Esophagus: Anatomy, Physiology,

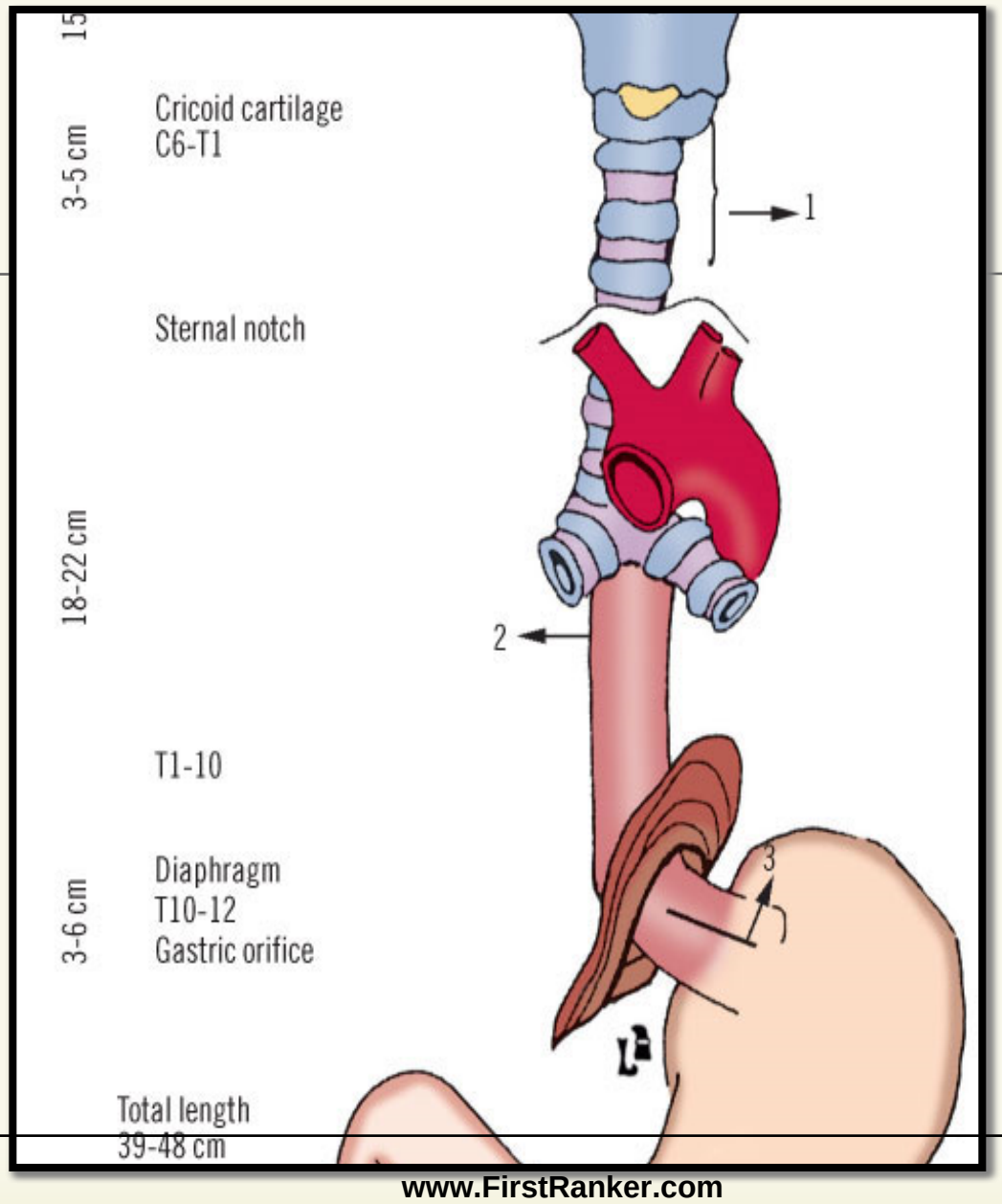
Corrosive stricture
&
Perforation of Esophagus

Anatomy

- The primitive foregut forms during the fourth week of gestation by a longitudinal folding and incorporation of the dorsal part of the yolk sac into the embryo.
- 34th day: The distal esophagus elongates first, followed by the proximal.
- 6th week: Mesenchymal circular muscle coat develops
- Three to nine weeks later, longitudinal musculature appears.

- Seventh to eighth week: Esophageal lumen is almost filled with cells from the proliferated esophageal epithelium.

