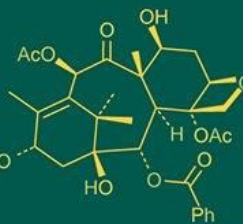
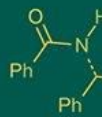


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Dr. Supreetkumar Sagar
MVSc (Veterinary Surgeon),
Department of Veterinary
Surgery and Radiology,
MAFSU, Nagpur, Karnataka,
India

Dr. SS Pitlawar
Head of the Department,
Department of Veterinary
Surgery and Radiology,
MAFSU, Nagpur,
Maharashtra, India

Corresponding Author:
Dr. Supreetkumar Sagar
MVSc (Veterinary Surgeon),
Department of Veterinary
Surgery and Radiology,
MAFSU, Nagpur, Karnataka,
India

Gastric dilatation-volvulus (GDV) in dogs: Etiology, pathophysiology, clinical presentation, diagnosis, treatment, and preventive strategies

Supreetkumar Sagar and SS Pitlawar

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Abstract

Gastric dilatation-volvulus (GDV) is a rapidly progressive and life-threatening emergency in dogs, characterized by acute gastric distension followed by rotation of the stomach along its mesenteric axis. This condition results in mechanical obstruction, vascular compromise, and severe systemic shock, leading to high mortality if not promptly treated. GDV occurs predominantly in large and giant breed dogs and is considered a multifactorial disorder influenced by genetic predisposition, thoracic conformation, gastric motility abnormalities, dietary practices, and environmental stressors. This chapter provides a comprehensive overview of GDV, encompassing its etiology, anatomy and pathophysiology, epidemiology, clinical presentation, diagnostic approach, emergency stabilization, surgical management, postoperative care, prognosis, and prevention strategies. Emphasis is placed on early recognition, rapid stabilization, and timely surgical intervention as key determinants of survival. Advances in perioperative care, diagnostic imaging, and prophylactic gastropexy techniques have significantly improved outcomes. Understanding the complex pathophysiological cascade and risk factors associated with GDV is essential for effective clinical decision-making and long-term prevention in high-risk canine populations.

Keywords: Gastric dilatation-volvulus, canine emergency, gastric torsion, large breed dogs, shock, gastropexy

1. Introduction

Gastric Dilatation-Volvulus (GDV) is one of the most critical and rapidly life-threatening emergencies in canine medicine. Characterized by acute distension of the stomach with gas, fluid, or feed material, followed by rotation of the organ around its mesenteric axis, GDV causes severe mechanical and functional obstruction of gastric outflow and inflow. With progressive distension, venous return to the heart is compromised, leading to obstructive shock and systemic collapse. Without immediate medical and surgical intervention, fatality is nearly certain. Even with treatment, mortality rates remain significant and depend heavily on the speed of diagnosis, degree of gastric necrosis at surgical exploration, and perioperative management.

GDV is particularly important in veterinary practice because of its prevalence in large and giant breed dogs. The condition has been documented for over a century, yet despite significant advances in veterinary anesthesia, intensive care, and surgical techniques, the pathogenesis of GDV remains incompletely understood. Numerous studies have attempted to characterize the interplay between conformation, genetics, gastrointestinal motility, nutritional patterns, and environmental factors, but no single cause has been definitively identified. The multifactorial nature of GDV complicates prevention and underscores the need for heightened clinical awareness.

This chapter provides a comprehensive overview designed for veterinary students, clinicians, and researchers. It synthesizes current knowledge on epidemiology, anatomy, pathophysiology, diagnostic strategies, treatment protocols, surgical considerations, postoperative management, and long-term prevention. Emphasis is placed on clinical decision-making and the physiologic mechanisms underlying the rapid deterioration seen in

dogs with GDV. Emerging research areas including genomics, gastric motility studies, and minimally invasive prophylactic procedures are also discussed.

2. Anatomy and Physiologic Considerations

Understanding GDV requires a detailed examination of the normal anatomical and physiological properties of the canine stomach, as well as the surrounding structures that influence gastric stability and motility. Although any breed can develop GDV, certain conformational features predispose specific populations to the condition.

2.1 Anatomical Structure of the Canine Stomach

The canine stomach is anatomically divided into the cardia, fundus, body, antrum, and pylorus. It is located primarily in the cranial abdomen, extending from the left cranial quadrant across the midline. Several anatomical features are relevant to GDV.

2.1.1 Ligamentous Support

The stomach is supported by the hepatogastric ligament, hepatoduodenal ligament, gastrosplenic ligament, and phrenicoesophageal ligament. These structures anchor the stomach but also allow substantial mobility, which becomes a key factor in volvulus. Dogs with increased ligamentous laxity may be more susceptible to rotation of the stomach.

2.1.2 Relationship with the Spleen

The spleen is attached to the greater curvature of the stomach via the gastrosplenic ligament. During gastric rotation, the spleen may be displaced, undergo torsion, or develop congestion. Splenic involvement can exacerbate cardiovascular compromise and contribute to postoperative complications.

