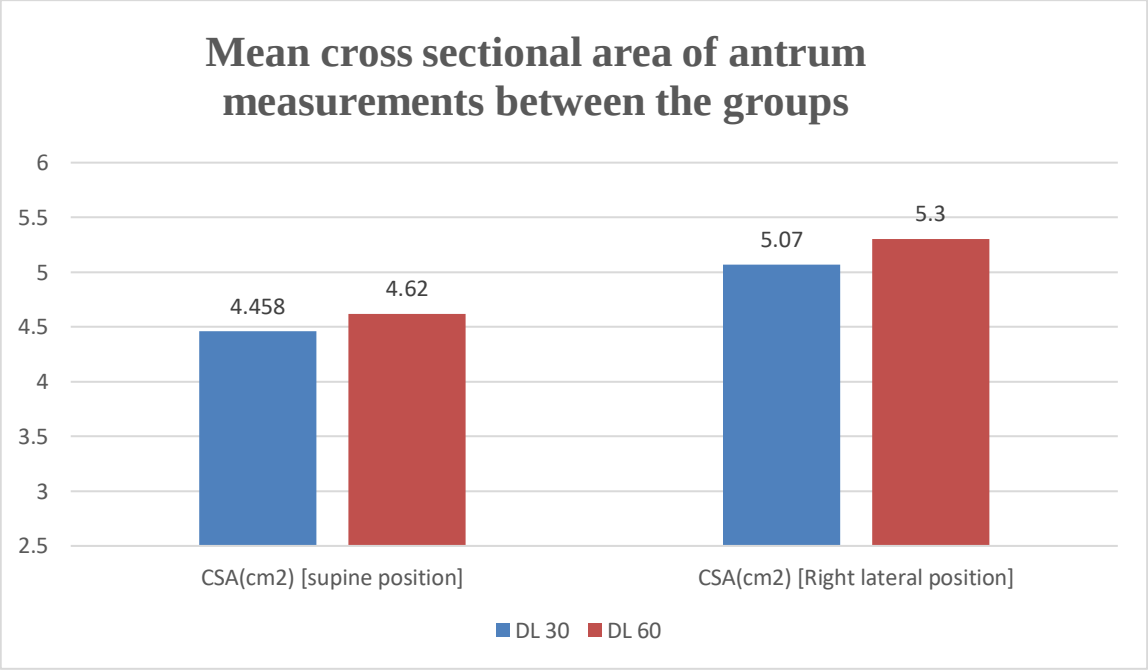


Figure 19: Mean cross sectional area of antrum measurements between the groups

Table 7: Comparison of the mean gastric volume between the groups in right lateral position

	DL 30	DL 60	p-value
	Mean ± SD	Mean ± SD	
Gastric Volume [Right lateral position]	39.72 ± 8.54	28.32 ± 7.58	0.01*

There is significant reduction of the grastric volume in the DL60 group (28.32±7.58) compared to DL30 group (39.72±8.54).($p<0.05$)

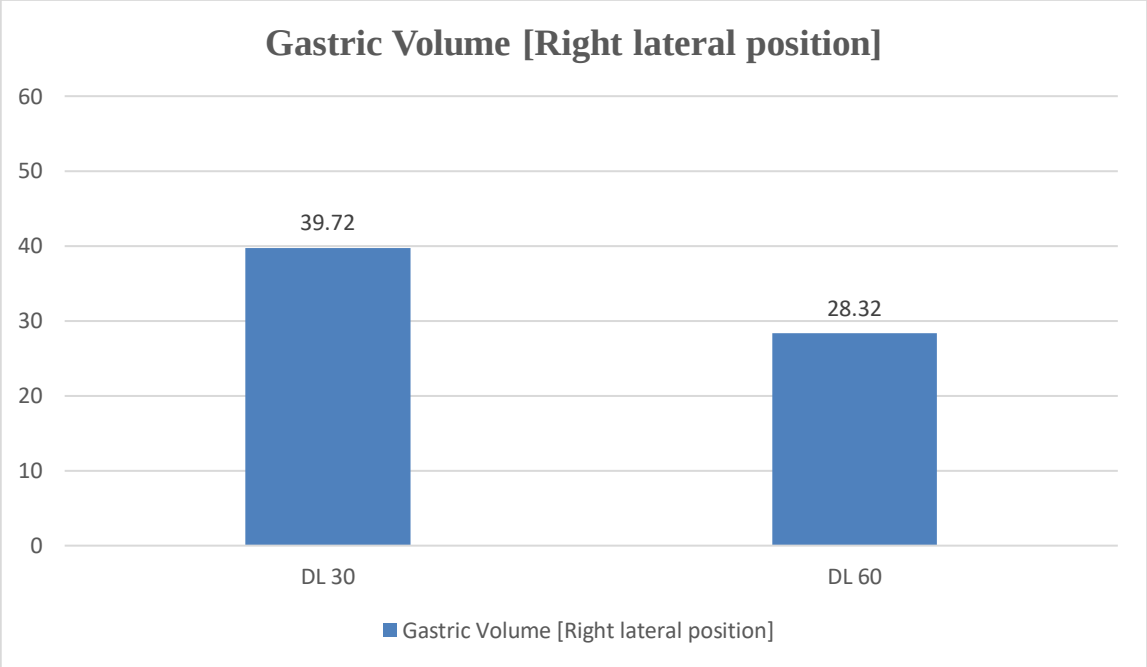


Figure 20: Comparison of the mean gastric volume between the groups in right lateral position

Table 8: Comparison of presence of regurgitation of content and aspiration risk between the groups

		DL 30	DL 60	Chi-square
		Count (N %)	Count (N %)	(p-value)
Regurgitation of contents	No	78 (84.8%)	89 (96.7%)	7.84 (0.01)
	Yes	14 (15.2%)	3 (3.3%)	
Aspiration risk	No	65 (70.7%)	85 (92.4%)	14.43 (0.01)*
	Yes	27 (29.3%)	7 (7.6%)	

The incidence of regurgitation and aspiration risk was significantly higher in the DL30 group compared to the DL60 group. In the DL30 group, 15.2% of patients experienced regurgitation, and 29.3% were at risk of aspiration, whereas in the DL60 group, these rates were notably lower at 3.3% and 7.6%, respectively ($p < 0.05$).