- During the 4th month, the muscularis mucosa appears

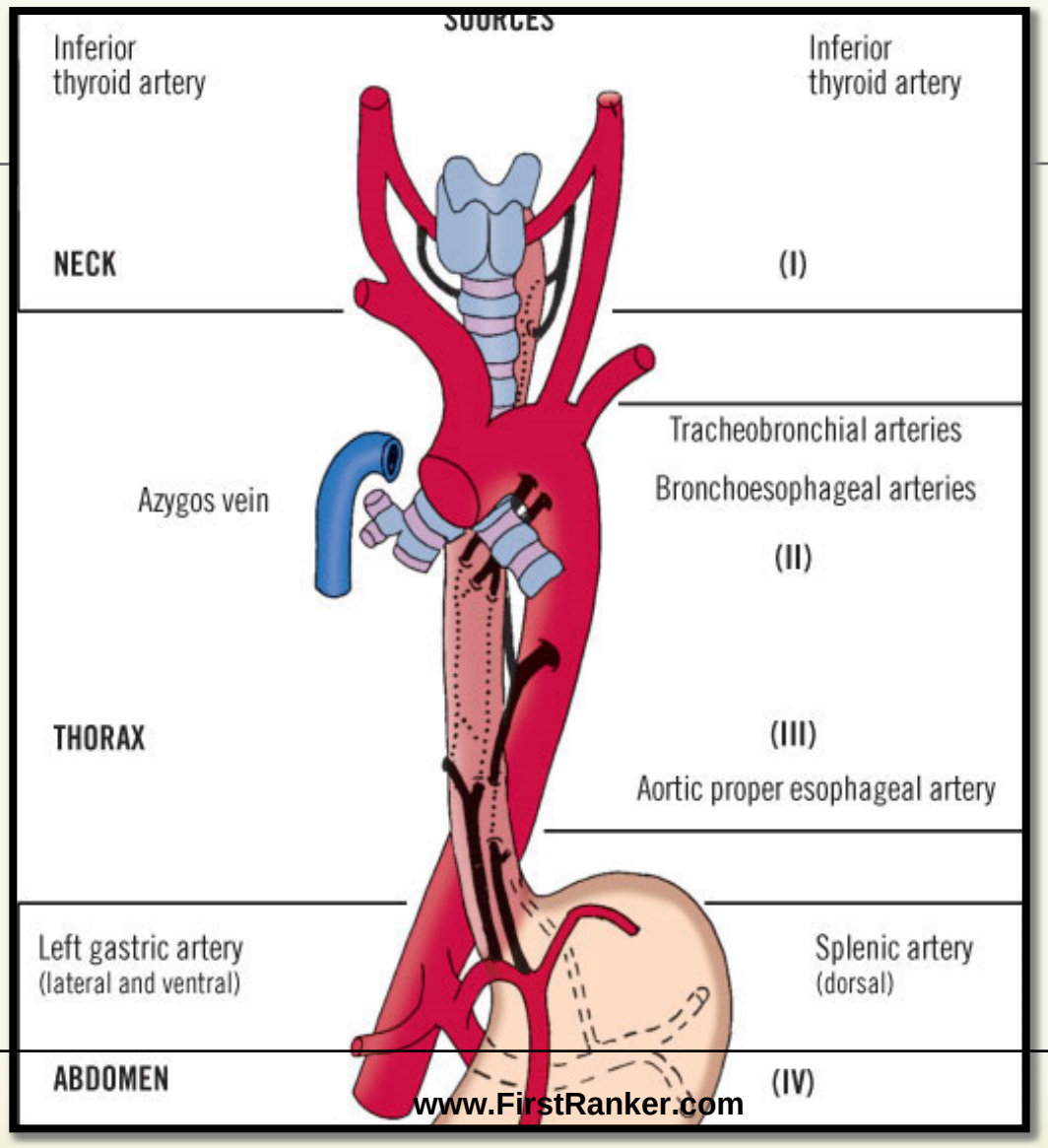


- narrowest tube of the gastrointestinal tract
- Midline structure anterior to the spine and posterior to the trachea
- Length: ranges from 21 cm-34 cm (27 cm average).

