Eponyms in Radiology of the Digestive Tract: Historical Perspectives and Imaging Appearances

Part I. Pharynx, Esophagus, Stomach, and Intestine

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Eponyms serve as a means of honoring individuals who have made important discoveries and observations. Eponyms are frequently encountered in the field of radiology, particularly in radiology of the digestive tract. However, the use of eponyms may fail to convey a precise meaning or definition and could lead to miscommunication. Moreover, in some instances, more than one individual may have contributed to the discovery or description of a particular anatomic structure or disease, whereas in others, an eponym may have been incorrectly applied initially and propagated for years in the medical literature. Nevertheless, radiologic eponyms are a means of honoring those who have made lasting contributions to the field of radiology, and familiarity with these eponyms is important for proper reporting and accurate communication. In addition, the acquisition of some historical knowledge about those whose names are associated with various structures or pathologic conditions brings some humanity back into the science of medicine.

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Introduction

In the context of medicine, an eponym is defined as “a name of a drug, structure, or disease based on or derived from the name of a person” (1). Eponyms are frequently encountered in radiology, particularly that of the digestive tract, and knowledge of these terms is important for proper reporting and communication. Eponyms are a means of honoring individuals who have made contributions to medicine, but use of these terms may fail to convey a precise meaning or definition and could lead to miscommunication. Furthermore, it may be that more than one individual contributed to the discovery or description of a structure or disease. In other cases, an eponym may have been incorrectly applied initially and propagated for years in the medical literature.

In this article, the first of a two-part series, we discuss and illustrate the imaging manifestations of eponyms encountered in radiology of the pharynx (Zenker diverticulum), esophagus (Mallory-Weiss tear, Boerhaave syndrome, Schatzki ring, Barrett esophagus), stomach (Zollinger-Ellison syndrome, Ménetrier disease, Carman meniscus sign), small bowel (Meckel diverticulum), and colon (Hirschsprung disease, crypts of Lieberkühn, Escherichia coli colitis). We also explore the historical background of the individuals for whom these eponyms were named.

Pharynx

Zenker Diverticulum

Zenker diverticulum (Fig 1) is a pulsion pseudodiverticulum that occurs posteriorly in the midline just proximal to the cricopharyngeus (2). It results from herniation of mucosa and submucosa through the dehiscence of Killian, a focal...
weakness in the hypopharynx at the normal cleavage plane between the fibers of the inferior pharyngeal constrictor and the cricopharyngeus. This phenomenon may lead to the creation of a sac with a narrow neck that can trap food and liquid, leading to dysphagia, halitosis, and regurgitation.

Friedrich Albert von Zenker (1825–1898) (Fig 2) was born in Dresden, Germany (the distinguished “von” in his name was added later when he was admitted to the German nobility in recognition of his accomplishments). He studied medicine at Leipzig and Heidelberg and then became the assistant to Professor Karl Freiherr von Rokitansky in Vienna. Von Zenker then became Professor of Pathology at Erlangen University and served as dean of the medical faculty and prosector there. His monograph entitled Krankheiten des Oesophagus was published in 1867 in collaboration with his clinical colleague Hugo Wilhelm von Ziemmsen, citing all previous reports of hypopharyngeal pulsion diverticula. Von Zenker was also known by his contemporaries for his discovery of the pathogenesis of trichinosis and his extensive work on pneumoconiosis (3).

Esophagus

Mallory-Weiss Tear

Mallory-Weiss tear (Fig 3) is a partial-thickness tear of the esophagus that involves only the mucosa and often extends into the gastric cardia (4). It occurs in the distal esophagus and is usually the result of violent retching. Mallory-Weiss tear is difficult to diagnose with esophagography, but when identified, it manifests as a 1–4-cm longitudinal collection of barium in the distal esophagus.

George Kenneth Mallory (1900–1986) (Fig 4) was born in Boston and received his medical degree from Harvard Medical School in 1926. Like his father, Frank Burr Mallory (1862–1941), for whom Mallory bodies are named, George Mallory was also a pathologist (5). He became Professor of Pathology at Boston University and worked for an extended period at the Mallory Institute of Pathology, the institution founded by his father. His primary interest was in disease of the kidneys and liver (6).
Soma Weiss (1898–1942) (Fig 5) was born in Bestereze, Hungary, and emigrated to the United States in 1920 during the chaos that engulfed Europe following World War I. He received his medical degree from Cornell Medical College in 1923 and eventually became Physician-in-Chief at Peter Bent Brigham Hospital in Boston (5). Weiss’s most significant contribution to medicine was his work in cardiovascular disease, particularly his focus on unusual causes and manifestations of heart failure and the importance of the nervous system in cardiovascular physiology. He had a reputation for being an outstanding bedside teacher and diagnostician, and word of his famous Tuesday night rounds at Boston City Hospital quickly spread throughout the United States and the world (7). Weiss died in 1942 from a ruptured cerebral aneurysm that he himself diagnosed on his deathbed (5).