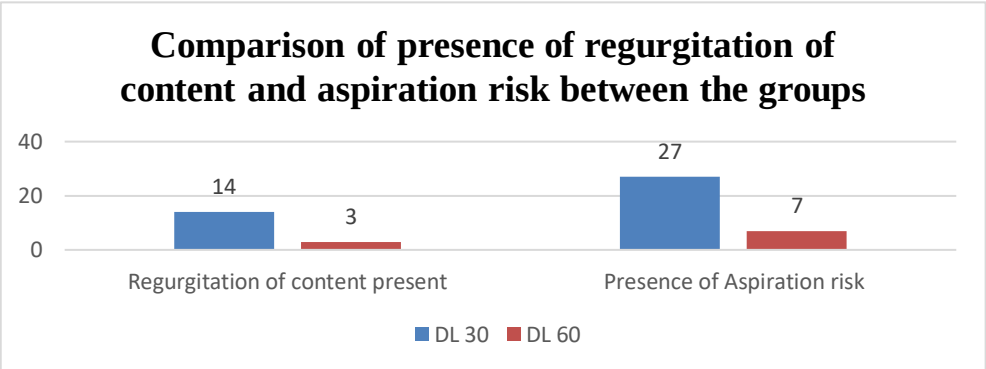


Figure 21: Comparison of presence of regurgitation of content and aspiration risk between the groups

DISCUSSION

Gastric emptying plays a crucial role in perioperative safety, particularly in diabetic patients who are prone to gastroparesis, a condition characterized by delayed gastric emptying without mechanical obstruction. This delayed gastric transit increases fasting gastric residual volume (GRV), which can significantly raise the risk of regurgitation and aspiration during anesthesia induction. Pulmonary aspiration of gastric contents remains a serious perioperative complication, contributing to aspiration pneumonitis and other respiratory complications. Therefore, strategies to minimize GRV in diabetic patients are essential for improving surgical outcomes.By evaluating GRV preoperatively, clinicians can identify patients who may require additional interventions to reduce gastric contents, thereby enhancing perioperative safety and improving overall surgical outcomes.

Proton pump inhibitors (PPIs) are commonly used for gastric acid suppression, but their role in reducing GRV in diabetic patients undergoing surgery remains underexplored. Dexlansoprazole, a novel dual-release PPI, has been shown to provide prolonged acid suppression and may have a potential effect on gastric volume reduction. However, the optimal dosing required to achieve a significant reduction in GRV without compromising safety is still debated.

This study was designed to evaluate the efficacy and safety of two different doses of Dexlansoprazole (30 mg and 60 mg) in reducing GRV, using ultrasonography as a non-invasive tool for accurate volume measurement. A total of 184 diabetic patients undergoing elective surgery were enrolled and randomized into two groups. The primary objective was to determine which dose was more effective in minimizing GRV and lowering the risk of regurgitation and aspiration.

By comparing GRV between the DL30 (30 mg) and DL60 (60 mg) groups, this study provides valuable insights into the clinical utility of Dexlansoprazole in preoperative gastric volume management. The findings could help guide anesthetic and surgical protocols to enhance patient safety and reduce perioperative complications in diabetic individuals.

Present study included total of 184 patients fulfilling inclusion criteria, who were divided into two groups as; Group DL30 – receiving 30mg of Dexlansoprazole and Group DL60 receiving 60mg of Dexlansoprazole. The mean ages between the groups were comparable, with no significant difference observed; the mean age in the DL30 group was 47.8 years, compared to 49.2 years in the DL60 group. The distribution according to gender, ASA grade and mean physical characteristics between the group were comparable with no significant difference noted.

Cunha DD et al., documented with mean age of the diabetic group was 49.3 ±16.4 years, while the non-diabetic group had a mean age of 49.4 ±16.8 years.[43]

On assessment of the measurements, there is no significant difference noted between the groups. The measurement of craniocaudal diameters, anteroposterior diameter and cross-sectional area were comparable between the groups. However, there is significant reduction of the grastric volume in the DL60 group (28.32±7.58) compared to DL30 group (39.72±8.54).($p<0.05$)

In study by Sharma G et al., they documented that Patients with diabetes and chronic kidney disease (CKD) showed a significant increase in cross-sectional area (CSA) in both supine and right lateral decubitus positions, while higher BMI was associated with increased estimated gastric capacity. The findings suggest that fasting beyond 6-10 hours does not guarantee an empty stomach, and individuals with comorbidities such as diabetes, obesity, and CKD are more likely to retain hazardous gastric contents.[37]In this study by Garg H et al., the "supine position, the craniocaudal (CC) and anteroposterior (AP) diameters were smaller in the control group compared to the diabetic group. In the right lateral decubitus (RLD) position, the CC and AP diameters were 2.28 ± 0.57 cm and 1.24 ± 0.42 cm in the control group, respectively, versus 2.54 ± 0.56 cm and 1.82 ± 0.56 cm in diabetics

group The cross-sectional area (CSA) was significantly larger in diabetics (2.57 ± 1.19 cm^2 supine, 3.73 ± 1.61 cm^2 RLD) compared to controls (1.41 ± 0.55 cm^2 supine, 2.30 ± 1.18 cm^2 RLD) (P = 0.001). Gastric volume (GV) was also higher in diabetics (9.15 ± 25.70 ml) compared to controls (4.20 ± 22.26 ml). These findings suggest that diabetic patients have a larger antral CSA and gastric volume than non-diabetic individuals, as measured by gastric ultrasonography."[2]

Theincidence of regurgitation and aspiration risk was significantly higher in the DL30 group compared to the DL60 group. In the DL30 group, 15.2% of patients experienced regurgitation, and 29.3% were at risk of aspiration, whereas in the DL60 group, these rates were notably lower at 3.3% and 7.6%, respectively ($p < 0.05$).