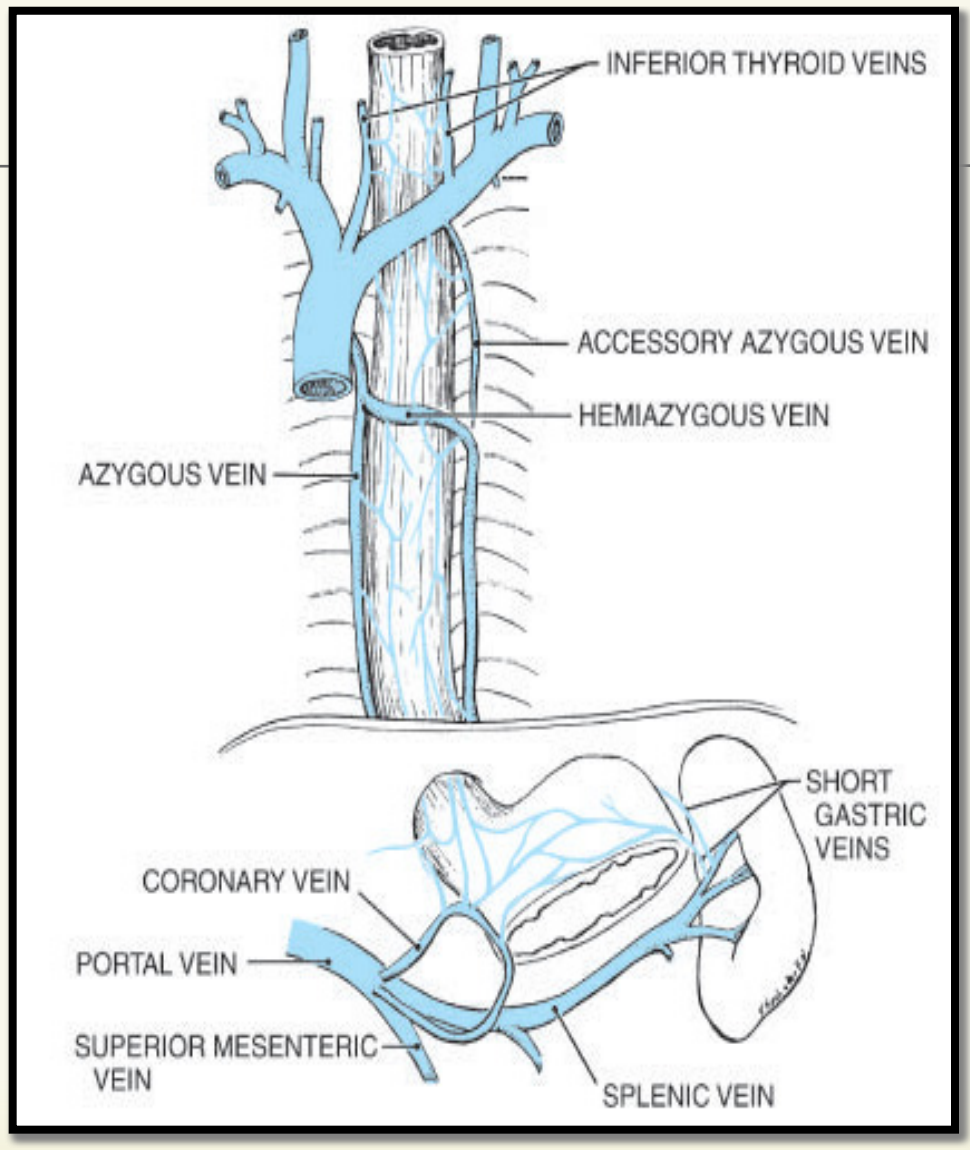
- Classical anatomy divides the esophagus into three parts:
  - Cervical
  - Thoracic
  - Abdominal

- Function divides the esophagus according to its differing forms of motility into the following three zones :
  - Upper esophageal sphincter (UES)
  - Esophageal body
  - Lower esophageal sphincter (LES)