2.1.3 Vascular Supply

Blood supply to the stomach is provided by multiple branches of the celiac artery, including the left gastric, right gastric, and gastropiploic arteries. During volvulus, these vessels are compressed, resulting in ischemia of the gastric wall. The degree of ischemia is strongly correlated with prognosis.

2.2 Physiological Function Relevant to GDV

2.2.1 Gastric Motility and Emptying

Normal gastric motility depends on coordinated contractions of smooth muscle layers, controlled by:

- Enteric nervous system
- Autonomic nervous system
- Gastrointestinal hormones
- Interstitial cells of Cajal

Disruptions in motility whether congenital, acquired, or secondary to stress may lead to gas retention and delayed gastric emptying. Several studies suggest that reduced gastric motility may precede GDV in predisposed dogs, although evidence remains inconclusive.

2.2.2 Gas Accumulation and Aerophagia

The stomach normally accumulates small amounts of gas during eating, but this is relieved by belching. Aerophagia excessive air swallowing can occur in dogs that gulp food, eat rapidly, or experience anxiety. In susceptible animals,

this may contribute to gastric dilatation, particularly after large meals.

2.2.3 Gastric Outflow Obstruction

Even partial obstruction of the pylorus or duodenum can increase gastric retention and distension. Structural disorders, foreign bodies, or functional gastroparesis may contribute, although their direct role in GDV remains debated.

2.3 Conformational and Genetic Predispositions

GDV occurs disproportionately in large and giant breeds, suggesting that thoracic conformation and genetic factors play substantial roles.

2.3.1 Thoracic Conformation

Deep-chested dogs those with a high thoracic height-to-width ratio demonstrate a significantly elevated risk of GDV. This shape may permit increased gastric mobility and displacement.

2.3.2 Breed Predisposition

Breeds with highest reported incidence include:

- Great Danes
- German Shepherd Dogs
- Standard Poodles
- Weimaraners
- Irish Setters
- Doberman Pinschers

In some high-risk breeds (e.g., Great Danes), lifetime prevalence may exceed 20%.

2.3.3 Heritability Factors

Evidence supports a strong genetic component:

- Familial clustering is documented in several breeds.
- First-degree relatives of affected dogs show increased risk.
- Ongoing genomics research aims to identify specific genetic markers.

Despite strong evidence of heritability, no single causative mutation has been identified to date.

2.4 Summary of Anatomical and Physiologic Considerations

The combination of stomach mobility, deep-chested conformation, ligamentous laxity, genetic inheritance, and potential motility abnormalities creates an environment in which GDV can occur. While no single anatomical defect directly causes the condition, the interplay of these predispositions amplifies susceptibility to gastric torsion during episodes of acute dilatation.

3. Epidemiology of GDV

GDV is recognized internationally as one of the most common acute surgical emergencies in dogs, with incidence varying based on population demographics, breed representation, and study methodology. Epidemiological investigations over the past four decades have consistently highlighted the disproportionate risk among large and giant

breed dogs; however, GDV can occur in any breed, age group, or sex.

3.1 Incidence and Prevalence

Reported incidence rates range from 0.1% to 1.7% of the general canine population annually. In high-risk breeds, incidence may be significantly higher. Great Danes, for instance, have been repeatedly documented to possess a lifetime risk approaching 20%-45%, making GDV one of the most clinically significant conditions affecting the breed. The condition appears more frequently in middle-aged and older dogs, with the peak occurrence between 7 and 10 years of age. However, young dogs especially those from genetically predisposed lines can also develop GDV. The condition remains rare in toy and small breeds, though case reports exist.

3.2 Sex and Reproductive Status

Sex-related predisposition remains debated. Some studies suggest that males particularly neutered males are at greater risk, whereas others indicate no significant difference. Spayed females may demonstrate slightly higher risk compared with intact females, though hormonal influences are not fully understood. Sex is likely a minor risk factor compared with conformation, genetics, and environmental influences.

3.3 Geographic and Environmental Variation

Incidence varies internationally and regionally. Factors contributing to these differences include:

- Breed popularity within regions
- Feeding practices and diet availability
- Veterinary expertise and emergency service prevalence
- Cultural differences in exercise and housing of dogs

Some epidemiological studies suggest that urban dogs may experience higher GDV rates due to increased stress, though evidence remains inconsistent.

4. Etiology and Multifactorial Risk Analysis

Despite extensive research, no single causative mechanism for GDV has been definitively established. Instead, GDV is widely regarded as a multifactorial syndrome, arising from interactions among genetic predisposition, anatomical structure, physiological dynamics, dietary influences, and environmental stressors.

4.1 Genetic and Heritable Factors

Heritability estimations in high-risk breeds suggest that genetics play a substantial role. Studies examining pedigrees of Great Danes, Standard Poodles, and German Shepherd Dogs indicate that first-degree relatives of affected animals have a markedly increased likelihood of developing GDV.

