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## Etiology of peptic ulcer

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Stomach - Ulcers  
Daniel S. Egbert  
1931  
Etiology of Peptic  
Ulcer

THE ETIOLOGY OF PEPTIC ULCER.

Daniel S. Egbert.

THE ETIOLOGY OF PEPTIC ULCER.

NAME Harder, John HOSPITAL NUMBER 3703  
ADDRESS 1412 Olive St.  
NATIONALITY Am. COLOR W RELIGION Protestant.  
OCCUPATION Bank clerk.  
AGE 27 SEX M. MARITAL Married.

That was the beginning of John Harder's hospital record. It also came very near being the last of John Harder.

However, neither a hospital record nor his termination was being considered by this thin, rather pale and slightly built young man, as he sat at his desk late one morning last summer. In fact, there was not much of anything occupying his consideration, unless it was that old gnawing pain in the pit of his stomach, which was a surer sign than any clock that lunch time was near.

He reached for a letter at the far corner of his desk. This action seemed to make the pain suddenly worse. Instinctively he drew back, muttering something to himself.

A girl at a nearby desk noticed him as he started to get up. He was bent forward, his arms clasped to his stomach. He staggered a bit, and then, without ceremony, lay down on the floor. His face was distorted with pain and he could not suppress an agonized moaning.

A scream from the girl announced to the office that something was wrong. In immediate sequence, Dr Pierce received a telephone call from an excited voice, to come to the State Bank at once; someone had taken poison and was dying. It did not

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take the doctor long to arrive; experience had made him able, not by what, but by the way he was told, to sense the urgency of the situation. A glance at the patient's position, motionless on the floor, and at the agonized expression gave him his clue. A few brief questions and a hasty examination of the patient's abdomen, which was hard as a board, practically confirmed the diagnosis.

About an hour later, at the hospital, the author stepped into the operating room to watch the emergency laparotomy for perforated gastric ulcer. The abdomen had been opened and the surgeon had just discovered the perforation.

"Here it is! On the posterior wall. Come here! See it?"

It was the first I had ever seen, but one could not hope for a more classical picture; a hole one-third as large as a dime and perfectly round as if it had been made with a punch. A white, watery, somewhat frothy material oozed from the opening. The remainder of the stomach was apparently intact and practically normal in appearance, so far as could be told.

It was at that moment, the idea came to me that peptic ulcer is due to some disturbance in a single gland. And to prove that, by induction or deduction, is the purpose of this thesis.

In the first place, to give the single gland theory a more logical basis, it is necessary to determine whether in all acute perforations the remainder of the stomach is changed or is as usual.

H. E. Eggers, professor of pathology and bacteriology, University of Nebraska, College of Medicine, states that a peptic ulcer is a localized lesion in which the rest of the gastric wall is essentially normal; he compares the condition to a tuberculosis of the intestine, in which the intestinal wall seems to be normal except for the tubercles.

J. A. Weinberg, associate professor of clinical pathology, University of Nebraska, College of Medicine, confirms the opinion that in acute perforation

of a peptic ulcer, the surrounding tissue seems to be practically normal. He believes that at some stage in the development of ulcer, there may be some sort of a general gastric involvement, such as an edema, although he has never seen such a condition. In at least five stomachs which he has had the opportunity to examine following perforation, he has found no apparent change in the remainder of the gastric wall. Both Eggers and Weinberg incline to a vascular or lymphatic obstruction theory.

Delafield and Prudden (1) state: "There is usually but little active inflammatory change about the ulcer though the edges are occasionally thickened by a growth of connective tissue. Moderate cellular infiltration may be seen near the eroded surface. The mucous membrane of the remainder of the stomach is apt to be in a condition of catarrhal inflammation."

At least, the opinions of the above authorities do not seriously conflict with the proposed theory of single gland involvement. Hence it is safe and reasonable to proceed and to inquire into prevalent theories and experiments related to the question at hand. Literature on the subject is not scarce. With the exception of the cure for cancer, it seems to be one of the most popular quests of the modern alchemist.

There is nothing like the following list of articles on peptic ulcer to give a comprehension of the present situation.