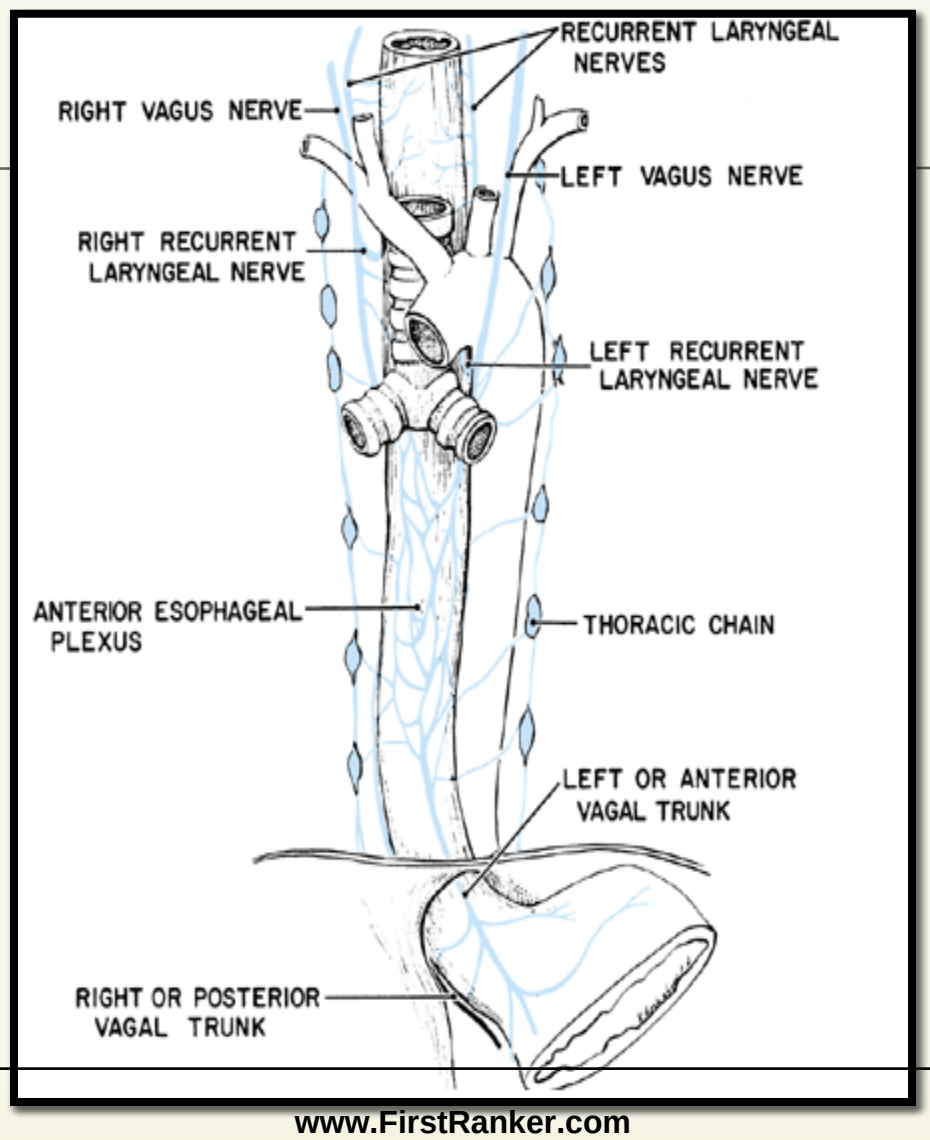
Arterial Supply



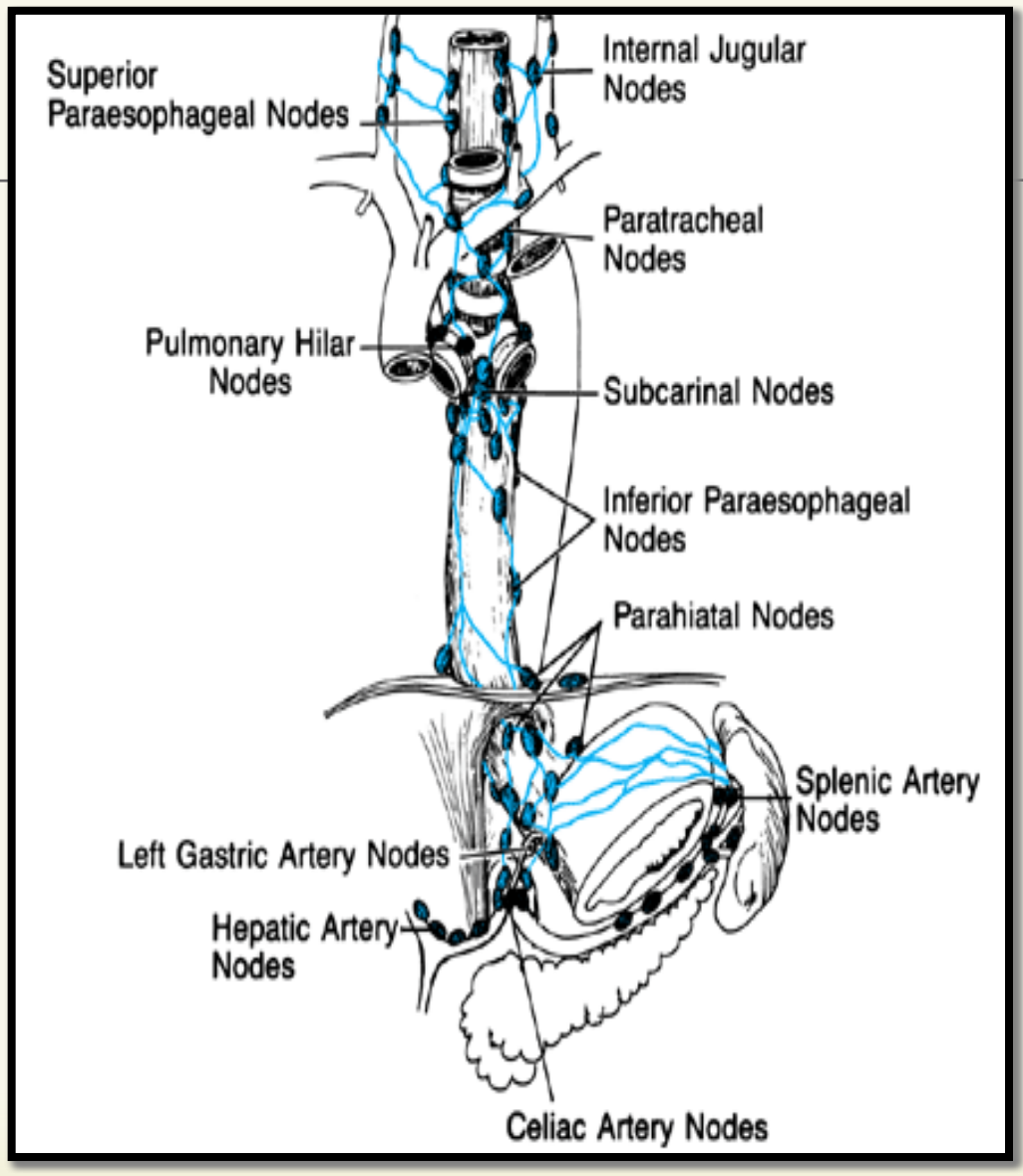
Venous Supply



Innervation



Lymphatic drainage



Histology

- It lacks a serosal coating
- The four layers are:
  - mucosa
  - Submucosa
  - muscularis
  - tunica adventitia

Corrosive stricture

Stricture formation, which usually develops between 3 and 8 weeks after the initial injury but sometimes requires a much longer period for evolution

Etiology

Alkaline caustics, acid or acidlike corrosives, and household bleaches. Hydrochloric, sulfuric, nitric, and phosphoric acids are contained in automobile battery acids.

Age

- 75% of injuries involving children younger than 5 years and a much lower, secondary peak occurring in 20-30

Type of caustic related to injury

Acid

- – Generally less severe injury
- – Coagulative necrosis
- – Coagulum lessen tissue penetration

Alkaline

- Liquefactive necrosis
- Sodium hydroxide
- Very hazardous
- 30% causes full thickness necrosis

The severity of esophageal and gastric damage resulting from a caustic ingestion depends on

- Corrosive properties
- Concentration of the agent
- Quantity swallowed

Pathogenesis

Corrosive enter to stomach - > reflex pyloric spasm

Limit passage of corrosive to duodenum

Regurgitation of corrosive against a closed cricopharyngeus - > damage to esophagus and Stomach

