Historical Aspects:

- The clinical presentation, diagnosis, treatment for gastric vs. duodenal ulcers are different. The significant similarities in pathophysiology and medical management make their discussion under one heading possible.
- Both gastric and duodenal ulcers are being referred to as peptic, although most attention is focused on acid and H.P infection.
- Acid + pepsin is much more ulcerogenic than acid alone, thus the peptic label is still appropriate.
- Gastric acid was long considered the primary cause for ulcers, but it is now understood that acid alone will rarely cause ulcer disease (except Zollinger-Ellison).
- It is still true that ulcers will not occur in the absence of acid. The old dictum: “no acid, no ulcer” still stands.
Historical Aspects:

- Gastric ulcers are more common in presence of low acid, indicating defective mucosal defense.
- The most drastic change has been the recognition of the association between peptic ulcer disease and H.P. making some people conclude that ulcer disease is actually a infective disease.
- Before the discovery of H2-receptor antagonists various anti-acids were used, but had to be used at frequent intervals in order to be affective.
- In the 1980’s H2-RA became the drugs of choice because of better patient compliance and drug effectiveness.
- With the discovery of Omeprazole, which blocks H/K exchange we have a drug that gives complete inhibition of acid secretion.
Historical Aspects:

- A large number of controlled trials now show patients who receive effective eradication of H.P. as well as a PPI virtually never develop recurrent ulcers.
- Surgical therapy has undergone significant changes since the 1950’s. Surgery has evolved to less radical procedures e.g. selective vagotony.
Incidence:

- From the 1900’s to the 1970’s occurrence increased steadily. Since then the incidence, especially duodenal ulcers, has been declining in the U.S.
- Some of this is because of better diagnosis, allowing doctors to differentiate ulcers from other causes of dyspepsia.
- The tendency towards outpatient management of ulcer disease with less patients reflected in statistics may be a reason for declining incidence.
- Rates of hospitalization for ulcer hemorrhage fell only slightly for duodenal ulcers and increased for gastric ulcers.
- The rate of disease is decreasing in younger patients and increasing in older individuals.
- It must be noted that the decline in ulcer incidence occurred 10 years before the use of H2-RA.
Incidence:

• Ulcer incidence is about 1% per year in H.P. positive people, a rate that is 6-10x higher than non-infected individuals.
• NSAID use as well as HP infection rates increase with age. This may explain shifting trends in incidence.
• Another factor is the prevalence of smoking. Rates of smoking are declining in younger people, particularly men, possibly influencing the male to female ratio. Previously a predominant male disease, currently with nearly comparable gender ratio in latest studies.
Location and Type of Ulcer:

- Type 1: Primary gastric ulcer. Associated with diffuse antral gastritis.
- Type 2: Gastric ulcers with duodenal ulcers, most likely secondary to duodenal ulcers.
- Type 3: Prepyloric or channel ulcer.
- Type 4: Proximal stomach or gastric cardia.

Acid hyper secretion common among type 2 and 3 ulcers. Type 1 and 4 pathophysiologically the same.
Pathogenesis:

- With the exception of ZE ulcer disease should be regarded as a reduction in normal mucosal defense. Considering the aggressive nature of acid/pepsin environment, ulcer disease is surprisingly uncommon.
- Factors such as HP, NSAIDS disrupt these normal defense mechanisms. Smoking interferes with healing and secretory regulation.
Pathophysiology:

- The first line of defense is mucus and bicarbonate secretion. It stabilizes the pH between the lumen and the surface epithelial cells. Mucus gel in patients with HP infection was found to be structurally weak. Duodenal mucus as well as bicarbonate secretion is reduced in patients who smoke.
- The second line of defense is the intrinsic epithelial cell defense. The mucosal surface is a barrier to acid back diffusion thus maintaining normal intra cellular pH.
Pathogenesis:

- The third line of defense is the rich mucosal blood flow. The blood provides a buffer for acid neutralization as well as adequate nutrition for the metabolic demand to maintain mucosal integrity.
- Gastric mucosa has the ability to repair minor injury and therefore prevent progression to deep ulcers. Restitution has been evident within one hour.
Pathogenesis: HP infection.

- Described in humans in the first decade of the 20th century. Only in 1983 was it described in association with ulcer disease.
- HP’s natural habitat is the human stomach. Without treatment infection is lifelong.
- In developing countries most children are infected by the age of 10. In developed countries there is a clear age related increase.
- IT has not been proven why most patients with HP do not develop ulcer disease.
- HP resides in the stomach but causes duodenal ulcers probably by colonizing pockets of metaplastic gastric mucosa.
Pathogenesis: NSAIDS

- NSAIDs impair normal mucosal defense.
- 10-20% of patients will develop gastric ulcers and 4-10% duodenal ulcers within 3 months of taking NSAIDS. Not all endoscopic ulcers are clinically symptomatic and trials generally overstate the risk. Probably closer to 1% in the first three months.
- NSAID users develop gastric ulcers twice as common as duodenal ulcers. (HP more duodenal).
- NSAID ulcers not usually associated with gastritis as is the case with HP infection. When NSAID use is stopped these ulcers do not recur.
Pathogenesis: Acid

• Adequate acid necessary for duodenal ulcers.

• Remember “no acid, no ulcer” withstood the test of time. Acid is an important cofactor in the developing of both duodenal and gastric ulcers.
Clinical Presentation:

- Patients present with dyspepsia, epigastric pain and or discomfort. Acid may irritate nerve endings or peristaltic waves passing the ulcer may cause discomfort.

- But there is great overlap in symptoms with non ulcer dyspepsia. 20% of patients will present with serious complications without previous ulcer symptoms.

- It is said that gastric ulcers present with pain associated or closely followed by eating, whereas duodenal ulcer pain is relieved by food.
Clinical Presentation:

• These two pain processes are very non specific.
• Pain tend to be chronic and recurrent. The two can generally not be differentiated on clinical grounds alone.
• Generally gastric ulcers present from age 50-65, whereas duodenal ulcers present in the thirties.
• Other non specific symptoms are nausea, weight loss, heart burn fatty food intolerance and bloating.
• Melena alone more frequently associates duodenal ulcers. Gastric ulcers present with hematemesis or melena in equal frequency.
Clinical Presentation:

• Ulcers may also present with a perforation. This occurs in 5-10% of patients.
• Gastric outlet obstruction usually develops in the context chronic ulcer disease. Seen in <5% of patients.
Diagnosis:

• Gastroscopy detects 90% of duodenal ulcers and 95% of gastric ulcers. Endoscopy allows a tissue diagnosis to be made.

Upper Gastro-intestinal radiography.
• With double contrast 80-90% can be diagnosed. Certain features may suggest malignancy.