(from Rehfuss (2) )

First to demonstrate bacteria in ulcer. Böttcher: *Dorpater Medicinische zeitschrift*, 1874, Bd. v. S. 148.

Pneumococcic infection. Dieulafoy: *Clinique Médicale de l'Hôtel Dieu de Paris*, 1893 - 1899, tome iii, p.219

Staphylococcus. Widal et Meslay: Bull. et Mém. de la Soc. Méd. des Hôp. de Paris, Mars 12, 1897, 3e série, t XIV, p. 379.

Bacteria through the lymphatics. Letulle: Bull. et Mém. de la Soc. Méd. des Hôp. de Paris, Août 10, 1888, p. 380.

Pus injected into vessels produced ulcer. Lebert: Traite d'Anat. Path., 1857, vol. i, p. 537.

staphylococcus pyogenes aureus. Letulle: Bull. et Mém. de la Soc. Méd. des Hôp. de Paris, Aout, 10, 1888, p. 380.

Bacillus pyocyaneus, injection filtered cultures in rabbits. Charrin et Ruffer: Bull. de la Soc. Anat. de Paris, 5e série, t. iii, p. 18.

Dysentery organisms, injection. Chantemesse et Widal: Bull. de l'Acad. de Méd. de Paris, 1888, 3e série, t. XIX, p. 522.

Lactic acid bacillus, injection. Wurtz et Leudet: Arch. de Méd. Expér. et d'Anat. Path., 1r série, t. iii, p. 485.

Diphtheria toxin, dogs, and guinea-pigs. Enriquez et Hallion: Compt. Rend. de la Soc. de Biolog., December 23, 1893, p. 1025, 9e série, t. v.

On horses. Rosenau and Anderson: Jour. Infect. Dis., January, 1907, No. 4, p. 1.

Pneumococcic septicemia. Bezançon et Griffon: Bull et Mém. de la Soc. Anat. de Paris, 6e série, t. i, p. 409.

Bacillus of Pfeiffer. Slatineano: Bull. et Mém. de la Soc. Anat. de Parle, 6e série, t. iii, p. 693.

Meningococcus of Weichselbaum. Gandy et Griffon: Bull. et Mém. de la Soc. Anat. de Paris. 6e serie, t. iii, 1901, p. 373.

Typhoid bacillus and Bacillus coli. Rodet et Zaidmann: Compt. Rend. de la Soc. de Biolog. de Paris, 10e série, 1900, p. 1007.

Feeding of colon bacillus. Turck: Brit. Med Jour., 1907, vol. i, p. 922.

Streptococcus. Rosenow: Jour. Infect. Dis., 19, 333, September 1916.

Streptococci and focal infection. Nahamura: Ann. Surg., 79, 29, January, 1924. Haden and Bohan, Jour. Amer. Med. Assoc., 84, February 7, 1925.

Infection from the cavity of the stomach. Martin: Diseases of the Stomach, 1895, p. 419.

Streptococcic infection, elective localization. Rosenow: Jour. Infect. Dis., January, 1907, No. 4, p. 1.

Tonsillar infection. Haden and Bohan. (Loc. cit.)

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Cutaneous burns experiment. Silbermann: Virch. Archiv. 1890, CXIX, S. 488.

Vassale et Sacchi: La Riforma Medica, Anno IX, 1893, vol. IV, p. 544.

Parascandolo: Archiv. de Physiologie Normale et Path., 5e série, t. X., p. 714.

Socoloff (Skin varnish experiment). Centralb. für de medicin. Wissenschaften, October 19, 1872, No 44, p. 689.

Gastrotoxic sera. Bolton: Ulcer of Stomach, London, 1913.

Heloderma venom. Rehfuss: University of Pennsylvania Medical Bulletin, t. XXII, No. 4, June, 1909 pp. 105-109.

#### EXTRINSIC POISONS.

Gray ointment (rubbing in). Overbach: Mercur. und Syphilis, Berlin, 1861.

Glycocholate of Hg. Von Mering: Archiv. für Exper. Path. und Pharmak., 1881, Bd. XIII, S. 86.

Perchlorid of Hg. Pilliet: Bull. de la Soc. Anat. de Paris, December 29, 1893, 5e série, t. VII, p. 711.