3 - 5 mins - > gastric atonia - > opening of pylorus

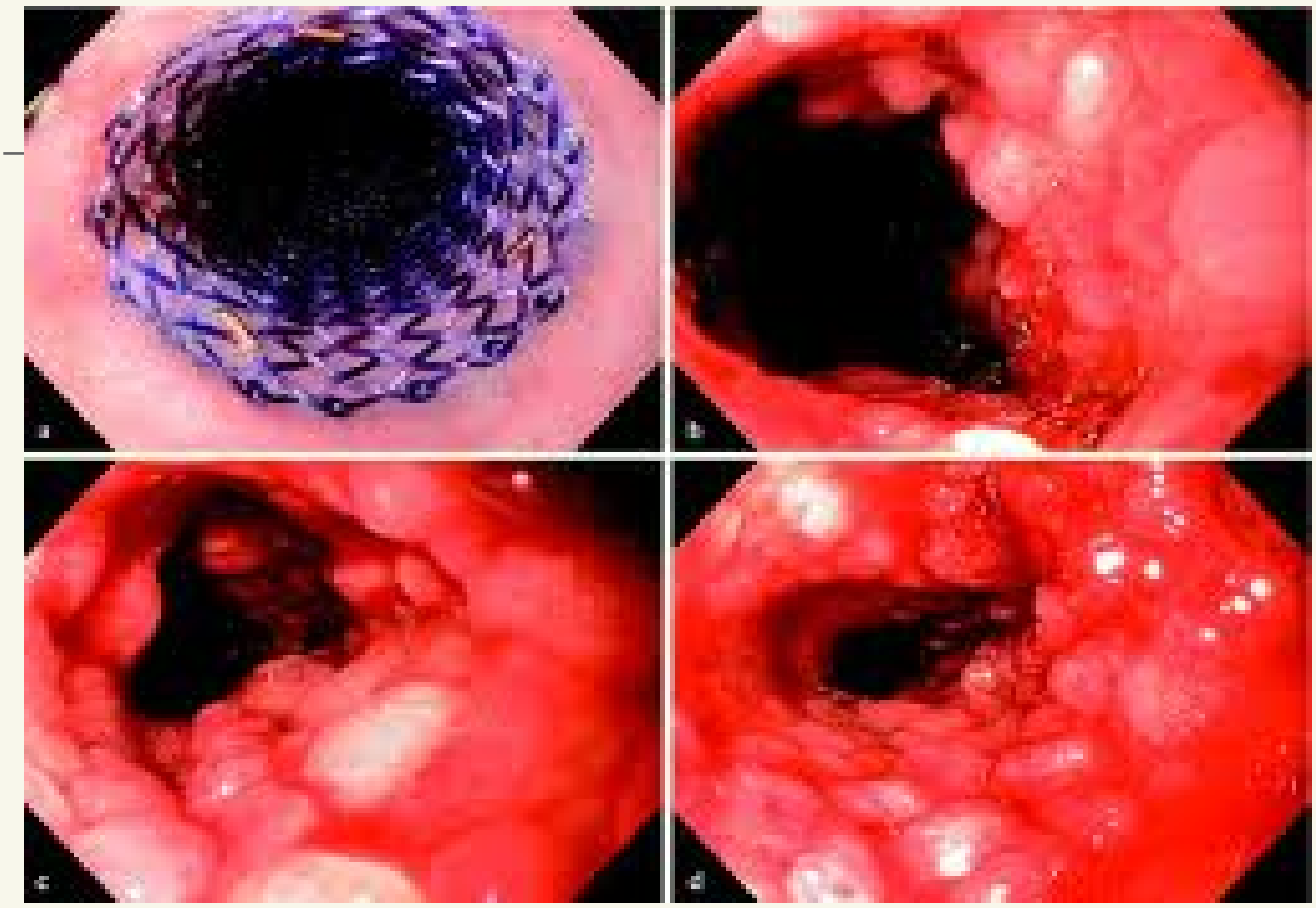
Goal of emergency management

- Limit and treat the immediately life-threatening consequences
- Control subsequent stricture formation

Endoscopic findings

Zargar's grading of mucosal injury caused by corrosive ingestion	
Grade 0	Normal examination
Grade 1	Edema & hypermia of the mucosa
Grade 2a	Superficial ulceration, erosions, friablility
Grade 2b	Grade 2a + deep discrete or circumferential ulcerations
Grade 3a	Small scattered areas of multiple ulceration & areas of necrosis with brown black / greyish discoloration
Grade 3b	Extensive necrosis

Zargar et al GIE 1991; Orringer 1993



Early management

Resuscitation

- Upper airway
  - Assessment of severity of damage
  - Secure the airway
- Fiberoptic intubation
- Tracheostomy

Contraindication

- Emetics
- OG or NG
- Neutralization

  Alkali ---try Milk

  Acid---- do not try anything

Surgery is warranted if evidence of

- Perforation of the esophagus or stomach
- Mediastinitis
- Peritonitis exists

Treatment

- Corticosteroids to modify the inflammatory response to the burn injury
- Antibiotics to control secondary bacterial infection
- Esophagoscopy within 12-24 hrs

- Bougienage
- Esophageal stents
- Colon interposition
  - Forearm tube
  - Free jejunal flap

Perforation of Esophagus

Introduction

- Grand Admiral of Holland died of spontaneous rupture of the esophagus in 1724
- J. R. Meyer of Berlin was the first to recognize this disease prior to death
- Barrett made the first early diagnosis and performed the first surgical repair in 1946

Anatomy

- Esophagus lacks serosa
  - More likely to rupture
- Site of rupture:
  - More commonly on left side
  - Due to instrumentation: distal esophagus
  - Spontaneous: posterolateral esophagus
- Tears are usually longitudinal

Etiology

- **Iatrogenic**
  - **Instrumentation (MC cause)**

    most common site of perforation during endoscopy is at the cricopharyngeus
  - **Surgical injury**

- **Boerhaave Syndrome** (barogenic perforation, postemetic perforation,spontaneous esophageal rupture)

- Always occurs on the left side of the distal third of esophagus

- Most tears occur along the longitudinal axis (0.6 to 8.9 cm) long

The **mucosal tear is often longer than the muscle tear,** which is important to repair the esophageal wall completely

- **Trauma** (8% to 15.3%)

The MC cause is chest injury by a steering wheel in a traffic accident

The incidence of esophageal perforation by penetrating injuries is 11% to 17%

Perforation is more common in the cervical than thoracic esophagus

The overall mortality rate remains high (15% to 40%).

- Tumor
- Foreign Body (7-14%)
- Caustic Injury
- Drug Induced eg. tetracycline, KCL,quinidine, NSAID's
- Infection
- Other Causes eg.Barrett ulcer and ulcerative esophagitis with Zollinger-Ellison syndrome

Pathophysiology

- Air, Saliva, and Gastric contents released
  - mediastinitis
  - pneumomediastinum
  - empyema
  - can progress to sepsis, shock, resp failure

Diagnosis

Chest X ray

- Chest radiographs appear normal in the early phase
- Emphysema becomes manifestated by 1 hour after the perforation
- Pleural effusion is detected several hours after the perforation
- Pneumomediastinum is present in 60% of cases.