Boerhaave Syndrome

Boerhaave syndrome describes rupture of the esophagus resulting from violent emesis (8). The tear almost always occurs in the left posterior wall near the left diaphragmatic crus, and esophageal contents can spill into the left pleural space or dissect between the parietal pleura and the left crus. Chest radiographic findings include pneumomediastinum, abnormal mediastinal contours, and hydropneumothorax.

Hermann Boerhaave (1668–1738) (Fig 6) was born in Voorhout, the Netherlands. His father, a preacher, educated him at home, teaching him Greek, Latin, and other languages, and encouraged his son to follow in his path. Boerhaave’s future in the clergy came to an abrupt halt when he was traveling by boat with a group of passengers who were discussing the doctrines of Spinoza, a man accused of heresy. Boerhaave asked one particular denouncer if he had actually read Spinoza, effectively silencing the critic. However, he was quickly labeled as one who shared Spinoza’s atheistic views.

Fortunately for Boerhaave, he had already been drawn to mathematics and the sciences during his studies in theology, and the logical choice for him was to turn to medicine. His great intellect allowed him to learn everything on his own, attending only dissections. He rose through the ranks at Leiden University, becoming Professor of Clinical Medicine, Botany, and Chemistry, and ultimately serving as president of the university.

Boerhaave syndrome was so named on the strength of Boerhaave’s detailed description of the ruptured esophagus of the ill-fated Baron Johannes von Wassenaar, the Grand Admiral of the Dutch fleet, who had the reputation of being a rather sedentary gourmand. Reportedly, the Baron dined on a rather large meal and, several hours later, because of dyspepsia, took several doses of an emetic. Subsequent retching caused a tearing chest pain, which the Baron described as
“something having broken or torn.” Despite undergoing extensive treatment by Boerhaave, the Baron died within 24 hours.

Upon opening the Baron’s chest at autopsy, Boerhaave discovered subcutaneous emphysema, a large volume of intraabdominal air, and the rancid odor of the Baron’s last meal. Both lungs were collapsed, and the pleural spaces were filled with large volumes of liquid. A finger-sized tear was identified on the left side of the esophagus, and Boerhaave concluded that the tear was the result of the violent retching resulting from the Baron’s ingestion of the emetic. Boerhaave published a detailed account of this disease, deeming it to be entirely incurable (9).

However, Boerhaave’s greatest contributions to medicine were as a clinician and teacher. Leiden had become the most outstanding medical school in Europe, attracting young men from all over the Continent. Boerhaave advocated bedside teaching, setting aside 12 dedicated clinical teaching beds at St Caecilia Gasthuis, to which he brought his students for daily teaching exercises. His method of clinical evaluation is not unlike our modern medical history and physical examination.

Boerhaave suffered from severe gout and, despite his medical expertise, was unable to successfully treat the debilitating pain or shorten the course of the disease. He died in 1738 from complications of a lung abscess. However, he died a wealthy man, reportedly leaving millions of guilders to his daughters (10).

Schatzki Ring
A Schatzki ring (Fig 7) is a symptomatic mucosal fold found at the squamocolumnar junction in the distal esophagus (11). The primary symptoms are dysphagia or pain associated with food impaction and generally develop when the diameter of the ring narrows to 11 mm (12).

Richard Schatzki (1901–1992) (Fig 8) was born in Clafeld, Germany, and received his medical training at the University of Berlin. He studied radiology under Hans Heinrich Berg (1889–1968), the leading diagnostic radiologist in Germany at that time, since he could not decide between a career in surgery or internal medicine. He was appointed Chief of the Radiology Department at the University Hospital in Leipzig in 1929 and studied radiology of esophageal and gastric varices. In 1933, Schatzki spent some time at Massachusetts General Hospital as an assistant in the radiology department. Following World War II, he was named Chief of the Radiology Department at Mount Auburn Hospital in Cambridge, Massachusetts. Schatzki held the position of Associate Clinical Professor of Radiology at Harvard Medical School and served as

Figures 7, 8.
(7) Schatzki ring. Air-contrast esophagogram shows concentric narrowing just above the esophagogastric junction. (8) Richard Schatzki (1901–1992). (Courtesy of Stefan Schatzki, MD, Department of Radiology, Mount Auburn Hospital, Cambridge, Mass.)
President of the New England Roentgen Ray Society. In addition to being a radiologist, Schatzki was an accomplished pianist and gave various benefit concerts in the Boston area (6).