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Arsenious acid by skin. Bohm et Unterberger: Archiv. für Exper. Path. und Pharmak., 1874, Bd. ii, S. 89.

Cantharadine injection. Aufrecht: Centralb. für die Medic. Wissenschaften, August 5, 1882, No. 31, p. 545.

Vegetable alkaloids. Pouchet: Bull. Gén. de Thérapeutique, Juin 23, 1899, p. 901.



B-tetrahydronaphthylamin. Elliot(referred to in preceding article)

Pilocarpin, atropin, paraldehyd, chloroformphenol, sodii fluorid, magnesium sulphate, and copper sulphate. Rehfuss. (Loc. Cit.)

#### ASEPTIC VASCULAR OCCLUSION.

Ulcer in arteriosclerosis. Ophuls: Arch. Int. Med., ii, May 1913.

Vascular block. Müller: Das Corrosive Geschwür im Magen und Darmkanal und dessen Behandlung, Erlangen, 1860.

Thrombus or infarct. Virchow: Virch. Archiv., 1853, Bd. v, S 362.

Vascular block. Merkel: Wien. med. Presse, 1866, No. 30, p. 755.

Case multiple ulcer from septic thrombus (celiac). VonRecklinghausen: Handb. der Allg. Path. d. Kreislaufs and der Ernährung., 1883, p. 350.

#### EXAMPLES OF BLOCKAGE OF GASTRIC VESSELS WITH AND WITHOUT ULCER.

Thrombosis, gastro-epiploic. Virchow: Virch. Archiv, 1853, Bd. v, S. 362.

Embolism, inferior mesenteric. Gerhardt: Würzburger Medicin. Zeitschrift, 1863, Bd. iv, S. 144.

Blockage, celiac. Ponfich: Virch. Archiv, 1870, Bd. 1, S. 628.

Merkel's idea in phthisis is a secondary amyloid degeneration of vessels accounting for ulcer. Wien. Med. Presse, 1869. No. 39, S. 913.

Compression gastric vessels by tumor. Netter: Bull. et Mem. de la Soc. Med. des Hop. de Paris, Juill. 6, 1894, 3e serie, t. xi, p. 504.

Obliteration gastric vessels unable to produce ulcer. Hauser: Das Chronische Magengeschwür, 1883, Leipzig, p. 30.

Injection of wax into femoral vessels, multiple ulcer. Panum: Virch. Archiv, 1862, Bd. XXV, S. 488.

Injection chromale lead gast. braches splenic a. Cohnheim: Lectures on General Pathology (New Sydenham Society), 1890, vol. iii, p. 878.

Fat embolism gave rise to ulcer. Schridde: Verhandlungen der Deutschen Pathol. Ges. Kiel., xi, 1907, 234.

Emboli of blood-corpuscles and pigment granules. Klebs and Welti: Handbuch der Path. Anat., 1869.

Ligating larger vessels does not give rise to ulcer. Cohn, Müller, Pavy, Roth, Litten, and others. (Bolton's Ulcer of the Stomach, p. 78.)

Litten (Virch. Archiv, 1875, Bd. lxiii, S. 289) found he could cut one-third of the stomach from its vascular supply and yet the nutrition was not interfered with.

Pressure atrophy from the costal margin. Rasmussen: Centralb. für die Medic. Wissenschaften, 1887, March 5, No. 10, p. 162.

Mechanical injury can, of course, induce changes.

Trophic disturbance of the gastric nerves, fanciful and purely theoretical. Siebert, Billes de la Tourette, Stockton-Bolton.)

Such a list depicts more clearly than lengthy exposition, the work that has been done, yet the uncertain status of the subject at the present time. It also represents the tremendous amount of time, effort and sacrifice which is expended in the investigation of a problem. Medicine progresses more slowly and embodies much more labor than most people realize.