The dexlansoprazole demonstrated a sustained improvement in GERDQ scores during the on-demand period, whereas esomeprazole did not. These findings suggest that while both medications are effective, dexlansoprazole may offer more consistent symptom control over time.[38]Sabry R et al., found that diabetes group had a greater aspirated volume through the nasogastric tube, with a strong correlation between ultrasound-measured residual volume and aspirated gastric contents. Even after fasting for eight hours before elective surgery, patients with long-standing diabetes exhibited significantly higher residual stomach capacity than healthy controls.[1]

LIMITATIONS

The study has limitations, firstly being a single center study conducted among the smaller sample size which makes it difficult to generalise the results to wide population. Although the sample size was calculated statistically the strict inclusion and exclusion criteria may affect the external validity of study. The gastric volume was estimated based on Ultra Sound guided measurement, which despite being non-invasive and validated, are operator dependent and may be subjected to the interobserver variability. Eventually, while we used two dosing groups were compared, the absence of placebo or baseline control group may limit the interpretation of absolute drug effect and efficacy hence this study requires further research.

CONCLUSION

The present study demonstrates that a higher dose of Dexlansoprazole (60 mg) is more effective in reducing fasting gastric residual volume compared to the lower dose (30 mg) in diabetic patients undergoing elective surgery. While both groups had comparable demographic, physical, and glycemic characteristics, the DL60 group showed a significantly lower gastric volume compared to the DL30 group ($p<0.05$). Additionally, there was a significantly lower incidence of regurgitation and aspiration risk in the DL60 group ($p<0.05$). These findings suggest that 60 mg of Dexlansoprazole is a more effective and safer option for reducing gastric residual volume and minimizing aspiration risk in this patient population.

SUMMARY

This Double-blinded comparative study, titled "**Efficacy and safety of two doses of Dexlansoprazole in assessing fasting gastric residual volume by ultrasonography in diabetic patients undergoing elective surgery"** was carried out in the Department of Anaesthesiology,B.L.D.E.(DU)'Shri.B.M.Patil Medical College and Hospital Research Centre Vijayapura Karnataka

The present study included a total of 184 patients who met the inclusion criteria. These patients were divided into two groups: Group DL30, which received 30 mg of Dexlansoprazole, and Group DL60, which received 60 mg of Dexlansoprazole. The mean age of the participants in both groups was comparable, with no significant difference observed; the mean age in the DL30 group was 47.8 years, while in the DL60 group, it was 49.2 years. Similarly, gender distribution was balanced between the two groups, with no statistically significant differences noted.

Furthermore, the ASA (American Society of Anesthesiologists) grades were comparable between the groups, indicating that the overall health status and perioperative risk factors were similar. Additionally, the mean physical characteristics of the patients did not show any significant differences between the two groups. Glycemic parameters were also assessed, and no

significant differences were found, suggesting that blood glucose levels and diabetes-related metabolic factors were well-matched across both groups.

When evaluating gastric measurements, no significant differences were observed between the groups in terms of craniocaudal diameters, anteroposterior diameters, and cross-sectional areas. However, a notable reduction in gastric volume was observed in the DL60 group (28.32±7.58) compared to the DL30 group (39.72±8.54), with a statistically significant difference ($p<0.05$). This suggests that a higher dose of Dexlansoprazole may be more effective in reducing gastric volume.

Additionally, a significantly higher incidence of regurgitation and aspiration risk was observed in the DL30 group compared to the DL60 group. Specifically, 15.2% of patients in the DL30 group experienced regurgitation, and 29.3% were at risk of aspiration, compared to only 3.3% and 7.6% in the DL60 group, respectively ($p<0.05$). These findings highlight the potential benefits of a higher Dexlansoprazole dose in reducing perioperative complications related to gastric contents.