Barrett Esophagus
Barrett esophagus (Fig 9) is defined as intestinal metaplasia of the esophageal mucosa, thought to arise from chronic reflux esophagitis. Traditionally, Barrett esophagus has been considered to be a risk factor for developing esophageal adenocarcinoma. However, recent years have seen a growing debate among gastroenterologists and pathologists as to the true cause and significance of the metaplasia (13).

Imaging findings that suggest Barrett esophagus include a benign-appearing esophageal stricture, particularly when the stricture occurs in the more proximal esophagus. Small, plaque-like mucosal irregularities may also be identified near a stricture at high-quality double-contrast esophagography (14).

Norman Rupert Barrett (1903–1979) (Fig 10), nicknamed “Pasty,” was a British surgeon born in Adelaide, Australia. He emigrated to England at 10 years of age and received his undergraduate education at Cambridge University. He served as editor of *Thorax* from 1946 to 1971 and as president of several surgical societies. Barrett spent most of his career at St Thomas Hospital, where he established one of the first thoracic surgery and intensive care units (6).

In 1950, Barrett published a report in which he defined the esophagus as “that part of the foregut, distal to the cricopharyngeal sphincter, which is lined by squamous epithelium” (15). Ironically, an esophagus lined extensively by columnar epithelium is referred to as Barrett esophagus. However, Barrett was not the first to describe this columnar epithelial lining of the esophagus. In his 1950 treatise, Barrett supported the view that the ulcerated, columnar epithelium–lined organ was a tubular segment of stomach that was tethered in the chest by a congenitally short esophagus. In 1953, Allison and Johnstone argued that this columnar epithelium–lined structure was indeed the esophagus and suggested that the ulcerations in this structure be called “Barrett’s ulcers.” Not until 7 years after his initial treatise did Barrett accept that this columnar epithelium–lined structure was the esophagus, suggesting that it be called the “lower esophagus lined by columnar epithelium.” Despite earlier reports, Barrett failed to mention that intestinal-type epithelium could be present (16).

Stomach
Zollinger-Ellison Syndrome
Zollinger-Ellison syndrome (Fig 11) (17) is caused by a gastrin-secreting islet cell neoplasm (gastrinoma) that stimulates acid hypersecretion by parietal cells in the gastric fundus and upper body. These two sites subsequently become grossly hyperplastic, a phenomenon that accounts for much of the fold thickening, to which inflammation adds more distally. Approximately 60% of
Gastrinomas are malignant, and these malignant neoplasms can be associated with multiple endocrine neoplasia syndrome type I. Gastrinomas most commonly occur in the pancreas, with the duodenum and other sites being less commonly affected. The continuous secretion of gastrin leads to increased gastric acid production, resulting in severe peptic ulcer disease. Characteristic radiographic findings include multiple gastric and duodenal ulcers, with distal duodenal ulcers being highly suggestive of the disease, as well as gastric and duodenal fold thickening. Hypersecretion can also lead to barium dilution with poor coating.

Robert Milton Zollinger (1903–1992) (Fig 12) was an American surgeon born on a farm in Milledgeville, Ohio. He joined the army in 1941 and became Assistant Chief of the Surgical Service for his unit. Zollinger served as Chairman of the Department of Surgery at Ohio State University from 1947 to 1974 and as Editor-in-Chief of the American Journal of Surgery from 1958 to 1986. He had a reputation for using “vigorous” and rather unconventional teaching methods. His gentler side was characterized by his skills as a champion grower of gourds and his role as a national rose judge (18,19).

Edwin H. Ellison (1918–1970) (Fig 13) was an American surgeon born in Dayton, Ohio, who
served as Chairman of the Department of Surgery at Marquette University. He was a prolific writer and a member of the editorial board of numerous journals. In 1955, he and Zollinger described the syndrome that now bears their names.

**Ménétrier Disease**

Ménétrier disease (Fig 14), also referred to as giant hypertrophic gastropathy, is a rare disease characterized by marked gastric mucosal fold thickening, hypersecretion, hypochlorhydria, and hypoproteinemia (20). Rugal thickening occurs primarily in the gastric fundus and body, often with antral sparing (21). At palpation, the folds remain pliable.