Potential heritable traits contributing to risk include:

- Thoracic and abdominal conformation
- Gastric ligament laxity
- Temperament (e.g., anxiety-prone behavior)
- Variations in gastric motility patterns

Genomic research is underway to identify genetic markers associated with increased susceptibility, but as of now, no single gene mutation has been recognized. GDV risk is likely polygenic.

4.2 Conformational Risk Factors

Conformation remains one of the most robustly documented contributors.

4.2.1 Deep-Chested Conformation

Dogs with a narrow thoracic width relative to height exhibit a higher likelihood of gastric displacement and volvulus. This body type provides increased space for the stomach to swing within the cranial abdomen and may predispose to rotation during episodes of dilatation.

4.2.2 Lean Body Condition

Multiple epidemiologic studies suggest that dogs with a thin or lean body condition score (BCS) are more susceptible than overweight dogs. This could reflect reduced visceral fat providing less stabilization of the stomach.

4.3 Age-Related Factors

While GDV can affect dogs of all ages, risk increases significantly with advancing age. Potential explanations include:

- Progressive weakening or stretching of gastric ligaments
- Age-associated decreases in gastric motility
- Higher prevalence of underlying comorbidities
- Declining muscle tone contributing to organ mobility

Interestingly, older dogs with lifelong stable anatomy may suddenly develop GDV due to age-related physiologic changes.

4.4 Behavioral and Psychological Factors

Behavioral characteristics particularly **stress reactivity** appear to play a notable role.

4.4.1 Stress and Anxiety

Dogs that exhibit anxious or fearful behavior are more likely to develop GDV. Stress increases aerophagia, alters gastric motility, and may induce hormonal changes that influence gastrointestinal function.

4.4.2 Eating Habits

Rapid eating is associated with:

- Increased air swallowing
- Faster gastric filling
- Greater risk of acute distension

Dogs in multi-animal households, where competition for food is common, often eat more quickly.

4.5 Dietary Risk Factors

Diet is frequently implicated in GDV pathogenesis, although many findings remain associative rather than causal.

4.5.1 Meal Size and Frequency

Large, infrequent meals significantly increase the risk of GDV. Dogs fed once daily show higher incidence than those fed two or more meals per day.

4.5.2 Kibble Size and Composition

Some studies suggest that:

- Dry kibble containing high-fat content as one of the first ingredients
- Foods with high fermentable carbohydrate levels

- Diets low in roughage

may elevate the probability of GDV. Whether this is due to delayed gastric emptying or gas accumulation remains a subject of investigation.

4.5.3 Feeding Bowl Height

Historically, elevated feeding bowls were recommended to reduce aerophagia. However, more recent data indicate that raised bowls may increase the risk of GDV in large breeds. The causative mechanism remains unclear.

4.6 Environmental and Management Factors

4.6.1 Exercise Timing

Strenuous activity immediately before or after meals has been correlated with GDV. Aerophagia, gas accumulation, and gastric displacement may be exacerbated during physical exertion.

4.6.2 Temperature and Weather

Some studies have suggested increased incidence during periods of rapid temperature or atmospheric pressure changes, though evidence is inconclusive.

4.6.3 Boarding, Travel, and Routine Disruption

Periods of environmental stress boarding kennels, long car rides, owner absence may precipitate GDV in predisposed dogs.

Functional abnormalities in gastric motility are frequently discussed as potential contributors.

4.7.1 Delayed Gastric Emptying

Delayed emptying may lead to gas accumulation and distension. Possible factors include:

- Autonomic dysfunction
- Reduced interstitial cell of Cajal density
- Subclinical inflammation
- Hormonal imbalances

4.7.2 Dysbiosis and Gas Production

Emerging research examines whether alterations in gastric or intestinal microbiota contribute to excessive fermentation and gas formation. While findings are preliminary, dysbiosis is a promising area of investigation.

4.8 Multifactorial Model

The most widely accepted model proposes that GDV results from the interaction of:

- Anatomic predisposition
- Genetic susceptibility
- Environmental triggers
- Stress and behavior
- Dietary influences
- Motility abnormalities

This multifactorial interplay explains why not all predisposed dogs develop GDV and why certain animals experience repeated episodes despite intervention.

5. Pathophysiology of Gastric Dilatation-Volvulus

GDV is a dynamic and rapidly progressive condition. Understanding its pathophysiology requires examining the sequence of events beginning with gastric dilatation, followed by rotation, obstruction, vascular compromise, and

ultimately systemic shock. Each stage contributes to the rapid deterioration characteristic of this syndrome. The interplay of mechanical, circulatory, metabolic, and inflammatory processes forms the core of the pathophysiological cascade.

5.1 Initiation: Gastric Dilatation

Gastric dilatation is the initial event, regardless of whether it is primary or secondary to volvulus. Several mechanisms can lead to accumulation of gas, fluid, or ingesta:

5.1.1 Aerophagia

Excessive air swallowing occurs in dogs that eat rapidly, exhibit anxiety, or compete for food. Gas accumulates quickly when eructation is impaired or delayed.