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ANNEXURE

MASTERCHART

Sr. No	Group	Age	Gender	ASA	Weight(KG	Height(CM)	BMI(Kg/M2)	History of diabetes	Fasting Duration	FBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	CC Diameter[supine position]	AP Diameter[supine position]	CSA(cm2)[supine position]	CC Diameter[in Right lateral position]	AP Diameter[Right lateral position]	CSA(cm2) [Right lateral position]	Gastric Volume[Right lateral position]	Grading of contents	Regurgitation of contents	spiration ris
1	DL 30	48	M	1	62	164	23.05	8	8	88	164	7	2.87	1.14	2.56	2.86	2.14	4.8	35.64	1	no	no
2	DL 30	49	M	2	64	165	23.5	10	8	102	186	7.5	2.94	1.18	2.72	3.02	2.13	5.05	38.01	1	yes	yes
3	DL 30	44	M	2	67	167	24.02	9	10	98	217	11	3.02	1.46	3.46	3.12	2.22	5.43	49.95	2	no	no
4	DL 30	45	M	2	68	164	25.28	8	11	112	196	13	3.08	2.11	5.1	3.14	2.34	5.77	53.64	2	no	yes
5	DL 30	41	F	3	69	171	23.59	7	10	106	184	12	3.12	2.06	5.04	3.15	2.12	5.24	51.02	2	no	no
6	DL 30	54	F	3	68	162	25.91	12	9	106	178	7.5	2.96	1.96	4.55	3.17	2.21	5.5	38.18	1	no	no
7	DL 30	39	F	2	71	163	26.72	7	10	103	208	11	2.84	1.88	4.19	3.01	2.01	4.75	46.43	2	no	no
8	DL 30	63	M	3	73	164	27.14	13	8	96	218	10	2.93	1.98	4.55	3.18	2.67	6.66	43.59	2	yes	yes
9	DL 30	58	M	2	72	168	25.51	10	8	115	176	6.5	2.78	1.84	4.01	2.95	2.11	4.88	24	0	no	no
10	DL 30	54	M	2	77	163	28.98	9	8	94	168	10	3.02	1.93	4.57	3.24	2.44	6.2	48.4	2	yes	yes
11	DL 30	48	M	3	73	161	28.16	10	10	108	195	12	3.12	2.12	5.19	3.32	2.47	6.44	59.58	2	no	no
12	DL 30	49	M	2	69	163	25.97	8	8	94	207	6.8	2.88	1.78	4.02	2.94	1.98	4.57	31	1	no	no
13	DL 30	48	M	2	64	163	24.08	8	8	112	187	6.8	2.94	2.12	4.89	2.98	2.01	4.7	34.18	1	no	yes
14	DL 30	39	F	3	68	165	24.97	7	9	114	223	12.5	2.84	1.98	4.41	2.92	2.24	5.13	51.97	2	no	no
15	DL 30	58	F	2	72	166	26.12	11	9	98	214	11.5	3.12	2.45	6.003	3.44	2.64	7.13	56.85	2	no	yes
16	DL 30	56	F	3	73	168	25.86	8	8	106	196	6.5	2.78	1.98	4.32	2.98	2.14	5	28.32	1	no	no
17	DL 30	48	F	3	66	171	22.57	9	8	104	179	7	2.65	2.14	4.45	2.87	2.01	4.53	31.69	0	no	yes
18	DL 30	56	M	3	68	159	26.89	10	8	92	184	6.3	2.77	2.16	4.69	2.88	2.11	4.77	24.96	0	yes	yes
19	DL 30	54	M	3	70	164	26.02	10	8	117	211	7	2.94	2.12	4.89	3.01	2.14	5.05	31.61	0	no	no

20	DL 30	59	M	2	71	164	26.39	12	8	106	188	7.5	3.01	2.12	5.01	3.12	2.44	5.97	38.64	0	no	no
21	DL 30	44	M	2	72	165	26.44	13	9	98	208	9	2.88	2.14	4.84	2.94	2.18	5.03	44.11	1	no	no
22	DL 30	39	M	3	69	167	24.74	9	8	114	214	8.5	2.67	2.17	4.55	2.81	2.11	4.65	44.97	1	no	no
23	DL 30	45	M	2	68	168	24.09	7	8	99	203	7.5	2.54	2.12	4.22	2.65	2.12	4.41	33.78	1	no	yes
24	DL 30	39	M	2	65	164	24.16	7	8	100	204	7.5	2.44	1.98	3.79	2.57	2.06	4.15	37.67	1	yes	yes
25	DL 30	46	M	2	67	163	25.21	8	8	113	211	7.5	3.01	1.94	4.58	3.11	2.01	4.9	39.66	1	no	no
26	DL 30	44	M	3	70	165	25.71	8	8	108	194	7	2.93	1.98	4.55	3.01	1.88	4.44	35.5	0	no	no
27	DL 30	49	M	2	64	165	23.5	10	8	94	196	7	2.87	2.11	4.75	2.98	2.12	4.96	36.69	0	no	no
28	DL 30	38	F	2	70	164	26.02	8	10	109	212	10	3.01	2.12	5.01	2.94	1.98	4.57	45.08	1	no	no
29	DL 30	62	F	2	58	158	23.23	12	10	121	216	10	2.98	2.12	4.96	3.23	2.54	6.44	41.66	2	no	no
30	DL 30	42	F	2	55	157	22.31	8	8	104	215	8.5	2.86	1.98	4.44	2.98	2.01	4.7	41.86	1	no	no
31	DL 30	39	F	2	59	164	21.93	7	11	99	221	11	2.76	2.11	4.57	2.86	2.16	4.85	47.89	1	no	yes
32	DL 30	42	F	2	60	167	21.51	8	9	121	208	7.5	2.64	1.94	4.02	2.84	1.99	4.43	37.91	0	no	no
33	DL 30	46	M	3	68	168	24.09	8	8	123	244	6.5	2.66	1.83	3.82	2.76	1.88	4.07	27.54	0	no	yes
34	DL 30	57	M	3	67	166	24.31	12	8	106	168	8.5	2.96	2.21	5.13	3.06	2.34	5.62	36.09	0	no	no
35	DL 30	47	M	3	65	165	23.87	9	8	111	221	9	2.89	2.16	4.9	2.98	2.21	5.17	42.32	2	yes	yes
36	DL 30	43	M	3	67	158	26.83	8	8	109	217	8.5	2.73	1.86	3.98	2.84	1.94	4.32	35.03	1	no	no
37	DL 30	39	M	2	68	159	26.89	7	9	97	174	10	2.88	1.93	4.36	2.92	1.92	4.4	41.32	1	yes	yes
38	DL 30	41	M	2	69	163	25.97	8	8	115	192	8.5	2.73	1.84	3.94	2.88	1.92	4.34	37.88	1	no	no
39	DL 30	39	M	2	70	166	25.4	7	8	125	217	9	2.82	1.82	4.03	2.93	1.92	4.41	41.46	2	yes	yes
40	DL 30	48	M	2	72	164	26.76	10	10	111	203	12	3.12	2.21	5.41	3.33	2.38	6.22	56.37	2	no	no
41	DL 30	54	M	2	69	168	24.44	12	8	103	216	8.5	3.01	2.11	4.98	3.14	2.26	5.57	39.2	1	no	no
42	DL 30	57	M	2	71	167	25.45	12	8	98	188	8.5	3.21	2.01	5.06	3.26	2.14	5.47	33.9	1	no	yes
43	DL 30	47	M	2	77	164	28.62	9	8	99	168	6.5	2.65	1.88	3.91	2.76	1.99	4.31	29.76	0	no	no
44	DL 30	48	F	2	72	170	24.91	8	8	105	144	6.2	2.74	1.92	4.13	2.88	2.01	4.54	31.84	0	no	no

