Amnesia in modern surgery: revisiting Wangensteen's landmark studies of small bowel obstruction

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Summary

Before the publications of Owen Wangensteen and his colleagues in the early 1930s, bowel obstruction was almost always fatal, and its treatment was ineffectual. Patients rarely survived surgical attempts to relieve the obstruction. Although other investigators were active in the field, the understanding of the pathophysiology of obstruction belongs almost entirely to Wangensteen. In this commentary, we review Wangensteen's landmark studies of small bowel obstruction and how they shaped the treatment of this condition.

During recent ward rounds, while reassessing a patient with adhesion-induced partial small bowel obstruction we learned that the nursing staff, at least some of the residents and even some attending staff were unaware of the exact role of nasogastric suction in its treatment. It called to mind some quotes of Dr. Owen Wangensteen, Chairman of the Department of Surgery at the University of Minnesota from 1930 to 1968. He said, "There exists a feeling among many physicians and students that anything over 10 years old has no pertinence," and "If you only look forward, it's tantamount to having a physician with total amnesia. How good would he be?"[1] These observations are particularly appropriate to our patient, as Wangensteen literally wrote the book on bowel obstruction and received the 1935 Samuel D. Gross Prize for his research in this area.[2]

Before the publications of Wangensteen and his colleagues in the early 1930s, bowel obstruction was almost always fatal, and its treatment was ineffectual. Most surgeons believed that death was due to the production of toxic factors in the gut and their absorption into the blood stream. The source of the gaseous distention was attributed to the action of bacteria on retained food and the production of methane. The excess of fluid in the gut above the point of obstruction was appreciated, but the failure of patients to improve with intravenous infusions of saline was a great disappointment. Patients rarely survived surgical attempts to relieve the obstruction.

Although other investigators were active in the field, the understanding of the pathophysiology of obstruction belongs almost entirely to Wangensteen. He reiterated others' experiments, showing where they were incorrect or misinterpreted. He then developed a series of convincing experiments in dogs that established the cause of the signs and symptoms and subsequently their treatment. His group measured the intraluminal pressures and absorption of fluids in the various parts of the gastrointestinal tract in healthy and in obstructed small bowel. They delineated the role of the lymphatics, the capillaries and the venules during obstruction. In addition, they defined the bacteriology of obstruction and its manifestations at various time points and in various areas of the obstructed gut. They tested whether the well-described lethality could be transferred to normal animals by the injection of intestinal secretions or peritoneal fluid from affected animals.

The most telling investigations were into the origin and make-up of the characteristic gas and fluid accumulations in distended gut. The initial part of the experiment was to ligate the mid-ileum in all the experimental animals. Then in half of the animals the esophagus was divided and the upper end brought out to the skin as a mucous fistula. In the other half the gastrointestinal tract, though completely obstructed, was otherwise left intact. All the animals received intravenous electrolyte infusions. The animals that had the esophagus diverted did not become distended with either gas or fluid and survived for prolonged periods. Those with an intact esophagus experienced the classical consequences of small bowel obstruction, from which they died despite the intravenous fluids. With this insightful series of experiments, Wangensteen proved that swallowed air causing distension was the culprit. In further trials, both in animals and in humans, he showed that removal of the air by means of gastric tubes rescued both experimental animals and patients alike. Under these circumstances, an operation could be done safely or, in many instances, could be avoided altogether. In the absence of swallowed air, healthy gut fluids; saliva; gastric, pancreatic and enteric juices; and bile could be absorbed by the obstructed small intestine. Wangensteen worked out the suction pressures that allow gastric air to be removed and constructed a bedside device that could do this effectively. He demonstrated that the excess fluid accumulation above the obstruction was due to the distension pressure of the swallowed air on the bowel wall impeding venous outflow but not arteriolar inflow. These conditions then interrupted fluid absorption by the intestinal mucosa. He also concluded that there was no advantage to advancing the tube beyond the stomach. The essential function of the gastric tube is to remove air, not fluid.

After having clarified the true function of the nasogastric tube on our ward rounds, a new question arose. If air is the important element removed by suction, but it is not measured by the suction device, how did Wangensteen know when to remove the tube? In his book, *The therapeutic problem in bowel obstructions: a physiological and clinical consideration*,[3] Wangensteen listed the following criteria: 1) cessation of "gas pains;" 2) decrease of abdominal distension; 3) the visualization of gas in the colon on the radiograph in complete obstructions, indicating that that the obstruction has been overcome; 4) less fluid aspirated through the tube, denoting that stasis is no longer prominent; and 5) toleration of temporary discontinuation of suction without recurrence of pain. It is interesting that the amount of fluid aspirated was number 4 on his list of criteria when for many surgeons today it is number 1 — a reflection of the misguided emphasis on intestinal fluid aspiration.

Wangensteen received the Gross Prize for these pioneering research studies, and their description is incorporated as Part I in his landmark book.[3] The remaining sections are devoted to clinical aspects and the recognition, diagnosis and guiding principles of treatment. Chapters are devoted to the various subtypes of congenital and acquired large and small bowel obstruction. In its subsequent revised editions, the book became a bible for the next 30 years and still guides the way surgeons approach this common problem.

Surgeons of a certain vintage will remember when the bedside apparatus was called the Wangensteen Suction. Paradoxically, the nasogastric tube is still called the Levin tube, although its invention in 1921 was as an investigative aid for radiology and antedated its use in bowel obstruction. Many of the authors writing in today's standard textbooks of surgery do not properly describe these principles on which current decompression of the gut is based, probably assuming that they are self-evident.

The aphorism that those who don't know history are condemned to repeat it happily applies to current surgeons who treat bowel obstructions as Wangensteen taught us — even if many don't understand why.

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