Perforation of the mid-thoracic esophagus is associated with right-sided pleural effusion and perforation of the distal thoracic esophagus is associated with left-sided pleural effusion

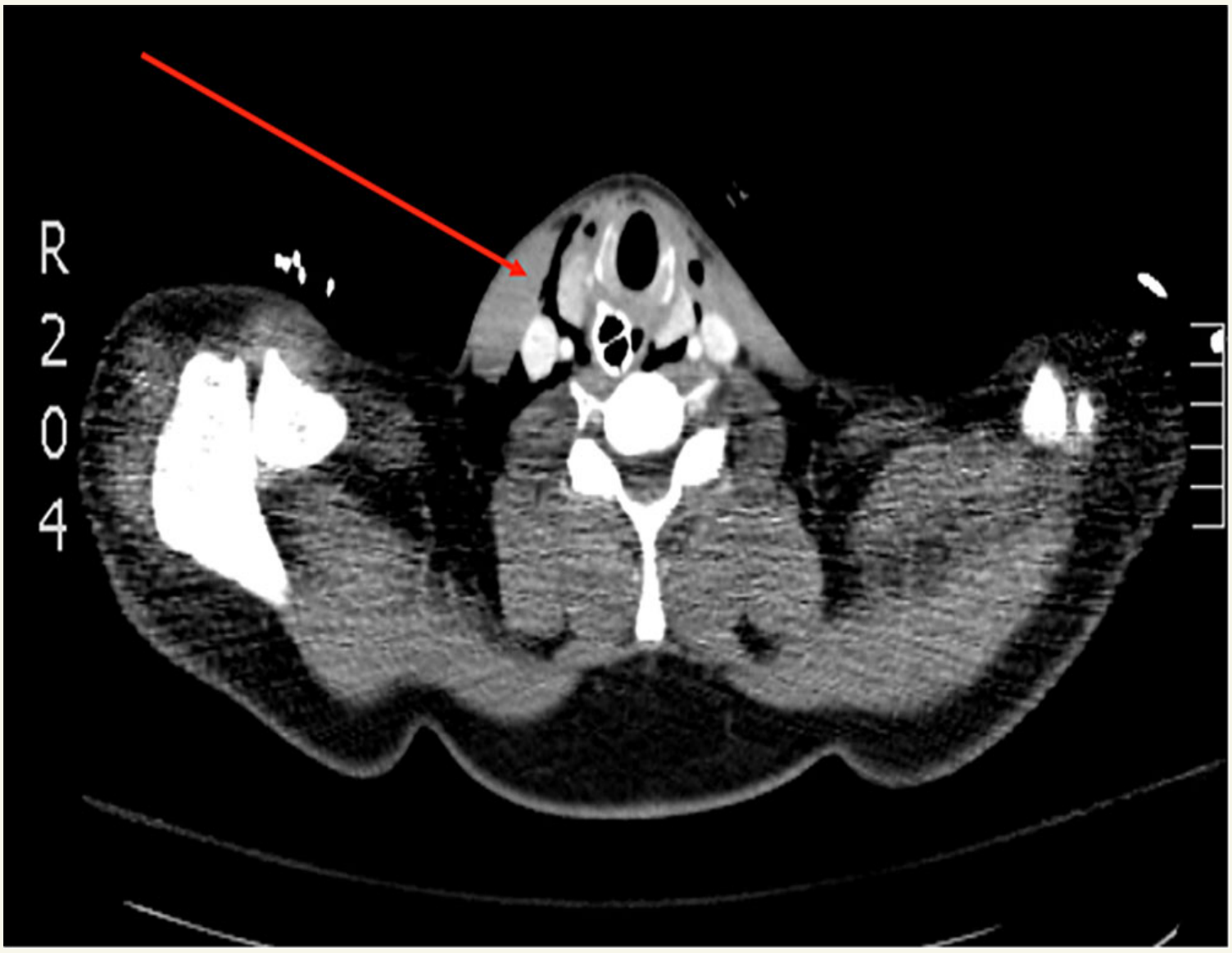
Esophagography

- The detection rate is 60% for cervical perforation and 90% for surgically confirmed perforations.

Computerized tomography (CT)

Endoscopy

Diagnostic thoracentesis



Treatment

The **goal** of treatment is to:

- Prevent further contamination
- Eliminate infection produced by contamination
- Restore the integrity and continuity of the GIT
- Restore and maintain adequate nutrition