45	DL 30	51	F	2	68	172	22.98	10	8	112	156	6	2.66	1.98	4.13	2.84	2.01	4.48	27.12	0	no	no
46	DL 30	44	M	2	69	164	25.65	9	9	115	167	7.5	2.72	2.11	4.5	2.88	2.11	4.77	40.32	2	yes	yes
47	DL 30	48	M	2	67	163	25.27	9	9	121	159	7.5	2.65	1.98	4.12	3.01	2.12	5.01	38.7	1	no	yes
48	DL 30	56	M	2	64	168	22.67	12	8	127	183	7	3.12	1.98	4.85	3.32	2.06	5.37	33.72	1	no	no
49	DL 30	52	M	3	70	162	26.67	11	8	124	188	6.5	2.76	1.92	4.16	2.93	2.01	4.62	27.89	1	no	no
50	DL 30	51	M	3	71	169	24.85	13	8	132	204	7	2.87	2.03	4.57	2.97	2.11	4.92	33.55	1	no	yes
51	DL 30	51	M	3	69	159	27.29	12	8	112	184	6.5	2.79	1.97	4.31	2.89	2.01	4.56	28.29	1	no	no
52	DL 30	44	F	2	68	163	25.59	10	10	104	213	10	2.95	1.94	4.49	3.03	2.15	5.11	45.28	2	no	yes
53	DL 30	48	M	1	62	164	23.05	8	8	88	164	7	2.87	1.14	2.56	2.86	2.14	4.8	35.64	1	no	no
54	DL 30	49	M	2	64	165	23.5	10	8	102	186	7.5	2.94	1.18	2.72	3.02	2.13	5.05	38.01	1	no	no
55	DL 30	44	M	2	67	167	24.02	9	10	98	217	11	3.02	1.46	3.46	3.12	2.22	5.43	49.95	2	no	no
56	DL 30	45	M	2	68	164	25.28	8	11	112	196	13	3.08	2.11	5.1	3.14	2.34	5.77	53.64	2	no	no
57	DL 30	41	F	3	69	171	23.59	7	10	106	184	12	3.12	2.06	5.04	3.15	2.12	5.24	51.02	2	no	no
58	DL 30	54	F	3	68	162	25.91	12	9	106	178	7.5	2.96	1.96	4.55	3.17	2.21	5.5	38.18	1	yes	yes
59	DL 30	39	F	2	71	163	26.72	7	10	103	208	11	2.84	1.88	4.19	3.01	2.01	4.75	46.43	2	no	no
60	DL 30	63	M	3	73	164	27.14	13	8	96	218	10	2.93	1.98	4.55	3.18	2.67	6.66	43.59	2	yes	yes
61	DL 30	58	M	2	72	168	25.51	10	8	115	176	6.5	2.78	1.84	4.01	2.95	2.11	4.88	24	0	no	no
62	DL 30	54	M	2	77	163	28.98	9	8	94	168	10	3.02	1.93	4.57	3.24	2.44	6.2	48.4	2	no	yes
63	DL 30	48	M	3	73	161	28.16	10	10	108	195	12	3.12	2.12	5.19	3.32	2.47	6.44	59.58	2	no	no
64	DL 30	49	M	2	69	163	25.97	8	8	94	207	6.8	2.88	1.78	4.02	2.94	1.98	4.57	31	1	no	no
65	DL 30	48	M	2	64	163	24.08	8	8	112	187	6.8	2.94	2.12	4.89	2.98	2.01	4.7	34.18	1	no	no
66	DL 30	39	F	3	68	165	24.97	7	9	114	223	12.5	2.84	1.98	4.41	2.92	2.24	5.13	51.97	2	no	no
67	DL 30	58	F	2	72	166	26.12	11	9	98	214	11.5	3.12	2.45	6.003	3.44	2.64	7.13	56.85	2	yes	no
68	DL 30	56	F	3	73	168	25.86	8	8	106	196	6.5	2.78	1.98	4.32	2.98	2.14	5	28.32	1	no	no
69	DL 30	48	F	3	66	171	22.57	9	8	104	179	7	2.65	2.14	4.45	2.87	2.01	4.53	31.69	0	no	yes
70	DL 30	56	M	3	68	159	26.89	10	8	92	184	6.3	2.77	2.16	4.69	2.88	2.11	4.77	24.96	0	yes	yes
71	DL 30	54	M	3	70	164	26.02	10	8	117	211	7	2.94	2.12	4.89	3.01	2.14	5.05	31.61	0	no	no

