Guest Editorial

What are Hemorrhoids and What is Their Relationship to the Portal Venous System?

WILLIAM C. BERNSTEIN, M.D.

Bernstein WC. What are hemorrhoids and what is their relationship to the portal venous system? Dis Colon Rectum 1983;26:829-834.

New concepts of the pathophysiology of hemorrhoids have been defined during the past eight or more years, yet medical education at the undergraduate and graduate levels has not kept pace with the newer concepts. The traditional concepts are being perpetuated in all medical dictionaries and in most textbooks of surgery, medicine, anatomy, and pathology. Hemorrhoids are not varicosities, but rather are vascular cushions composed of arterioles, venules, and arteriolar-venular communications which slide down, become congested and enlarged, and bleed. The pathogenesis begins in the fibromuscular supporting layer in the submucosa, above the vascular cushions. The bright red bleeding, which accompanies hemorrhoidal disease, is arteriolar in origin. Portal hypertension has been shown not to be the cause of hemorrhoids. The use of rubber bands, sclerosing solutions, cryosurgery, or the infra-red beam in the early stages of hemorrhoidal disease can take care of prolapse and bleeding and can prevent the development of third and fourth degree hemorrhoids. [Key words: Varicosities; Vascular cushions; Sliding-down process; Early treatment and prevention; Portal hypertension]

From the Division of Colon and Rectal Surgery, Department of Surgery, University of Minnesota, Minneapolis, Minnesota

PROGRESS IN SCIENTIFIC KNOWLEDGE is, at times, caught in the cobwebs of tradition, and slowed. Traditions in medicine, as related to concepts of disease and methods of treatment, are very difficult to change. Witness the time that it has taken for the high-fiber diet to replace the soft, smooth, low-residue diet in the treatment of constipation, the spastic bowel syndrome, and chronic diverticular disease. Witness also the time that it has taken the profession to discard the theory that massive bright red bleeding from the rectum was usually caused by diverticulitis. The concepts of what hemorrhoids are, the proper methods of treatment, and their relationship to the portal venous system have undergone marked changes in recent years, yet medical teaching, at both the undergraduate and graduate levels, has not kept pace with the newer concepts.

What are the traditional concepts of hemorrhoids? Briefly stated, they are as follows: 1) Hemorrhoids are varicosities of the hemorrhoidal veins; 2) they are caused chiefly by increase in pressure in the portal venous system; 3) the bright red bleeding which accompanies a large percentage of cases of hemorrhoidal disease is considered to be venous in origin.

Dorland's Medical Dictionary, 26th edition, published in 1981,[1] defines hemorrhoids as "a varicose dilatation of a vein of the superior or inferior hemorrhoidal plexus, resulting from a persistent increase in venous pressure." Buie, in *Practical Proctology,* second edition, published in 1960,[2] stated, "Hemorrhoids are tumors composed of collections of varicose veins..." Goligher, in *Surgery of the Anus, Rectum and Colon,* third edition, published in 1975,[3] states, "Internal piles are essentially varicosities of the venous plexuses in the wall of the anal canal and lowermost half inch or so of the rectum." All medical dictionaries and almost all standard textbooks of medicine, surgery, gastroenterology, anatomy, and pathology use essentially the same definitions and continue to perpetuate the traditional concepts.

We shall attempt in this report to examine the supporting evidence for the newer concepts, to place them in historical perspective, and to correlate them with our own clinical, anatomic, and gross and microscopic studies.

The long-awaited change in concepts of hemorrhoidal disease began in 1975, when W.H.F. Thomson[4] published his master's thesis, "The Nature of Haemorrhoids," submitted to London University the previous year. His

Read at the meeting of the American Society of Colon and Rectal Surgeons, Boston, Massachusetts, June 5 to 9, 1983.

This study was conducted under a grant from the Phillips Family Foundation, Minneapolis, Minnesota.

