During a four-year period, over 300 patients developed problems from acute stress gastric bleeding (ASGB). A prospective study of this entity was performed by doing serial photography with the gastric camera, gastric acid and gastric pH measurements, gastric mucosal mucin studies, and clinical correlations with the severity of injury or sepsis. Patients requiring operation for control of bleeding had full-thickness gastric biopsies removed at surgery; these were freshly fixed and studied by light and electron microscopy. Based on these studies, a thorough clinical pathological correlation of this entity was made and recommendations for treatment were provided. [The SCI® indicates that this paper has been cited in over 180 publications since 1971.]

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During my residency, the chief surgical resident stopped me during “ward rounds,” pointed to a sick patient, and told me he was bleeding from the stomach. He directed me to insert a nasogastric tube, irrigate the stomach free of blood, start intravenous therapy, and call the patient’s resident physician. I was amazed when blood returned from the nasogastric tube and was told bleeding was obvious from the “look on the patient’s face.”

Armed with this memory, I set out to learn more about this entity, acute stress gastric bleeding (ASGB), when I finished my residency and became a staff physician. A multidisciplinary study was organized to analyze the clinical, photographic, microscopic (light and electron), and surgical aspects of ASGB beginning before bleeding developed and continuing until the patient either died or fully recovered. Such a comprehensive review of ASGB was unique; some of the photographic and electron microscopic prints are still unduplicated in detail and quality.

Many obstacles were encountered. Although I directed the project, I was the youngest in age. Multidisciplinary meetings, therefore, had to be conducted in the offices of one of my older colleagues. One such meeting led to heated discussions regarding the role of gastric mucosal mucin in prevention of ASGB. My elder colleague insisted that mucin was important, referred to the work of Horace Davenport, and wished that we had access to his current viewpoints. By coincidence, Davenport, the originator of the concept about gastric mucosal defenses, was giving a lecture two floors above us in the old Detroit Receiving Hospital where he was the annual visiting lecturer for our Alpha Omega Alpha chapter. Consequently, I delivered Davenport to my colleague within 30 minutes; Davenport supported my view that mucin deficiency was not responsible for ASGB.

This article has been frequently cited because it provides many previously unpublished facts about ASGB and provides therapeutic guidelines based upon a very large clinical experience. Subsequent work on ASGB is summarized in “Stress ulceration: the clinical problem.”


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