5.1.2 Impaired Gastric Outflow

Functional or partial obstruction of the pylorus slows gastric emptying. Although not a sole cause of GDV, delayed emptying can significantly increase intragastric pressure.

5.1.3 Gas Production

Microbial fermentation of dietary substrates within the stomach may release gas. Diet composition influences the extent of fermentation.

5.1.4 Fluid Secretion

Distension stimulates gastric secretions, further adding to stomach volume.

The increase in gastric volume elevates pressure against the diaphragm and abdominal walls, causing discomfort and rapid abdominal enlargement.

5.2 Progression: Gastric Volvulus

Volvulus occurs when the distended stomach rotates on its mesenteric axis. In the majority of cases, rotation is clockwise, typically ranging from 180° to 360°.

5.2.1 Anatomical Rotation

During volvulus:

- The pylorus moves dorsally and to the left.
- The fundus shifts ventrally and to the right.
- The esophageal and pyloric orifices become obstructed.

This anatomical displacement traps gas and stomach contents, preventing normal release. Rotation of the duodenum and occlusion of gastric vasculature initiate ischemic changes.

5.3 Mechanical Consequences of Volvulus

The rotation of the stomach precipitates several critical mechanical outcomes:

5.3.1 Gastric Outflow and Inflow Obstruction

Both the esophageal and pyloric openings become occluded, creating a one-way valve effect. Gas accumulates continuously, rapidly increasing intragastric pressure.

5.3.2 Compression of Surrounding Organs

The enlarged stomach compresses:

- The caudal vena cava
- The portal vein
- The diaphragm

This compression compromises respiration and venous return.

5.3.3 Spleen Displacement

Because the spleen is connected to the stomach by the gastrosplenic ligament, it often rotates with the stomach. Splenic congestion, thrombosis, or infarction may occur.

5.4 Vascular Compromise and Ischemia

Vascular effects are central to the severity of GDV.

5.4.1 Venous Obstruction

Obstruction of gastric veins occurs early, leading to:

- Venous congestion
- Edema of the stomach wall
- Hemorrhage

Arterial flow may initially persist, causing worsening engorgement before complete ischemia ensues.

5.4.2 Arterial Blood Flow Reduction

As pressure continues to rise, arterial supply becomes compromised. Tissue hypoxia and ischemic necrosis of the gastric wall follow.

5.4.3 Splenic Ischemia

The spleen may develop:

- Venous thrombosis
- Torsion
- Infarction
- Rupture (rare)

These changes can contribute to intraoperative hemorrhage and complicate surgical management.

5.5 Systemic Circulatory Collapse

As vascular compromise worsens, systemic effects become profound.

5.5.1 Obstructive Shock

Compression of the caudal vena cava and portal vein reduces venous return, leading to:

- Decreased preload
- Reduced cardiac output
- Compensatory tachycardia
- Systemic hypotension

This constitutes obstructive shock, one of the defining features of GDV.

5.5.2 Hypovolemic Shock

Fluid shifts into the stomach and peritoneal cavity contribute to:

- Intravascular volume depletion
- Hemoconcentration
- Decreased tissue perfusion

Combined obstructive-hypovolemic shock rapidly progresses to cardiovascular collapse if untreated.

5.6 Metabolic and Biochemical Disturbances

Several metabolic consequences result from impaired circulation and tissue perfusion.

5.6.1 Metabolic Acidosis

Reduced perfusion leads to lactic acid production. Elevated lactate is not only a marker of shock but also a predictor of gastric necrosis.

5.6.2 Electrolyte Imbalances

Common disturbances include:

- Hypokalemia
- Hypochloremia
- Hyponatremia

These arise from vomiting, gastric fluid sequestration, and compromised renal perfusion.

5.6.3 Gastric Reperfusion Injury

Although essential to recovery, restoring blood flow after ischemia can release free radicals and inflammatory mediators that damage tissues.

5.7 Inflammatory and Coagulation Responses

GDV triggers systemic inflammatory cascades.

5.7.1 Systemic Inflammatory Response Syndrome (SIRS)

Ischemia-reperfusion, endotoxin absorption, and cytokine release may lead to SIRS, characterized by:

- Leukocytosis or leukopenia
- Hyper- or hypothermia
- Tachycardia
- Tachypnea

5.7.2 Coagulopathy and DIC

Disseminated intravascular coagulation (DIC) is a feared sequela, arising from:

- Endothelial injury
- Platelet activation
- Consumption of clotting factors

Dogs developing DIC have significantly higher mortality.

5.8 Gastrointestinal Wall Changes

The degree of gastric wall damage is a major prognostic factor.

5.8.1 Mucosal Damage

Mucosal ischemia develops early, even before full-thickness necrosis. This can lead to bacterial translocation into the bloodstream.