The above names are just as worthy of commemoration as the names of soldiers on a World War honor roll, but it seems to be a peculiar characteristic of human beings, which probably has sound basis, to admire a skillful killer rather than to venerate a healer. At any rate, the above list is an honor roll, for the names are those of truly courageous warriors, because they have been engaged in a strenuous battle against a formidable foe of humanity. In comparison, we writers of senior thesis are given a sort of amateur standing like the R.O.T.C., in this great struggle against peptic ulcer. When an individual finishes R.O.T.C. at college, he either hopes to be through with war forever or considers himself to be somewhat of an authority on the subject.

Medical research associated with noteworthy exposition,

seems to outrank the ordinary practice of medicine. It is usually an ascent away from, rather than into a more efficacious practice. Hence the fallacy of expecting a senior thesis to be more than a review of the subject which it concerns. This review, mostly copied, is as follows:

The distribution of peptic ulcers indicates that they are due to a digestion of the tissues by gastric juice. Why, then, does the gastric juice normally not digest the stomach wall? Not only is such digestion very rare, but even when lesions of the mucous membrane have been produced by various means, these usually heal with great rapidity so that the gastric wall is normal at the end of two to three weeks. Some mechanism exists in the gastric mucous membrane which causes it to resist peptic digestion.

John Hunter believed that the resistance to digestion was a general property of all living tissue. This varies considerably in different tissues. The leg of a living frog and the ear of a living rabbit are digested by gastric juice. Living gastric wall even when denuded of the mucous membrane or of the mucous membrane and the muscular coats offers a considerable resistance to gastric digestion. The same is true of portions of the intestine and the living spleen. To resist digestion it is necessary for the blood supply to be maintained in these structures.

What is the mechanism of this resistance? So far as the gastric mucous membrane is concerned there is a certain amount of protection by mucous. In patients with gastric ulcer, the

amount of mucus recovered from the stomach may be less than normal.

Weinland showed that intestinal parasites resist digestion because they contain an antiferment which neutralizes the ferments to which they are exposed. Similar antipeptic and antitryptic substances have been isolated from the mucous membrane of the stomach and upper intestine and from the mucus covering the surface.

Katzenstein (3) has attempted to show that the development of peptic ulcer is due in part, to a disturbance of the relationship which should exist between the ferments and antiferments of the stomach and duodenum. Kawamura found antipepsin in all living tissues. Kohler (4) compared the antipeptic activity of the blood of ulcer patients with the activity of their own juice and found that the second far exceeded the first; while in those free from ulcer, the first exceeded the second. Making use of this method, he succeeded in making a correct diagnosis in practically every case. Wolff (5) found no decrease of antipepsin in the stomach of the ulcer patients. Orator (6) was unable to verify Kohler's findings. Gunzburg (7) found an increase in peptic activity in the stomach contents of ulcer patients, while those free from ulcer were normal. Levine concludes from a survey of these experiments that there is an antipepsin present in the stomach preventing it from digesting itself.

Infection is probably one of the most important factors. Not only is it true that ulcer of the stomach can be produced

by a variety of bacteria, but these bacteria can produce ulcer in a variety of ways, such as by entrance through intravenous, subcutaneous or intraperitoneal injection or by ingestion.

In 1913, Rosenow (8) stated that ulcers of the stomach and duodenum were produced by the intravenous injection of streptococci in rabbits, dogs and monkeys. Strains were obtained from cases of rheumatism and most of these were acute. In 1916, he reported the experimental production of ulcer and hemorrhage in sixty percent of onehundred three animals injected with eighteen strains of streptococci from human gastric ulcers. In 1921, sixty-five animals were reported injected with cultures from tonsils, six with organisms in pus from tonsils, seven with streptococci from three excised ulcers, and twenty-three from infected teeth. The incidence of lesions was about eighty percent in each group. From human ulcers, thirty-seven strains of streptococci were isolated and injected into one hundred sixty-eight animals and in sixty-eight of these, there were lesions of the stomach and duodenum. In 1923, he reported that ulcer and hemorrhage was produced in eighty-six percent of rabbits and dogs when they were injected with freshly isolated cultures. He maintained that these results were not obtained with streptococci from sources other than ulcer. Following is a table by Rosnow summarizing the results of some of his experiments.

Further Results on Elective Localization of Streptococci.