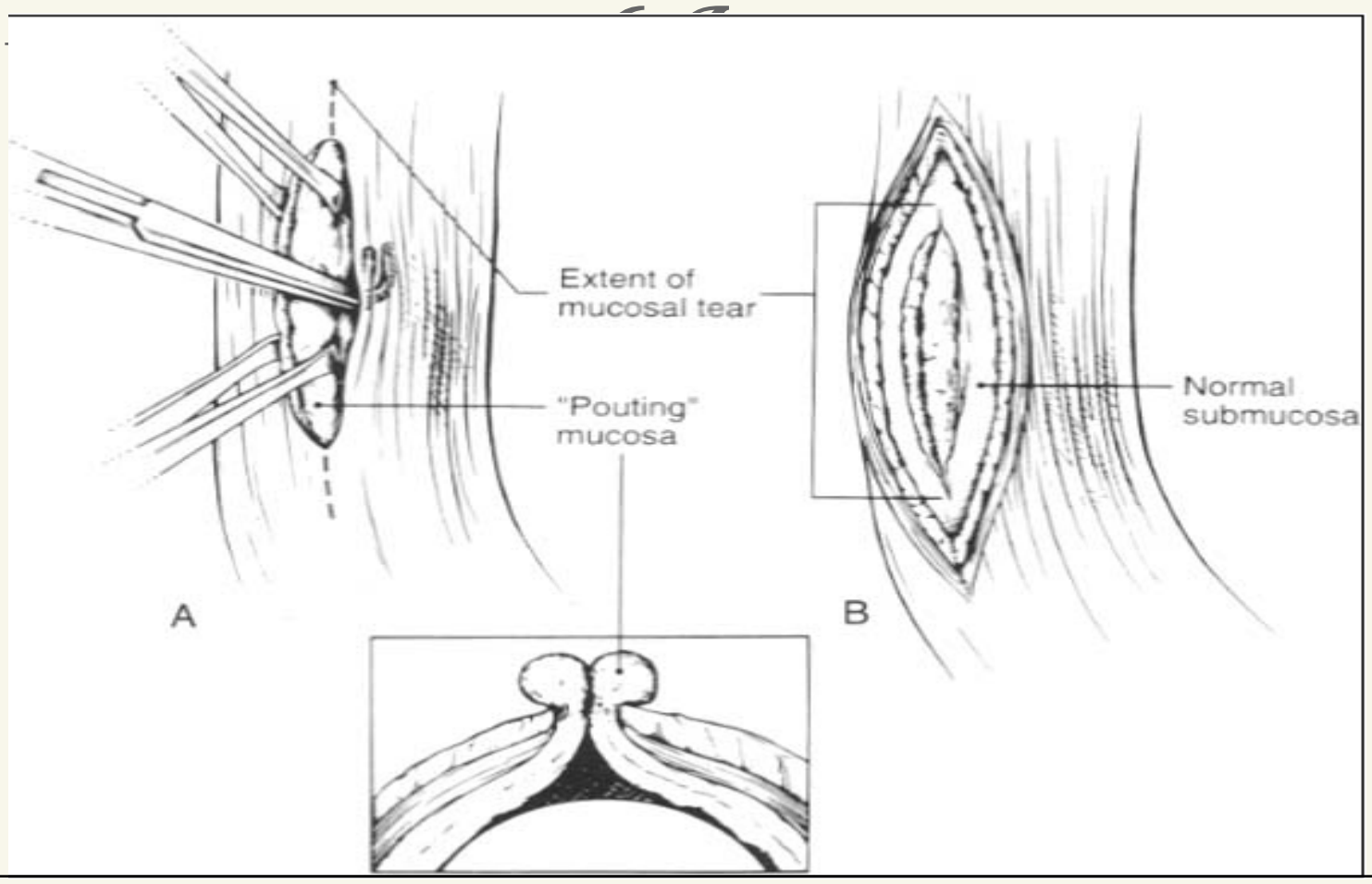
There are **two** major types of treatment

- Surgical
- Nonsurgical

Surgical treatment

- Primary closure
- Reinforced closure
- Resection
- Drainage alone
- T-tube drainage
- Exclusion and diversion
- Intraluminal stents

Primary repair of Esophagus



The principles of surgical treatment are :

- Debridement of all infected and necrotic tissue
- Secure closure of the perforation
- Correction or elimination of distal obstruction
- Drainage of contaminated and infected areas

An **enteral nutrition route, such as a jejunostomy**, should be added for nutritional support to any surgical method

Choice of Treatment

Surgical

Non Surgical

Patient selection according to strict criteria is necessary to make such comparisons

Indications for nonsurgical treatment are limited.

- Survival depends on rapid diagnosis and surgery
  - Within 24 hours of rupture: 70-75% survival
  - Within 25-48 hours: 35-50% survival
  - Beyond 48 hours: 10% survival

Conclusion

- Diagnosis & treatment of esophageal perforation remains a challenge to surgeons
- Early diagnosis and treatment are important to prevent morbidity and mortality
- Optimal treatment consists of complete repair with tissue reinforcement and elimination of distal obstruction
- Esophagectomy should be performed in patients with cancer or extensive necrosis of the esophagus
- Nonsurgical treatment may be used in carefully selected patients

Thank you