Pierre-Eugene Ménétrier (1859–1935) (Fig 15) was born in Paris. Although his father, a literary critic, inspired in his son a lifelong interest in history and literature, Ménétrier opted to pursue a career in medicine, enrolling at the medical school of the University of Paris. He became a pathologist, was promptly promoted to professor, and was recognized as a talented investigator. He was among the first to recognize the transformation from benign to malignant neoplasia, implied in his description of the exuberant gastric mucosal hyperplasia now bearing his name. Although Ménétrier recognized the debility of patients afflicted with this disease, the associated protein-losing gastroenteropathy was not recognized until later. His other works include writings on Byzantine and Greco-Roman medicine. Ménétrier died from injuries sustained in an automobile accident in Limieux, France, in 1935 (22).

**Carman Meniscus Sign**

The Carman meniscus sign (Fig 16) is created by a large, flat ulcer with heaped-up edges. The edges of the ulcer trap a lenticular barium collection that is convex relative to the lumen when the edges are folded upon themselves during compression. These findings are indicative of a malignant gastric ulcer (23).

Russell Daniel Carman (1875–1926) (Fig 17) was born in Iroquois, Ontario, Canada, and, as a teenager, emigrated first to Butte, Montana, and later to St Paul, Minnesota, with his mother. He studied medicine, first at the University of Minnesota and then at Marion Sims College of Medicine in St Louis, from where he received his MD degree in 1901. He studied under William Osler at Johns Hopkins Medical School from 1901 to
1902 and then returned to St Louis, where he worked as a general practitioner.

Carman acquired an x-ray machine early in his career and, through detailed observation and diligent work, became a recognized expert in x-ray diagnosis in the St Louis area. Prior to being named head of the Mayo Section on Roentgenology in 1913, he was appointed Professor of Roentgenology at the medical schools of St Louis University and Washington University.

Carman’s work pioneered gastrointestinal radiology, resulting in the publication of *The Roentgen Diagnosis of Diseases of the Alimentary Canal* in 1917. He made frequent trips to the operating room to confirm his radiologic findings. In improving roentgenographic evaluation of the digestive tract, Carman advocated rapid screening fluoroscopy with manual palpation in an era of unreliable spot radiographs. To increase examination throughput, he emphasized making brief reports that omitted normal findings, as well as direct dictation into a tape recorder. He advocated meticulous recordkeeping and collection of cases, stating that “progress in roentgen diagnosis lies along three definite lines: ample material, full recording of all interpretations of this material, and careful comparison of those interpretations with surgical and pathologic findings.”

Carman became concerned about early abuse of the x-ray and advocated that roentgenology become a separate medical specialty. In 1910, he wrote, “Successful employment of the x-ray demands an intimate knowledge of a highly complex apparatus, practical acquaintance with the essentials of a good radiogram, ability to interpret a radiograph properly . . . and an appreciation of the dangers which may attend their careless and unskilled application.” In 1915, Carman became one of the 30 charter members of the Radiological Society of North America, serving as president in 1923. He was subsequently elected President of the American Roentgen Ray Society in 1924, the only person to serve as president of both organizations.

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Carman became ill in the fall of 1925 while returning to Rochester, Minnesota, from Washington, D.C., by train. His colleagues performed a fluoroscopic examination of his stomach and,
upon seeing the images, left them on his desk without comment. Carman held the films up to the window and stated, “Cancer of the stomach, inoperable.” Within the hour, he drove to St Paul and lectured before 2,600 physicians at the Interstate Postgraduate Assembly of North America. Carman remained active in roentgenology until his death in 1926. He bequeathed funds for a Carman Scholarship in Roentgenology and donated his medical library to the Department of Radiology at the Mayo Clinic (24).

Meckel Diverticulum
Meckel diverticulum (Fig 18) is the remnant of the omphalomesenteric (vitelline) duct and is the most common congenital anomaly of the gastrointestinal tract, occurring in 2%–3% of the population (25,26). The diverticulum usually occurs within 60 cm of the ileocecal valve on the antimesenteric side of the bowel and can be up to 8 cm in length. Its tip may be attached to the umbilicus, and about two-thirds of diverticula contain ectopic gastric mucosa. Patients most commonly present with gastrointestinal hemorrhage, although intussusception and diverticulitis can also occur.