Address reprint requests to Dr. Bernstein: Box 327 Mayo, University of Minnesota Hospitals, Minneapolis, Minnesota 55455.

anatomic and radiologic studies are most impressive and convincing as to the cause of and the principles of treatment necessary for clinical hemorrhoids. The term "vascular cushions" was introduced by Thomson and has proven to be a particularly appropriate and descriptive term. Since vascular cushions in the anal canal can be demonstrated at birth and are present in most adults in three areas of the anal canal even when no symptoms of protrusion or bleeding are present, it must be assumed that they are normal structures; it has been suggested that they contribute to the maintenance of anal continence. Only when the supporting tissues in the submucosa of the rectal lining above and around the vascular cushions permit the cushion to slide down do the symptoms of protrusion and bleeding occur.

Thomson's studies were influenced in part by the report of Gass and Adams in 1950[5] who suggested that piles result from degeneration of supporting tissues in the anal canal. The description of the smooth-muscle layer in the submucosa by Treitz in 1853[6] appears also to have contributed to Thomson's ideas.

Again, in 1975 the newer concepts were further detailed in a paper by J. Alexander-Williams and Andrew R. Crapp[7] when they stated:

> Modern concepts of the pathophysiology of hemorrhoidal disease are based on the belief that vascular anal cushions are part of the normal anatomical state and it is believed that only when these cushions are prolapsed through the anal canal and congested by the internal sphincter on defecation that the abnormal state referred to as hemorrhoids exists...If the prolapse of the vascular cushion can be prevented, or if the congesting effect of a tight anal canal can be abolished, then the anal cushions will return to their normal state and symptoms will be prevented without the necessity to remove the cushions themselves.

It was Stelzner, Staubesand, and Machleidt in 1962[8] who demonstrated the arteriovenous communications in the submucosa of the anal canal and likened this tissue to erectile tissue, which they called corpus cavernosum recti, an observation which had been made years earlier by Velpeau (1826), Malpaigne (1837), Bourgery (1840), and Cruveilheir (1852). These early authors felt that hemorrhoids were due to metaplasia of this erectile tissue and that the bright red bleeding could be explained on the basis of the arteriovenous communications.

In 1977, Spiro,[9] in the second edition of *Clinical Gastroenterology,* stated:

> The clinician should not think of hemorrhoids as large dilated blood-filled spaces but, looking at a cross-section, should recognize that a hemorrhoid is composed of spongy vascular tissue which will tend to ooze and not bleed massively.

Our studies (Fig. 1) of microscopic sections of internal and external hemorrhoids confirm Spiro's observations and show that hemorrhoids are, in fact, masses of spongy, vascular tissue composed of arterioles, venules, and arteriolar-venular communications in a stroma of fibrocollagenous fibers. The absence of varicosities is striking.

In 1978, Sleisinger and Fordtran,[10] in the second edition of *Gastrointestinal Disease,* stated, "Hemorrhoids are dilated veins of the hemorrhoidal plexus in the anal canal and lower rectum." In subsequent paragraphs, however, the authors refer to the newer concepts and hypotheses, stating that they are "attractive, but the issue has not been settled." In 1979, Goldberg and Nivatvongs, in the chapter on Colon, Rectum and Anus in Schwartz's *Principles of Surgery,* third edition,[11] state:

> In the human being, the upper anal canal is lined by separate, bulky cushions of specialized submucosal vascular tissue, likened to erectile tissue composed of stroma of elastic muscle fibers derived from the outer longitudinal coat of the colon.

In 1980, Goldberg, Gordon, and Nivatvongs, in *Essentials of Anorectal Surgery,* stated[12]:

> A precise definition of hemorrhoids does not exist because the exact nature of the condition is not completely understood. Upon examination a mass of vascular tissue is seen in the anal canal. Various theories have been proposed regarding the exact nature of this tissue. For many years these swellings have been considered varicosities of the hemorrhoidal plexus, but this is probably an oversimplification.

However, in 1982, Nivatvongs and Goldberg stated[13]: "Traditionally, rubber band ligation was applied in the hemorrhoid itself based on the old concept that the underlying problem was in the hemorrhoidal plexus." They now subscribe to the idea of Alexander-Williams and Crapp and are ligating the redundant mucosa above the hemorrhoid and are obtaining excellent results.