Source of Streptococci.	Percentage of animals showing lesions in														
	Strains	Animals	Muscles	Joints and ligaments	Nerves	Stomach and duodenum.	Gall-bladder.	Appendix.	Kidneys	Lungs	Skin	Pericardium	Myocardium	Endocardium	Central nervous system.
Appendicitis	17	71	12	29	0	11	1	70	0	0	0	0	9	21	?
Ulcer of the stomach.	37	168	4	12	0	68	21	1	3	0	0	2	3	10	?
Cholecystitis	12	41	7	17	0	29	80	0	5	5	2	0	2	10	?
Myositis	28	202	80	33	9	13	3	1	9	7	4	4	17	8	?
Neuritis and sciatica.	10	59	42	15	66	5	2	3	12	15	0	5	10	3	14
Miscellaneous	71	212	12	9	4	9	1	1	9	7	4	0	4	12	?

Experimental confirmation of Rosenow's work was made by Nakamura and more recently by Haden and Bohan (9) and by Scoll Loria and Haden (10). Several clinicians have attempted to use vaccines in the treatment of ulcer. Rehfuss has used autogenous vaccines from the nose, throat, teeth and duodenum.

Rosenow's theory may be summed up in that foci of infection harboring streptococci, having elective localizing power, are important factors in explaining either the cause or the persistence of ulcers of the stomach.

Furthermore, bacterial toxins, endogenous poisons, a variety of chemical non-bacterial and even non-serological poisons, drugs like pilocarpin and atropin; vasodilators such as chloroform; poisons such as copper sulphate and phenol, sodium chloride; and even injections of strong dilute

hydrochloric acid all produced acute ulceration with hemorrhagic erosion and even perforation of the gastric mucous membrane, with the latter.

On an anatomical basis, the mode of origin of ulcers is often described as being due to an interference with the blood supply, since the ulcers most commonly occur where the blood supply is supposed to be least. This is attributed to embolism, thrombosis or spasm of the arteries. As they are not end vessels, simple obstruction cannot account for it.

Another anatomical factor is the "Magenstrasse". In German literature, reference is continually made to the importance of this portion of the stomach in the causation of ulcer. Ninety-five percent of all gastric ulcers affect this region of the stomach wall. The musculature of the stomach consists of three layers: outer longitudinal, middle circular, and inner oblique. In the human, the oblique fibers surround the cardia like a girdle and run along the anterior and posterior surfaces up to the pyloric canal where they become a part of the circular fibers. They do not cover the lesser curvature and parts of the anterior and posterior wall adjacent to it. Thus a free space is formed above the oblique fibers which is transformed into a groove, when they contract, this being called the "Magenstrasse". Thus, it is not synonymous with the lesser curvature. The longitudinal fibers which run along its internal surface are tightly stretched even during peristalsis. Here the mucous membrane is firmly attached to the underlying tissue while that of the fundus is freely movable. Injury to the



"Magenstrasse does not result in the production of mucin or a contraction of the muscularis mucosa, and epithelialization, if it occurs at all, is tardy.

The causes of ulcer, in summary, are:

1. The initial stage of ulcer formation is a local damage or devitalization of the gastric cell. This process is probably in no way specific, in as much as a variety of substances, totally different in their physiological effects, can all produce devitalization. Bacteria of many varieties of which streptococci, colon bacilli and numbers of others of the pathogenic group, seem most prominent; metabolic toxins as a result of disturbances in the endocrine glands (supra-renal, etc.); circulating cytotoxins; circulating poisons from vitiated intestinal, renal, hepatic structures and other sources, as well as injected toxins, can all produce, under certain conditions, this devitalization. That this may occur through injection or through the circulation seems probable from experimental evidence.

2. This primary damage is followed by secondary digestion and usually hemorrhage of the mucosa.

3. Necrosis is not necessary to the destruction of the mucous membrane; it is only necessary that the gastric cells be damaged. (Bolton)

4. Gastric secretion per se does not produce ulcer, but in the presence of the above factors, it is capable of causing extension and persistence of the lesion.

5. Any protoplasmic poison in itself too weak to induce a lesion can do so in the presence of blood poison or altered gastric states. (Bolton)

6. The gross effect of cell damage with toxemia and gastric secretion is an ulceration probably chronic.

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