5.8.2 Gastric Necrosis

Necrosis usually affects the fundus and greater curvature because these areas receive terminal arterial supply. Necrotic regions appear:

- Dark purple to black
- Thin or friable
- Avascular upon incision

5.8.3 Risk of Perforation

Severe necrosis may lead to gastric perforation, causing septic peritonitis a life-threatening complication.

5.9 Cardiopulmonary Compromise

5.9.1 Respiratory Dysfunction

The distended stomach compresses the diaphragm, restricting lung expansion and contributing to hypoventilation.

5.9.2 Cardiac Arrhythmias

Arrhythmias, particularly ventricular premature contractions (VPCs), are common. Causes include:

- Myocardial hypoperfusion
- Electrolyte disturbances
- Reperfusion injury

Persistent arrhythmias are associated with increased mortality, though many resolve with stabilization.

5.10 Summary of Pathophysiology

GDV represents a severe and rapidly escalating cascade involving:

- Mechanical obstruction
- Vascular collapse
- Tissue ischemia
- Systemic inflammatory response
- Metabolic derangement

Understanding this progression underscores the urgency of rapid diagnosis, aggressive stabilization, and surgical intervention to prevent irreversible organ damage.

6. Clinical Presentation

The clinical presentation of GDV is typically dramatic and rapidly progressive. Dogs often deteriorate over a period of hours, though early signs may initially appear subtle. Prompt recognition is crucial since survival improves significantly with early intervention. Although presentation may vary depending on duration and severity of torsion, several hallmark signs are consistently reported.

6.1 Early Clinical Signs

During the early stages particularly when dilatation precedes complete volvulus signs may be vague or nonspecific:

6.1.1 Restlessness and Anxiety

Affected dogs often display agitation or pacing due to abdominal discomfort. They may repeatedly change position in an attempt to relieve pressure.

6.1.2 Hypersalivation and Drooling

Stimulation of gastric and esophageal afferent nerves leads to excessive salivation, which may be accompanied by foamy drool.

6.1.3 Non-Productive Retching

This is considered one of the most characteristic early signs. Dogs attempt to vomit but produce only small amounts of froth or nothing at all, due to esophageal obstruction created by the rotated stomach.

6.1.4 Mild Abdominal Distension

Distension may begin as subtle abdominal fullness, more noticeable in short-haired breeds.

6.2 Progressive Clinical Signs

As volvulus develops and intragastric pressure rises, clinical signs become more pronounced.

6.2.1 Severe Abdominal Distension

The abdomen becomes markedly distended and tympanic. The cranial abdomen is often firm to palpation.

6.2.2 Tachypnea and Respiratory Distress

Diaphragmatic compression restricts lung expansion. Dogs may exhibit:

- Rapid, shallow breathing
- Extended neck posture
- Increased effort on inhalation

6.2.3 Hypovolemic and Obstructive Shock

Signs include:

- Pale mucous membranes
- Prolonged capillary refill time
- Weak peripheral pulses
- Tachycardia

As shock progresses, dogs may become recumbent, obtunded, or unresponsive.

6.2.4 Hypothermia

Reduced perfusion results in decreased body temperature, particularly in advanced cases.

6.3 Terminal Signs

Without intervention, GDV progresses to multiorgan failure. Terminal signs include:

- Collapse
- Bradycardia (late shock)
- Cyanosis
- Loss of consciousness
- Respiratory arrest

These signs indicate imminent death unless immediate, aggressive resuscitation and surgery are performed.

6.4 Physical Examination Findings

Veterinarians should assess:

6.4.1 Abdominal Palpation

A distended, drum-like stomach is strongly suggestive of GDV. Palpation often elicits discomfort.

6.4.2 Cardiovascular Assessment

Indicators of shock:

- Weak femoral pulses
- Prolonged CRT (>2 seconds)
- Tachycardia (commonly >160-180 bpm)
- Arrhythmias, particularly ventricular premature contractions (VPCs)

6.4.3 Respiratory Assessment

Dyspnea caused by diaphragmatic compression can be severe.

6.4.4 Mentation

Affected dogs may range from anxious to stuporous, depending on perfusion status.

7. Diagnostic Approach

GDV diagnosis requires rapid assessment. Immediate stabilization often occurs concurrently with diagnostic procedures. Definitive diagnosis is based on a combination of clinical suspicion, radiographic confirmation, and laboratory findings.

7.1 Initial Assessment and Triage

GDV patients should be immediately triaged as critical emergencies. The first priority is to:

- Assess cardiovascular stability
- Establish vascular access
- Initiate fluid resuscitation
- Provide oxygen supplementation

These steps improve the patient's ability to tolerate diagnostic procedures and subsequent anesthesia.

7.2 Radiographic Evaluation

7.2.1 Right Lateral Abdominal Radiograph

This is the gold standard for GDV diagnosis.

Key findings:

- **"Double bubble" sign:** Separates the pyloric antrum from the fundus, indicating compartmentalization.
- **"Popeye arm" appearance:** The pylorus displaced dorsally and to the left.
- Distended gas-filled stomach with compartmental lines.