> This is based on the current concept that a hemorrhoid is caused by the downward displacement of the anal cushions and that anal cushions may contribute to the mechanism of anal closure and of anal continence. Hence, if at all possible, the hemorrhoidal tissue should be preserved.

The bright red blood, which is observed on the toilet tissue, on the surface of the stool, or in the water in the toilet bowl, is due to congestion of the hemorrhoid and erosion of the tiny arterioles on its surface. Occasionally, a larger arteriole can become eroded, and bright red blood will spurt from it. This can lead to the passage of large amounts of bright red blood.

Most cases of external hemorrhoids accompany, or are secondary to, the sliding down of the vascular cushions above the pectinate line. Thus it is that causing a fibrous fixation to prevent the sliding down by rubber bands, sclerosing solutions, or other methods will lessen the

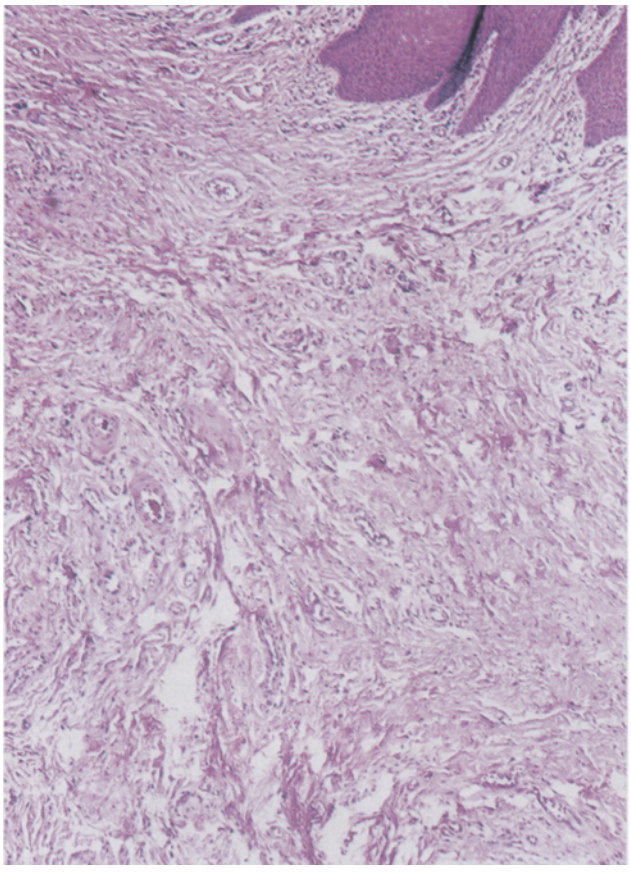

FIG. 1. Hemorrhoidal tissue (low power). Note arterioles, venules and A-V communication. Note absence of varices (original magnification ×25).

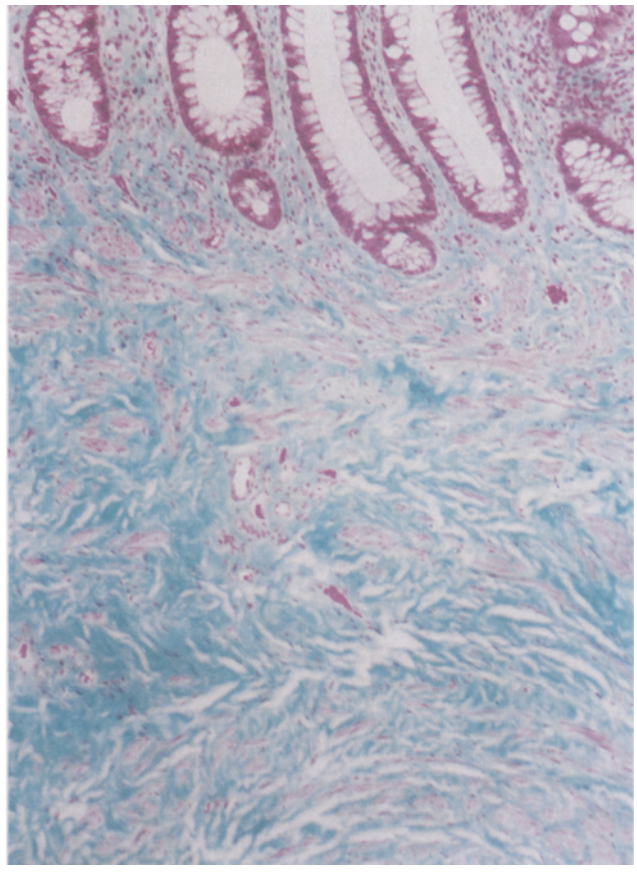

FIG. 3. Submucosa above prolapsed hemorrhoid. Note complete disintegration and fragmentation of muscle layer (trichrome stain; original magnification ×63).