Johann Friedrich Meckel, the Younger (1781–1833) (Fig 19), was born in Halle, Germany, to a family of distinguished anatomists. He received his medical degree from the University of Halle. While on a tour of the great medical centers of Europe, he was forced to hurry home to preserve his family’s extensive anatomic collection when French soldiers commandeered the family home as Napoleon’s temporary headquarters. After the war, he succeeded his father as Chairman of the Departments of Surgery and Pathological Anatomy at the University of Halle. Meckel was not
the first to describe the enteric diverticulum that bears his name, nor was he the first to recognize it as a remnant of the vitelline duct. However, his meticulous description of its embryonic origin fixed his name to it. Meckel was forced to retire at 50 years of age after developing severe paranoia and died a recluse 2 years later (27).

**Colon**

**Hirschsprung Disease**

Hirschsprung disease (Fig 20), or colonic aganglionosis, is a functional colonic obstruction resulting from the failure of neural crest cells to migrate the entire length of the gastrointestinal tract. Although the rectum is always involved, involvement of the more proximal colon varies as the neural crest cells migrate distally. Hirschsprung disease occurs in about one in every 5,000 live births, and 80% of cases manifest during the neonatal period. Barium enema examination is the imaging study of choice. The most common finding is a transition zone between a normal or narrowed aganglionic distal segment that is free of stool and a dilated, stool-filled proximal segment (28).

Harald Hirschsprung (1830–1916) (Fig 21) was the firstborn son of a successful cigar manufacturer in Copenhagen, Denmark. However, he...
forewent the family business for a career in medicine. Hirschsprung became interested in pediatrics after encountering six cases of tracheoesophageal fistula and, in 1877, was appointed Professor of Pediatrics in Copenhagen. He published extensively on pediatric conditions, including spinal dysraphism, hiatal hernia, and biliary atresia. However, he became a leader in pediatric surgery because of his work in congenital megacolon, intussusception, and pyloric stenosis. Hirschsprung was a pioneer in advocating the use of hydrostatic pressure for reducing intussusception and correctly predicted that treatment of pyloric stenosis would require surgery.

Hirschsprung was not the first to describe the condition that bears his name, but his experience with two cases led to a detailed description of congenital megacolon as a clinical entity (29). However, he failed to recognize that the cause of megacolon was the nondilated segment of distal bowel, and it was not until later in the 20th century, after his death, that intestinal aganglionosis was identified as the pathologic basis of the disease (30).

**Crypts of Lieberkühn**

The crypts of Lieberkühn (Fig 22) are the large, straight tubular glands of the colonic mucosa that consist of simple columnar epithelial cells and are responsible for water and electrolyte reabsorption as well as mucin production (31). Johann Nathanael Lieberkühn (1711–1756) (Fig 23) was born in Berlin and initially pursued a career in theology. However, science was his true passion, and, while studying medicine at Leiden University, he became interested in the new field of microscopy. Lieberkühn invented a device for illuminating specimens for microscopic examination, ultimately leading to his description of the crypt-like architecture of the colonic epithelium (32). However, to his contemporaries, his most significant contribution was assembling a collection of over 400 vascular tissue specimens (33).
**E coli Colitis**

*E coli* is one of the most studied and well-known enteric bacteria. It is a common cause of urinary tract infections but can also be responsible for food-borne illness. The O157:H7 strain of *E coli* was identified in 1982 during an outbreak of severe bloody diarrhea related to the ingestion of undercooked hamburger meat. This strain produces a powerful toxin that can result in hemolytic uremic syndrome in young children and elderly adults. In patients with *E coli* colitis and hemolytic uremic syndrome, abdominal radiography and barium enema examination may show “thumbprinting,” a finding that represents submucosal edema and hemorrhage (Fig 24) (28).

Theodor Escherich (1857–1911) (Fig 25) was born in Bavaria and studied medicine at several institutions (as was common in his day), including the University of Würzburg, where he focused on pediatrics. Influenced by the work of Robert Koch (1843–1910), Escherich became skillful in the new and growing field of bacteriology. In 1886, he published a monograph on intestinal bacterial flora in infants, in which he described several new organisms, including the one that now bears his name: *Escherichia coli* (34). While on the faculty at the University of Graz in Austria, Escherich studied nutrition in infants. His reputation continued to grow, and he was appointed head of St Anna Hospital in Vienna. However, his successful career was abruptly ended at the age of 53 years by a fatal stroke (35).

**Conclusions**

Numerous eponyms are encountered in radiology of the digestive tract, and this two-part series is by no means comprehensive. However, familiarity with many of these eponyms is important for accurate communication. Although some purists may argue against their use, eponyms serve as a means of honoring those who have made important discoveries and observations. Acquiring a little historical knowledge about these individuals brings some humanity back into the science of medicine.
Suggested Readings


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