7.2.2 Ventrodorsal and Dorsoventral Views

Rarely necessary for diagnosis and may be difficult for unstable patients. These views can further characterize splenic involvement but are generally secondary in urgency.

7.3 Laboratory Evaluation

Diagnostic laboratory tests do not confirm GDV but are essential for prognostic assessment and treatment planning.

7.3.1 Serum Lactate

Lactate is one of the most important prognostic markers.

- Elevated levels (>6 mmol/L) may indicate gastric necrosis.
- Improvement after fluid therapy is associated with higher survival rates.

7.3.2 Complete Blood Count (CBC)

Common abnormalities:

- Stress leukogram (neutrophilia, lymphopenia)
- Hemoconcentration due to hypovolemia
- Thrombocytopenia (possible early DIC)

7.3.3 Serum Biochemistry

Important values include:

- Electrolytes (K, Na, Cl)
- Renal parameters (BUN, creatinine)
- Liver enzymes
- Glucose

Electrolyte abnormalities guide perioperative management.

7.3.4 Coagulation Profile

Indicated when DIC is suspected. Signs include:

- Prolonged PT/aPTT
- Low fibrinogen
- Elevated D-dimers

Dogs with coagulopathies require careful intraoperative monitoring.

7.4 Electrocardiography (ECG)

Cardiac monitoring is vital due to arrhythmia prevalence.

7.4.1 Ventricular Arrhythmias

Most common in GDV, generally caused by:

- Myocardial hypoxia
- Acidosis
- Reperfusion injury

7.4.2 Monitoring Strategy

Continuous ECG monitoring should begin during stabilization and continue for 24-48 hours postoperatively.

7.5 Point-of-Care Ultrasound (POCUS)

While not used to confirm volvulus, ultrasound can assist in assessing:

- Gastric wall viability
- Free abdominal fluid
- Splenic perfusion

POCUS is particularly useful when radiography is delayed or the patient is too unstable to reposition.

7.6 Differential Diagnoses

Conditions that may mimic early GDV signs include:

- Gastric dilatation without volvulus
- Mesenteric torsion
- Severe pancreatitis
- Peritonitis
- Intestinal obstruction

Radiographic confirmation is critical since clinical signs alone can be misleading.

7.7 Diagnostic Priorities in GDV Management

Given the time-sensitive nature of GDV, diagnostics must be streamlined:

1. Rapid triage
2. Immediate IV catheter placement and fluid resuscitation
3. Fast, high-yield diagnostics (right lateral radiographs, serum lactate)
4. Concurrent preparation for emergency surgery

Time lost during diagnosis correlates directly with increasing morbidity.

8. Emergency Stabilization and Medical Management

Management of GDV begins the moment the patient arrives. Early stabilization is essential for improving perfusion, reducing shock, and optimizing the patient's ability to tolerate anesthesia and surgical correction. Stabilization should occur concurrently with diagnostic procedures whenever feasible.

8.1 Initial Priorities

The first goals in emergency management are:

1. Restoring circulatory volume
2. Decompressing the stomach
3. Stabilizing the cardiopulmonary system
4. Correcting metabolic derangements

This approach reduces morbidity and mortality while preparing the patient for surgery.

8.2 Intravenous Access and Fluid Therapy

8.2.1 Immediate IV Catheterization

Two large-bore (16-18 gauge) IV catheters should be placed, ideally in the cephalic veins. The saphenous veins are typically avoided because caudal venous return is compromised by the distended stomach.

8.2.2 Shock Fluid Therapy

Aggressive isotonic crystalloid therapy is indicated:

- Boluses of 20-30 mL/kg over 10-15 minutes
- Repeated while monitoring perfusion parameters

In some dogs, colloids or hypertonic saline may be added to improve perfusion more rapidly, especially if marked hypovolemia is present.

8.2.3 Endpoint Monitoring

Key parameters to evaluate response include:

- Heart rate
- Pulse quality
- Mucous membrane color
- Capillary refill time
- Blood pressure
- Lactate concentration

Failure to respond suggests severe vascular compromise or ongoing obstruction.

8.3 Analgesia and Sedation

Pain control is essential as GDV is extremely painful.

8.3.1 Opioids

Pure mu-agonists (e.g., hydromorphone, fentanyl) are recommended because they provide rapid, effective analgesia with minimal cardiovascular depression.

8.3.2 Sedatives

Benzodiazepines (diazepam or midazolam) may be used for anxiolysis. Alpha-2 agonists are generally avoided due to cardiovascular depressive effects.

8.4 Gastric Decompression

Decompression is performed as soon as the patient is stable enough to tolerate the procedure.

8.4.1 Orogastric Intubation

The preferred initial method.

Technique considerations

- A well-lubricated stomach tube is gently advanced through the esophagus into the stomach.
- Resistance may occur due to torsion; gentle rotation or repositioning may help.
- Once placed, gas, fluid, and ingesta are relieved.

8.4.2 Percutaneous Gastrocentesis

If intubation fails or the dog cannot tolerate it, needle decompression is performed.