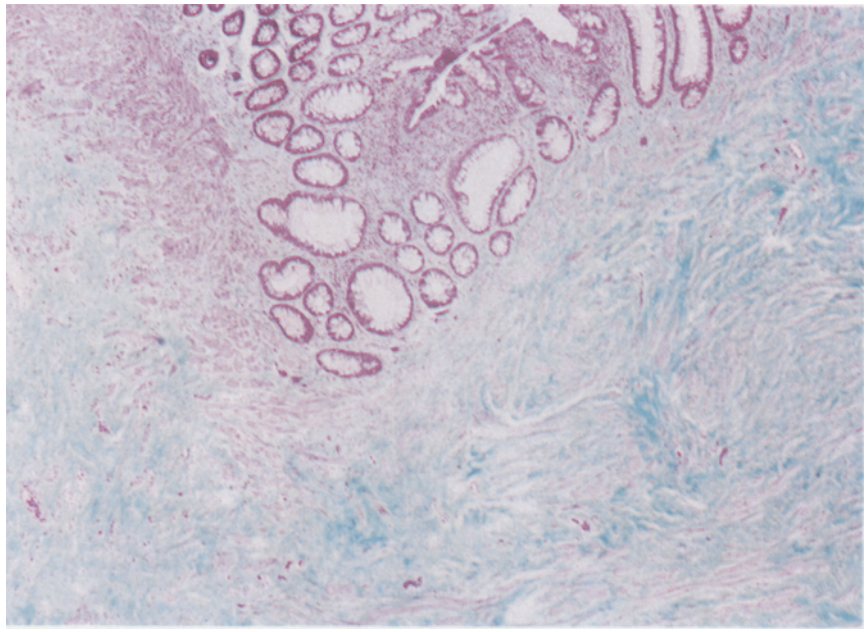

FIG. 2. Submucosa above prolapsed hemorrhoid. Note intact submucosal muscle layer on left; disintegrated and fragmented muscle on right (trichrome stain; original magnification ×25).

occurrence both of internal and external hemorrhoids.

As early as 1932, Hiller[14] stated that his technique for the injection treatment of internal hemorrhoids differed from

> ...that commonly recommended in that the injection is not made into the hemorrhoid itself but well above the pile in an area free from ulceration and infection... bleeding piles cease to bleed within a few days and the procedure is painless and without danger.

This method prevented further prolapse and caused shrinkage of the pile to occur.

Haas and Fox, of the Henry Ford Hospital, in 1980[15] concluded that, "The disintegration of the submucosal connective tissue fibers with advancing age appears to be a likely cause of asymptomatic hemorrhoid formation."

Our studies, showing the disintegrated, fragmented muscle and connective tissue fibers in the submucosal layer of the rectal wall above the prolapsing hemorrhoid, are convincing evidence of the cause of the prolapse and support the findings of Haas and Fox (Figs. 2 & 3). However, in our experience, many young people, in the third and forth decades of life, have third and fourth degree hemorrhoids. Therefore, we cannot support their contention that advancing age is the all-important factor.

We do not wish to minimize the role played by pregnancy, straining during the passage of hard stools, rectal tumors, and severe diarrhea in aggravating the hemorrhoidal condition, but the basic etiologic factor appears to be the sliding down of the vascular cushion, as described above. After stating that hemorrhoids are varicosities of the venous plexuses, Goligher [3] goes on to say that placing the sclerosing solution into the submucosa above the piles "is found to give a much better devascularizing effect than injection into the submucosa of the piles themselves..." Obviously Goligher, too, subscribes to the sliding-down theory.