72	DL 30	59	M	2	71	164	26.39	12	8	106	188	7.5	3.01	2.12	5.01	3.12	2.44	5.97	38.64	0	no	no
73	DL 30	44	M	2	72	165	26.44	13	9	98	208	9	2.88	2.14	4.84	2.94	2.18	5.03	44.11	1	no	no
74	DL 30	39	M	3	69	167	24.74	9	8	114	214	8.5	2.67	2.17	4.55	2.81	2.11	4.65	44.97	1	no	no
75	DL 30	45	M	2	68	168	24.09	7	8	99	203	7.5	2.54	2.12	4.22	2.65	2.12	4.41	33.78	1	no	no
76	DL 30	39	M	2	65	164	24.16	7	8	100	204	7.5	2.44	1.98	3.79	2.57	2.06	4.15	37.67	1	no	no
77	DL 30	46	M	2	67	163	25.21	8	8	113	211	7.5	3.01	1.94	4.58	3.11	2.01	4.9	39.66	1	no	no
78	DL 30	44	M	3	70	165	25.71	8	8	108	194	7	2.93	1.98	4.55	3.01	1.88	4.44	35.5	0	no	no
79	DL 30	49	M	2	64	165	23.5	10	8	94	196	7	2.87	2.11	4.75	2.98	2.12	4.96	36.69	0	no	no
80	DL 30	38	F	2	70	164	26.02	8	10	109	212	10	3.01	2.12	5.01	2.94	1.98	4.57	45.08	1	no	no
81	DL 30	62	F	2	58	158	23.23	12	10	121	216	10	2.98	2.12	4.96	3.23	2.54	6.44	41.66	2	no	no
82	DL 30	42	F	2	55	157	22.31	8	8	104	215	8.5	2.86	1.98	4.44	2.98	2.01	4.7	41.86	1	no	no
83	DL 30	39	F	2	59	164	21.93	7	11	99	221	11	2.76	2.11	4.57	2.86	2.16	4.85	47.89	1	yes	yes
84	DL 30	42	F	2	60	167	21.51	8	9	121	208	7.5	2.64	1.94	4.02	2.84	1.99	4.43	37.91	0	no	no
85	DL 30	46	M	3	68	168	24.09	8	8	123	244	6.5	2.66	1.83	3.82	2.76	1.88	4.07	27.54	0	no	no
86	DL 30	57	M	3	67	166	24.31	12	8	106	168	8.5	2.96	2.21	5.13	3.06	2.34	5.62	36.09	0	no	no
87	DL 30	47	M	3	65	165	23.87	9	8	111	221	9	2.89	2.16	4.9	2.98	2.21	5.17	42.32	2	no	no
88	DL 30	43	M	3	67	158	26.83	8	8	109	217	8.5	2.73	1.86	3.98	2.84	1.94	4.32	35.03	1	no	no
89	DL 30	39	M	2	68	159	26.89	7	9	97	174	10	2.88	1.93	4.36	2.92	1.92	4.4	41.32	1	no	no
90	DL 30	41	M	2	69	163	25.97	8	8	115	192	8.5	2.73	1.84	3.94	2.88	1.92	4.34	37.88	1	no	yes
91	DL 30	39	M	2	70	166	25.4	7	8	125	217	9	2.82	1.82	4.03	2.93	1.92	4.41	41.46	2	no	no
92	DL 30	48	M	2	72	164	26.76	10	10	111	203	12	3.12	2.21	5.41	3.33	2.38	6.22	56.37	2	no	no
93	DL 60	58	M	1	62	164	23.05	8	8	88	164	7	2.87	1.14	2.56	2.86	2.14	4.8	35.64	1	no	no
94	DL 60	56	M	2	64	165	23.5	10	8	102	186	7.5	2.94	1.18	2.72	3.02	2.13	5.05	38.01	1	no	no
95	DL 60	48	M	2	67	167	24.02	9	10	98	217	11	3.02	1.46	3.46	3.12	2.22	5.43	49.95	2	no	no
96	DL 60	56	M	2	68	164	25.28	8	11	112	196	13	3.08	2.11	5.1	3.14	2.34	5.77	53.64	2	no	no

97	DL 60	41	F	3	69	171	23.59	7	10	106	184	12	3.12	2.06	5.04	3.15	2.12	5.24	51.02	2	no	no
98	DL 60	54	F	3	68	162	25.91	12	9	106	178	7.5	2.96	1.96	4.55	3.17	2.21	5.5	38.18	1	no	no
99	DL 60	39	F	2	71	163	26.72	7	10	103	208	11	2.84	1.88	4.19	3.01	2.01	4.75	46.43	2	no	no
100	DL 60	63	M	3	73	164	27.14	13	8	96	218	10	2.93	1.98	4.55	3.18	2.67	6.66	43.59	2	no	no
101	DL 60	58	M	2	72	168	25.51	10	8	115	176	6.5	2.78	1.84	4.01	2.95	2.11	4.88	24	0	no	yes
102	DL 60	54	M	2	77	163	28.98	9	8	94	168	10	3.02	1.93	4.57	3.24	2.44	6.2	48.4	2	no	no
103	DL 60	48	M	3	73	161	28.16	10	10	108	195	12	3.12	2.12	5.19	3.32	2.47	6.44	59.58	2	no	no
104	DL 60	49	M	2	69	163	25.97	8	8	94	207	6.8	2.88	1.78	4.02	2.94	1.98	4.57	31	1	yes	no
105	DL 60	48	M	2	64	163	24.08	8	8	112	187	6.8	2.94	2.12	4.89	2.98	2.01	4.7	34.18	1	no	no
106	DL 60	39	F	3	68	165	24.97	7	9	114	223	12.5	2.84	1.98	4.41	2.92	2.24	5.13	51.97	2	no	no
107	DL 60	58	F	2	72	166	26.12	11	9	98	214	11.5	3.12	2.45	6.003	3.44	2.64	7.13	56.85	2	no	no
108	DL 60	56	F	3	73	168	25.86	8	8	106	196	6.5	2.78	1.98	4.32	2.98	2.14	5	28.32	1	no	no
109	DL 60	48	F	3	66	171	22.57	9	8	104	179	7	2.65	2.14	4.45	2.87	2.01	4.53	31.69	0	no	no
110	DL 60	56	M	3	68	159	26.89	10	8	92	184	6.3	2.77	2.16	4.69	2.88	2.11	4.77	24.96	0	no	no
111	DL 60	54	M	3	70	164	26.02	10	8	117	211	7	2.94	2.12	4.89	3.01	2.14	5.05	31.61	0	no	no
112	DL 60	59	M	2	71	164	26.39	12	8	106	188	7.5	3.01	2.12	5.01	3.12	2.44	5.97	38.64	0	no	yes
113	DL 60	44	M	2	72	165	26.44	13	9	98	208	9	2.88	2.14	4.84	2.94	2.18	5.03	44.11	1	no	no
114	DL 60	39	M	3	69	167	24.74	9	8	114	214	8.5	2.67	2.17	4.55	2.81	2.11	4.65	44.97	1	no	no
115	DL 60	45	M	2	68	168	24.09	7	8	99	203	7.5	2.54	2.12	4.22	2.65	2.12	4.41	33.78	1	no	no
116	DL 60	39	M	2	65	164	24.16	7	8	100	204	7.5	2.44	1.98	3.79	2.57	2.06	4.15	37.67	1	no	no
117	DL 60	46	M	2	67	163	25.21	8	8	113	211	7.5	3.01	1.94	4.58	3.11	2.01	4.9	39.66	1	no	no
118	DL 60	44	M	3	70	165	25.71	8	8	108	194	7	2.93	1.98	4.55	3.01	1.88	4.44	35.5	0	no	no
119	DL 60	49	M	2	64	165	23.5	10	8	94	196	7	2.87	2.11	4.75	2.98	2.12	4.96	36.69	0	no	no
120	DL 60	38	F	2	70	164	26.02	8	10	109	212	10	3.01	2.12	5.01	2.94	1.98	4.57	45.08	1	no	no
121	DL 60	62	F	2	58	158	23.23	12	10	121	216	10	2.98	2.12	4.96	3.23	2.54	6.44	41.66	2	no	no
122	DL 60	42	F	2	55	157	22.31	8	8	104	215	8.5	2.86	1.98	4.44	2.98	2.01	4.7	41.86	1	no	no