- Large-bore 14-16 gauge needles or catheters are inserted percutaneously into the stomach.
- Regional anesthesia may be used.

Gastrocentesis often provides immediate relief of pressure and improves ventilation.

8.5 Oxygen Therapy

Given the combined effects of hypovolemia, shock, and diaphragmatic compression, supplemental oxygen is recommended in all patients. Oxygen may be delivered via:

- Flow-by
- Nasal cannula
- Oxygen cage

8.6 Management of Cardiac Arrhythmias

Arrhythmias often emerge during stabilization or postoperatively.

8.6.1 Ventricular Arrhythmias

VPCs and ventricular tachycardia are the most common. Treatment is indicated when arrhythmias:

- Cause hemodynamic instability
- Are sustained
- Have R-on-T phenomenon
- Develop into multiform VPCs

8.6.2 Antiarrhythmic Therapy

Commonly used drugs include:

- Lidocaine (bolus + CRI)
- Procainamide

Electrolytes, particularly potassium and magnesium, should be corrected as needed.

9. Surgical Management

Surgery is the definitive treatment for GDV. Once the patient is stabilized and decompressed, immediate surgical intervention is necessary to derotate the stomach, assess tissue viability, perform partial gastrectomy if required, and carry out gastropexy to prevent recurrence.

9.1 Timing of Surgery

Surgery should occur as soon as the patient is hemodynamically stable enough to tolerate anesthesia. Delaying surgery increases the risk of:

- Gastric necrosis
- Splenic torsion
- Progressive shock
- Perforation

9.2 Anesthetic Considerations

Dogs with GDV present unique anesthetic challenges:

- Hypovolemia and shock
- Potential arrhythmias
- Risk of aspiration
- Compromised ventilation due to gastric distension

9.2.1 Induction

Etomidate or a combination of opioids and benzodiazepines may be used for cardiovascular stability. Propofol is commonly used but may cause hypotension.

9.2.2 Maintenance

Isoflurane or sevoflurane are typical inhalants.

9.2.3 Monitoring

Continuous monitoring of ECG, blood pressure, oxygen saturation, capnography, and temperature is essential.

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Etomidate or a combination of opioids and benzodiazepines may be used for cardiovascular stability. Propofol is commonly used but may cause hypotension.

9.2.2 Maintenance

Isoflurane or sevoflurane are typical inhalants.

9.2.3 Monitoring

Continuous monitoring of ECG, blood pressure, oxygen saturation, capnography, and temperature is essential.

9.4 Derotation of the Stomach

Once the abdomen is opened, the stomach is identified by its distended, gas-filled appearance and abnormal orientation. The surgeon manually derotates the stomach by:

- Grasping the pyloric antrum and fundus
- Rotating the stomach counter to the direction of the volvulus (typically counterclockwise)

Derotation must be performed gently to avoid tearing weakened tissue.

9.5 Assessment of Gastric Viability

Determining gastric viability is one of the most critical intraoperative decisions.

9.5.1 Assessment Criteria

Surgeons evaluate:

- Color (pink/red = viable; black/green = necrotic)
- Thickness and texture
- Arterial pulsations
- Bleeding upon incision
- Fluorescein staining (rarely used now)

9.5.2 Gastric Necrosis

If necrotic tissue is identified, a partial gastrectomy is performed. The fundus and greater curvature are most often affected.

9.6 Management of Splenic Involvement

Because the spleen rotates with the stomach, splenic complications are common.

9.6.1 Indications for Splenectomy

- Irreversible torsion
- Severe congestion
- Thrombosis
- Infarction
- Suspected neoplasia (incidental finding)

Splenectomy is performed only when necessary, as removing the spleen may increase postoperative risk in some patients.

9.7 Gastropexy Techniques

Gastropexy is essential in preventing recurrence, which otherwise can exceed 70%.

9.7.1 Incisional Gastropexy

The most common method; involves creating incisions in the stomach and abdominal wall and suturing the two structures together.

9.7.2 Circumcostal Gastropexy

Provides strong attachment but is more invasive.

9.7.3 Belt-Loop Gastropexy

Creates a loop of stomach wall anchored to the body wall.

9.7.4 Laparoscopic-Assisted Gastropexy

Minimally invasive and often used prophylactically.

9.8 Abdominal Lavage and Closure

After addressing all abnormalities, the abdomen is lavaged if contamination is present. Closure requires careful management of compromised tissues to avoid postoperative complications such as dehiscence.

10. Postoperative Management

Successful surgical correction is only the beginning; intensive postoperative care is critical for survival.

10.1 Monitoring in the Immediate Postoperative Period

Patients should be monitored continuously for:

- Arrhythmias
- Hypotension
- Hypothermia
- Pain
- Hemorrhage
- Gastric ileus
- Signs of peritonitis

10.2 Fluid and Electrolyte Management

Fluid therapy continues postoperatively to maintain perfusion and correct ongoing losses. Electrolytes should be monitored every 4-6 hours initially.