At the University of Minnesota and its affiliated hospitals, the number of surgical hemorrhoidectomies performed has been declining steadily since the present method of treatment has been adopted. It is our firm conviction that if the early symptoms (protrusion and bleeding) of hemorrhoidal disease are treated by banding, injecting, cryosurgery, or infrared beam to the submucosa above the hemorrhoid, surgical hemorrhoidectomy will become a less indicated procedure.

Let us now examine the case of portal hypertension as the cause of clinical hemorrhoids. This concept has been the accepted one for several centuries and is still listed as a principal cause in modern textbooks. In the 1977 edition of *Pathology* by Anderson and Kissane,[16] we find, "Hemorrhoids are varicosities of the hemorrhoidal vein... they result from increased venous pressure related to such causes as portal hypertension,..." However, as early as 1860, in speaking of the relationship between hemorrhoids and portal hypertension, Frericks, as quoted by Liebowitz,[17] stated, "Enlargement of hemorrhoidal veins are also by no means frequent; their occurrence has been assumed by many writers a priori, rather than actually observed."

The facts that there are no valves in the portal venous system, and that human adults are in the erect position much of the time, should lead one to expect hemorrhoids to be congested and enlarged frequently in patients with portal hypertension, if hemorrhoids are, in fact, tributaries of the portal veins.

In 1958, Hunt[18] reported that for two years all patients with portal hypertension had been sigmoidoscoped without the discovery of anything of significance, and in 1980, Bubrick *et al.*[19] studied the records of 188 patients with portal hypertension seen at the Hennepin County Medical Center in Minneapolis. They found an incidence of 28 per cent of identifiable hemorrhoids in this population, while reports of identifiable hemorrhoids in the general adult population range from 50 to 80 per cent. There were 23 patients with documented hemorrhoidal bleeding, of whom six bled massively. An interesting finding was that, in this study, 52 per cent of the entire group had coagulation defects. In those who bled massively, 82 per cent had coagulation defects.

In our present study of 50 patients with portal hypertension, 25 of whom were seen and examined on the service of Drs. Marshall J. Orloff and Richard H. Bell at the University Hospitals in San Diego and 25 on the service of Dr. Melvin Bubrick at the Hennepin General Hospital in Minneapolis, there was again an incidence of 28 per cent with identifiable hemorrhoids. All of these patients had had esophageal bleeding, and many had abdominal ascites. The patients were seen before or after receiving portal-systemic shunts. One patient with marked coagulopathy died from renal failure and pneumonia one month after surgery for rectal hemorrhage; another, who had severe rectal bleeding, was well-controlled by banding of the hemorrhoids and of the mucosa above the hemorrhoids.

Varices of the colon, according to some reports in the literature, occur infrequently. This is supposed to be due to the free communication between the portal and systemic venous systems. In 1957, Levy *et al.*[20] reported an unusual case of a massive varix of the cecum, which penetrated into the lumen of a patient with portal hypertension, causing a severe hemorrhage. They could find no reports of similar cases in the literature. In 1966, Lopata and Berlin[21] reported that bleeding varices of the colon are extremely rare, there having been but seven cases reported previously in the literature. They added the eighth case. However, by 1980, Izsak and Finlay,[22] in an extensive review of the literature, found 29 previously reported cases and added three additional ones.

Dr. Marshall J. Orloff, at the University of California in San Diego, a foremost investigator in this field, in a personal communication, stated that, in his experience:

> Varices of the colon are not uncommon, but that bleeding from these varices is an infrequent occurrence. It is not known why esophagogastric varices often rupture, while those commonly found throughout the remainder of the gastrointestinal tract and peritoneal cavity bleed infrequently. It has been postulated that an important difference between esophagogastric varices and those found elsewhere is related to the wide swings in pressure caused by respiratory movements of the diaphragm, which weaken the walls of the varices located in the region of the cardia and make them particularly vulnerable to rupture.