123	DL 60	39	F	2	59	164	21.93	7	11	99	221	11	2.76	2.11	4.57	2.86	2.16	4.85	47.89	1	no	no
124	DL 60	42	F	2	60	167	21.51	8	9	121	208	7.5	2.64	1.94	4.02	2.84	1.99	4.43	37.91	0	no	no
125	DL 60	46	M	3	68	168	24.09	8	8	123	244	6.5	2.66	1.83	3.82	2.76	1.88	4.07	27.54	0	no	no
126	DL 60	57	M	3	67	166	24.31	12	8	106	168	8.5	2.96	2.21	5.13	3.06	2.34	5.62	36.09	0	no	no
127	DL 60	47	M	3	65	165	23.87	9	8	111	221	9	2.89	2.16	4.9	2.98	2.21	5.17	42.32	2	no	no
128	DL 60	43	M	3	67	158	26.83	8	8	109	217	8.5	2.73	1.86	3.98	2.84	1.94	4.32	35.03	1	no	no
129	DL 60	39	M	2	68	159	26.89	7	9	97	174	10	2.88	1.93	4.36	2.92	1.92	4.4	41.32	1	no	no
130	DL 60	41	M	2	69	163	25.97	8	8	115	192	8.5	2.73	1.84	3.94	2.88	1.92	4.34	37.88	1	no	no
131	DL 60	39	M	2	70	166	25.4	7	8	125	217	9	2.82	1.82	4.03	2.93	1.92	4.41	41.46	2	no	no
132	DL 60	48	M	2	72	164	26.76	10	10	111	203	12	3.12	2.21	5.41	3.33	2.38	6.22	56.37	2	yes	yes
133	DL 60	54	M	2	69	168	24.44	12	8	103	216	8.5	3.01	2.11	4.98	3.14	2.26	5.57	39.2	1	no	no
134	DL 60	57	M	2	71	167	25.45	12	8	98	188	8.5	3.21	2.01	5.06	3.26	2.14	5.47	33.9	1	no	no
135	DL 60	47	M	2	77	164	28.62	9	8	99	168	6.5	2.65	1.88	3.91	2.76	1.99	4.31	29.76	0	no	no
136	DL 60	48	F	2	72	170	24.91	8	8	105	144	6.2	2.74	1.92	4.13	2.88	2.01	4.54	31.84	0	no	no
137	DL 60	51	F	2	68	172	22.98	10	8	112	156	6	2.66	1.98	4.13	2.84	2.01	4.48	27.12	0	no	no
138	DL 60	44	M	2	69	164	25.65	9	9	115	167	7.5	2.72	2.11	4.5	2.88	2.11	4.77	40.32	2	no	no
139	DL 60	48	M	2	67	163	25.27	9	9	121	159	7.5	2.65	1.98	4.12	3.01	2.12	5.01	38.7	1	no	no
140	DL 60	56	M	2	64	168	22.67	12	8	127	183	7	3.12	1.98	4.85	3.32	2.06	5.37	33.72	1	no	no
141	DL 60	52	M	3	70	162	26.67	11	8	124	188	6.5	2.76	1.92	4.16	2.93	2.01	4.62	27.89	1	no	no
142	DL 60	51	M	3	71	169	24.85	13	8	132	204	7	2.87	2.03	4.57	2.97	2.11	4.92	33.55	1	no	no
143	DL 60	51	M	3	69	159	27.29	12	8	112	184	6.5	2.79	1.97	4.31	2.89	2.01	4.56	28.29	1	no	no
144	DL 60	44	F	2	68	163	25.59	10	10	104	213	10	2.95	1.94	4.49	3.03	2.15	5.11	45.28	2	no	no
145	DL 60	48	M	1	62	164	23.05	8	8	88	164	7	2.87	1.14	2.56	2.86	2.14	4.8	35.64	1	no	no
146	DL 60	49	M	2	64	165	23.5	10	8	102	186	7.5	2.94	1.18	2.72	3.02	2.13	5.05	38.01	1	no	yes
147	DL 60	44	M	2	67	167	24.02	9	10	98	217	11	3.02	1.46	3.46	3.12	2.22	5.43	49.95	2	yes	no