10.3 Pain Management

Opioids remain the mainstay, often supplemented with:

- Lidocaine CRIs
- Ketamine CRIs
- Local blocks
- NSAIDs (only when stable and well hydrated)

10.4 Arrhythmia Management

Many arrhythmias resolve within 24-48 hours after correction of shock and reperfusion injury. Persistent arrhythmias require:

- Continuous ECG
- Lidocaine or other antiarrhythmic therapy
- Correction of underlying metabolic disturbances

10.5 Nutritional Support

Feeding typically resumes within 12-24 hours, beginning with:

- Small, frequent meals
- Easily digestible food
- Slow transitions back to normal diet

Early feeding promotes gastrointestinal motility and improves recovery.

11. Long-Term Prognosis and Outcomes

Survival outcomes for dogs with GDV have improved significantly with advancements in emergency medicine, anesthesia, and surgical techniques. However, prognosis depends heavily on multiple factors related to the timing of treatment, the degree of gastric compromise, and the patient's overall physiologic state.

11.1 Factors Influencing Prognosis

11.1.1 Time to Presentation and Treatment

The single greatest predictor of survival is the speed with which:

- GDV is recognized
- Stabilization is initiated
- Surgery is performed

Delays contribute to worsening ischemia, necrosis, and shock.

11.1.2 Gastric Necrosis

Presence of full-thickness gastric necrosis significantly worsens prognosis, with reported survival rates dropping by 30-50%. Dogs requiring partial gastrectomy face greater anesthetic and postoperative risks.

11.1.3 Serum Lactate Concentration

Admission lactate >6-7 mmol/L is associated with increased mortality especially when levels do not decrease after fluid therapy. However, lactate should not be used alone to determine prognosis.

11.1.4 Hypotension and Shock Severity

Poor perfusion before surgery increases the likelihood of:

- Disseminated intravascular coagulation (DIC)
- Ventricular arrhythmias
- Acute kidney injury

11.1.5 Age and Concurrent Disease

Older dogs and those with significant comorbidities (cardiac disease, endocrine disorders) may require more intensive postoperative care.

11.2 Short-Term Survival Rates

Modern studies report survival rates:

- **80-90%** for dogs without gastric necrosis
- **50-70%** when necrosis is present
- **>95%** for prophylactic gastropexy performed electively (no GDV involved)

Mortality remains highest in dogs with severe shock, multiple organ dysfunction, or late presentation.

11.3 Postoperative Complications

11.3.1 Cardiac Arrhythmias

Arrhythmias often appear within the first 24-48 hours and may resolve with supportive care. Persistent arrhythmias require treatment and are associated with poorer outcomes.

11.3.2 Gastric Ileus

Postoperative motility disorders can lead to vomiting, regurgitation, or anorexia, necessitating prokinetic therapy (metoclopramide, cisapride).

11.3.3 Peritonitis

Occurs due to gastric perforation, necrosis, or surgical contamination. Prognosis is guarded if septic peritonitis develops.

11.3.4 DIC

A life-threatening complication in severe or late cases of GDV.

11.3.5 Incisional Complications

Dehiscence, infection, or seroma formation may occur, particularly in hypotensive patients.

11.4 Long-Term Quality of Life

Most dogs that survive surgical treatment and recover from GDV resume a normal quality of life. Owners report full return to activity, appetite, and function. Dogs undergoing gastropexy (incisional or laparoscopic) generally experience:

- Permanent stabilization of the stomach
- No long-term digestive impairment
- Extremely low recurrence risk (<5% with proper technique)

12. Prevention Strategies

Because GDV carries significant morbidity and mortality, prevention particularly in high-risk breeds is crucial.

12.1 Prophylactic Gastropexy

Gastropexy is by far the most effective preventive measure.

12.1.1 Indications

Recommended for:

- High-risk breeds (Great Dane, Weimaraner, Standard Poodle, Irish Setter, etc.)
- Dogs with first-degree relatives that experienced GDV
- Dogs undergoing elective abdominal surgery

12.1.2 Timing

Often performed at the time of:

- Spay/neuter
- Laparoscopic procedures
- Other abdominal surgeries

12.1.3 Techniques

Laparoscopic-assisted gastropexy is increasingly popular due to:

- Less postoperative pain
- Smaller incisions
- Faster recovery
- Strong, reliable attachment

Prophylactic gastropexy does not prevent gastric dilatation but prevents life-threatening volvulus.

12.2 Dietary and Feeding Management

12.2.1 Meal Size and Frequency

Feeding smaller, more frequent meals reduces the risk of acute gastric overload.

12.2.2 Avoidance of High-Fat Diets

Dietary fat affects gastric emptying. High-fat foods may delay motility and promote distension.

12.2.3 Avoid Raised Feeding Bowls

Current evidence suggests elevated bowls may increase risk in large breeds.

12.2.4 Discourage Rapid Eating

Tools include:

- Slow-feeder bowls
- Puzzle feeders
- Dividing food into multiple bowls

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