Regarding the actual paucity of large, prolapsing, bleeding hemorrhoids in cases of portal hypertension, a more positive explanation would be welcome. Does the outflow of venous blood from the vascular cushions and clinical hemorrhoids proceed to the portal system via the superior rectal vein? To date, no one, including ourselves, has been able successfully to do an intravenous injection of contrast material into a small vessel in a hemorrhoid to determine its exact course radiographically. Such a demonstration would settle this question. We have also attempted to perform microdissection of the venous outflow of hemorrhoids on cadaver specimens. Our findings were not conclusive, because of the inability to trace the tiny vessels into the larger veins.

Further studies on the arterial supply and venous drainage from hemorrhoids are currently in progress at the University of Minnesota and will be the basis of a future report.

From our own studies and the well-documented studies of the arterial and venous blood supplies of the rectum reported by Thomson in 1975,[4] the conclusion can be drawn that there is sufficient evidence to support the thesis that venules in vascular cushions and clinical hemorrhoids are direct tributaries of the systemic venous system rather than of the portal system. This statement is also supported by *Morris' Human Anatomy*,[23] which states that "...the internal hemorrhoids are associated with the middle rectal veins." This textbook of anatomy no longer refers to the hemorrhoidal veins but has adopted superior, middle, and inferior rectal veins as current terminology.

In Thomson's study,[4] after injecting both the superior rectal vein and the superior rectal artery, vascular dilatations were seen and demonstrated in neonates and in a 3-month-old infant, as well as in adults. These dilatations were similar to the dilatations described by Quénu[24] in 1895, who likened them to "bunches of grapes." These dilatations were present circumferentially and did not conform to the location of the three vascular cushions. Thomson also showed that there was free communication between the superior, middle, and inferior rectal veins in all specimens that they were able to dissect after injection with latex. Since the middle and inferior rectal veins are part of the systemic venous system, and with free communication with the superior rectal vein, the nonoccurrence of hemorrhoids in portal hypertension becomes more understandable.

Orloff, in 1980,[25] after stating that hemorrhoids are no more common in the portal hypertension population than in the general population, noted that, in his experience of over 1,100 portocaval shunts, he had seen severe rectal bleeding from rectal varices in fewer than five cases. Rectal varices, which are not to be confused with clinical hemorrhoids are, in themselves, also very infrequent in portal hypertension. Spiro, in 1977,[9] published the first photograph of rectal varices in portal hypertension and in Johansen, Bardin, and Orloff's[25] paper of 1980, there is an artist's drawing of their case of true rectal varices. We have not encountered a single case of true rectal varices in our experience at the University of Minnesota Hospitals.

From the evidence published in the literature and from our own studies, it appears that the case against portal hypertension as a cause of hemorrhoids is strong and valid. In those patients who have portal hypertension and do have concurrent hemorrhoids, severe bleeding may be a problem, because of coagulation defects. Banding of the mucous membrane above the hemorrhoids, in their early stages, could prevent serious problems, including bleeding, even in patients with portal hypertension.

Summary and Conclusions

It is the considered opinion of this essayist that the time has come to abandon the traditional concepts of hemorrhoidal disease and of its relationship to portal hypertension. Hemorrhoids are not varicose veins, but rather vascular cushions composed of arterioles and venules, with their communications in a stroma of fibrocollagenous supporting tissue. Vascular cushions are normal structures and do not produce symptoms until there is deterioration of the fibromuscular supporting tissue in the mucosa above the cushions, which permits a sliding-down process of the cushion to occur. Only then do the cushions prolapse, become congested, enlarge, and bleed. Early symptoms should be treated and progression of the disease prevented by simple methods, using elastic bands, sclerotherapy, cryotherapy, or the infra-red beam.

The cause of hemorrhoids should no longer be attributed to portal hypertension. When portal hypertension and hemorrhoids coexist in a patient, bleeding may become a major problem due to coagulopathy, which is present in a large percentage of portal hypertension patients.

The bright red bleeding seen in a large percentage of hemorrhoidal cases should not be looked upon as venous bleeding. This bleeding is arteriolar in origin.

The newer concepts that have been presented should, if accepted, lead to a new era of treatment for early symptoms of hemorrhoidal disease. This new approach would, in all likelihood, lessen the need for surgical hemorrhoidectomy.