148	DL 60	45	M	2	68	164	25.28	8	11	112	196	13	3.08	2.11	5.1	3.14	2.34	5.77	53.64	2	no	no
149	DL 60	41	F	3	69	171	23.59	7	10	106	184	12	3.12	2.06	5.04	3.15	2.12	5.24	51.02	2	no	no
150	DL 60	54	F	3	68	162	25.91	12	9	106	178	7.5	2.96	1.96	4.55	3.17	2.21	5.5	38.18	1	no	no
151	DL 60	39	F	2	71	163	26.72	7	10	103	208	11	2.84	1.88	4.19	3.01	2.01	4.75	46.43	2	no	no
152	DL 60	63	M	3	73	164	27.14	13	8	96	218	10	2.93	1.98	4.55	3.18	2.67	6.66	43.59	2	no	no
153	DL 60	58	M	2	72	168	25.51	10	8	115	176	6.5	2.78	1.84	4.01	2.95	2.11	4.88	24	0	no	no
154	DL 60	54	M	2	77	163	28.98	9	8	94	168	10	3.02	1.93	4.57	3.24	2.44	6.2	48.4	2	no	no
155	DL 60	48	M	3	73	161	28.16	10	10	108	195	12	3.12	2.12	5.19	3.32	2.47	6.44	59.58	2	no	no
156	DL 60	49	M	2	69	163	25.97	8	8	94	207	6.8	2.88	1.78	4.02	2.94	1.98	4.57	31	1	no	yes
157	DL 60	48	M	2	64	163	24.08	8	8	112	187	6.8	2.94	2.12	4.89	2.98	2.01	4.7	34.18	1	no	no
158	DL 60	39	F	3	68	165	24.97	7	9	114	223	12.5	2.84	1.98	4.41	2.92	2.24	5.13	51.97	2	no	no
159	DL 60	58	F	2	72	166	26.12	11	9	98	214	11.5	3.12	2.45	6.003	3.44	2.64	7.13	56.85	2	no	no
160	DL 60	56	F	3	73	168	25.86	8	8	106	196	6.5	2.78	1.98	4.32	2.98	2.14	5	28.32	1	no	no
161	DL 60	48	F	3	66	171	22.57	9	8	104	179	7	2.65	2.14	4.45	2.87	2.01	4.53	31.69	0	no	no
162	DL 60	56	M	3	68	159	26.89	10	8	92	184	6.3	2.77	2.16	4.69	2.88	2.11	4.77	24.96	0	no	no
163	DL 60	54	M	3	70	164	26.02	10	8	117	211	7	2.94	2.12	4.89	3.01	2.14	5.05	31.61	0	no	no
164	DL 60	59	M	2	71	164	26.39	12	8	106	188	7.5	3.01	2.12	5.01	3.12	2.44	5.97	38.64	0	no	no
165	DL 60	44	M	2	72	165	26.44	13	9	98	208	9	2.88	2.14	4.84	2.94	2.18	5.03	44.11	1	no	no
166	DL 60	39	M	3	69	167	24.74	9	8	114	214	8.5	2.67	2.17	4.55	2.81	2.11	4.65	44.97	1	no	yes
167	DL 60	45	M	2	68	168	24.09	7	8	99	203	7.5	2.54	2.12	4.22	2.65	2.12	4.41	33.78	1	no	no
168	DL 60	39	M	2	65	164	24.16	7	8	100	204	7.5	2.44	1.98	3.79	2.57	2.06	4.15	37.67	1	no	no
169	DL 60	46	M	2	67	163	25.21	8	8	113	211	7.5	3.01	1.94	4.58	3.11	2.01	4.9	39.66	1	no	no
170	DL 60	44	M	3	70	165	25.71	8	8	108	194	7	2.93	1.98	4.55	3.01	1.88	4.44	35.5	0	no	no
171	DL 60	49	M	2	64	165	23.5	10	8	94	196	7	2.87	2.11	4.75	2.98	2.12	4.96	36.69	0	no	no
172	DL 60	38	F	2	70	164	26.02	8	10	109	212	10	3.01	2.12	5.01	2.94	1.98	4.57	45.08	1	no	no
173	DL 60	62	F	2	58	158	23.23	12	10	121	216	10	2.98	2.12	4.96	3.23	2.54	6.44	41.66	2	no	no
174	DL 60	42	F	2	55	157	22.31	8	8	104	215	8.5	2.86	1.98	4.44	2.98	2.01	4.7	41.86	1	no	no

175	DL 60	39	F	2	59	164	21.93	7	11	99	221	11	2.76	2.11	4.57	2.86	2.16	4.85	47.89	1	no	no
176	DL 60	42	F	2	60	167	21.51	8	9	121	208	7.5	2.64	1.94	4.02	2.84	1.99	4.43	37.91	0	no	no
177	DL 60	46	M	3	68	168	24.09	8	8	123	244	6.5	2.66	1.83	3.82	2.76	1.88	4.07	27.54	0	no	no
178	DL 60	57	M	3	67	166	24.31	12	8	106	168	8.5	2.96	2.21	5.13	3.06	2.34	5.62	36.09	0	no	no
179	DL 60	47	M	3	65	165	23.87	9	8	111	221	9	2.89	2.16	4.9	2.98	2.21	5.17	42.32	2	no	no
180	DL 60	43	M	3	67	158	26.83	8	8	109	217	8.5	2.73	1.86	3.98	2.84	1.94	4.32	35.03	1	no	no
181	DL 60	39	M	2	68	159	26.89	7	9	97	174	10	2.88	1.93	4.36	2.92	1.92	4.4	41.32	1	no	no
182	DL 60	41	M	2	69	163	25.97	8	8	115	192	8.5	2.73	1.84	3.94	2.88	1.92	4.34	37.88	1	no	no
183	DL 60	39	M	2	70	166	25.4	7	8	125	217	9	2.82	1.82	4.03	2.93	1.92	4.41	41.46	2	no	yes
184	DL 60	48	M	2	72	164	26.76	10	10	111	203	12	3.12	2.21	5.41	3.33	2.38	6.22	56.37	2	no	no