Acknowledgments

The author wishes to acknowledge with thanks the valuable library research assistance of Jeffrey D. Palarski.

Permission to do proctoscopic examinations on portal hypertension patients of Marshall J. Orloff, M.D., and Richard H. Bell, Jr., M.D., at the University Hospital in San Diego, and of Melvin P. Bubrick, M.D., at the Hennepin County Medical Center in Minneapolis, is gratefully acknowledged.

The Department of Surgical Pathology at the University of Minnesota Hospitals has been most cooperative and helpful in making special sections and using special stains in this study.

The secretarial and special assistance of Carol Bonemeyer and Colleen Duffy is gratefully acknowledged. Their help took on increased significance, due to the physical handicaps of the author.

References

1. Dorland's Medical Dictionary. 26th ed. Philadelphia: WB Saunders, 1981:700.
2. Buie LA Sr. Practical proctology. 2nd ed. Springfield: Charles C Thomas, 1960.
3. Goligher JC. Surgery of the anus, rectum and colon. 3rd ed. London: Bailliere Tindall, 1975.
4. Thomson WH. The nature of haemorrhoids. Br J Surg 1975; 62:542-52.
5. Gass OC, Adams J. Hemorrhoids: etiology and pathology. Am J Surg 1950;79:40-2.
6. Treitz. Ueber einen neuen muskel am duodenum des menschen, über elastische sehnen und einige andere anatomische verhältnisse. Verhaltnisse Viertel Jahrschrift Prar. Heilkunde (Prager) 1853;1:113-44.
7. Alexander-Williams J, Crapp AR. Conservative management of haemorrhoids. Clin Gastroenterol 1975;4:595-601.
8. Stelzner F, Staubesand J, Machleidt H. Das corpus cavernosum recti: die grundlate der inneren hammorrhoiden. Langenbecks Arch Chir 1962;299:302-12.
9. Spiro HM. Clinical gastroenterology. 2nd ed. New York: Macmillan, 1977.
10. Sleisinger MH, Fordtran JS. Gastrointestinal disease. 2nd ed. Philadelphia: WB Saunders, 1978.
11. Schwartz SI. Principles of surgery. 3rd ed. New York: McGraw-Hill, 1979.
12. Goldberg SM, Gordon PH, Nivatvongs S. Essentials of anorectal surgery. Philadelphia: JB Lippincott, 1980.
13. Nivatvongs S, Goldberg SM. An improved technique of rubber band ligation of hemorrhoids. Am J Surg 1982;144:379-80.
14. Hiller RI. Internal hemorrhoids. Am J Surg 1932;16:64-6.
15. Haas PA, Fox TA. Age-related changes and scar formations of perianal connective tissue. Dis Colon Rectum 1980;23:160-9.
16. Anderson WA, Kissane JM. Pathology. 7th ed. St. Louis: CV Mosby, 1977: Vol. 1.
17. Liebowitz HR. Bleeding esophageal varices: portal hypertension. Springfield: Charles C Thomas, 1959.
18. Hunt AH. A contribution to the study of portal hypertension. Edinburgh: Livingstone, 1958.
19. Jacobs DM, Bubrick MP, Onstad GR, Hitchcock CR. The relationship of hemorrhoids to portal hypertension. Dis Colon Rectum 1980;23:567-9.
20. Levy JS, Hardin JH, Shipp H, et al. Varices of cecum as unusual cause of gastrointestinal bleeding. Gastroenterology 1957; 33:637-40.
21. Lopata HI, Berlin L. Colon varices: a rare cause of lower gastrointestinal bleeding. Radiology 1966;87:1048-50.
22. Izsak EM, Finlay JM. Colonic varices. Am J Gastroenterol 1980;73:131-6.
23. Anson BJ, ed. Morris' human anatomy. 12th ed. New York: McGraw-Hill, 1966.
24. Quénu E, Hartmann H. Chirurgie du rectum. Paris: G Steinheil, 1895:335-73.
25. Johansen K, Bardin J, Orloff MJ. Massive bleeding from hemorrhoidal varices in portal hypertension. JAMA 1980;244:2084-5.