Seminars in Colon and Rectal Surgery 29 (2018) 160–166

Contents lists available at ScienceDirect

Seminars in Colon and Rectal Surgery

journal homepage: www.elsevier.com/locate/yscrs

Reprint of: Why are hemorrhoids symptomatic? the pathophysiology and etiology of hemorrhoids

William C. Cirocco, MD, FACS, FASCRS*

Department of Surgery, University of Missouri–Kansas City, Kansas City, Missouri.

A B S T R A C T

Hemorrhoids are a normal component of the anorectum and contribute to the mechanism of anal closure, thus providing fine adjustment of anal continence. There are numerous myths and legends associated with the disordered and diseased state of hemorrhoids. Fortunately, information obtained from modern technologies including microscopic histopathology defined first the actual substance and makeup of hemorrhoids and was later combined with anorectal physiology to provide evidence establishing the underlying pathophysiology of this universal finding of the human anorectum. The sliding anal canal theory of Gass and Adams has held up and is further supported by other anatomic studies including the work of WHF Thomson, who popularized the term "cushions" to describe the complex intertwining of muscle, connective tissue, veins, arteries, and arteriovenous communications which constitute hemorrhoids. A loss of muscle mass in favor of connective tissue over time helps explain the role of aging as a predisposing factor for symptomatic hemorrhoids. Other factors include the modern "rich" or low-residue diet leading to constipation and straining which contributes to prolapsing cushions. Pathologic studies also demonstrated arteriovenous communications explaining why hemorrhoid bleeding is typically bright red or arterial in nature as opposed to dark or venous bleeding. Technology associated with anorectal physiology studies have demonstrated an increase in pressures of the internal sphincter, which return to normal following surgical intervention. This information backs the conclusion that high sphincter pressures are not the cause of hemorrhoid symptoms, but rather the effect of symptomatic hemorrhoids because of the observed return to normal baseline pressures after surgical removal. While there may be a genetic component to this disorder, there are no known genetic markers for hemorrhoid disease at this time.

There has been much confusion regarding the nature and pathophysiology of hemorrhoids. Hemorrhoids are a normal component of the anorectum and contribute to the mechanism of anal closure, thus providing fine adjustment of anal continence. Factors contributing to this confusion include the false definitions that have been propagated in nearly every medical textbook and dictionary, which has only added credence to the various myths and legends associated with hemorrhoids and hemorrhoidal disease.[1]

Eber's Papyrus from Egypt (about 1550 B.C.) describes the condition probably with associated dysentery.[2] The Hindus (about 1000 B.C.) refer to the disease as does Hippocrates (460-370 B.C.).[3] In the Talmud, a sedentary mode of life is mentioned among the causes of hemorrhoids.[4] In the First Book of Samuel in the Old Testament of the Bible,[5] the Philistines captured "the ark of the God of Israel" in battle and brought it in succession to Ashdod, then Gath, then Ekron. The ark was moved because in each city the people were stricken with hemorrhoids among other plagues. After the third city had been ravaged, the people of Ekron demanded that the philistine lords "send away the ark of the God of Israel. Let it return to its own place, that it may not kill us and our kindred." Once again, "the hand of God has been very heavy upon it. Those who escaped death were afflicted with hemorrhoids, and the outcry from the city went up to the heavens."[6]

According to Ebstein, "in the Medieval Ages the Greek term of hemorrhoids was almost lost, since the monks were even more imperficient in Greek than in Latin. And as they did not understand the Greek word they formed all kinds of misnomers, such as emeroides, einorides, eneroyde, ammorroides, by which they understood all kinds of anal tumors."[7]

Celsus considered hemorrhoids to be "a means of purification, and maintains that they should not be suppressed lest the unsound matters in the body be carried to the cardiac region and to the viscera."[3]

It has taken careful microscopic study of anatomic specimens over several decades to provide the pieces of the puzzle and eventual

* Address reprint requests to William C. Cirocco, MD, 8901 W. 74th Street, Suite 148, Shawnee Mission, KS 66204.
E-mail address: wcirocco@kc.rr.com

https://doi.org/10.1053/j.scrs.2018.11.002
1043-1489/

solution of the problem of etiology and pathophysiology of the diseased state of hemorrhoids. The histopathologic findings provide the foundation on which to establish the pathophysiology and natural history of hemorrhoids so as to dispel the various myths and legends which have been handed down over time. Finally a working theory could be assembled that can accommodate all of the established anatomic findings into a logical solution to explain this universal part of human anatomy, formed early in embryonic life,[8] with an obscure function, yet responsible for so much morbidity for a significant part of the world's population.

Varicose Vein Theory

Many early writings expressed the opinion that bleeding from hemorrhoids was desirable and Galen, writing in the 2nd century, was one who believed that "unsound juices" were discharged from the body by this route.[5] The anatomist Hyrtl noted that "the golden veins of the anus" were not mentioned before the 16th century by Ryff (1541).[7] The name denotes the typical spontaneous bleeding which, in folklore, was believed to remedy the various maladies of the ages. This provided a cure without a visit to the doctor for medicinal bloodletting, thus saving the patient from the doctor's customary fees—hence, the name "golden veins."[3,5]

Anatomic findings dating to the classic dissections of John Hunter (Hunterian Museum) showed discrete venous dilatations within hemorrhoid specimens supporting the varicose vein theory.[9] These dilatations were thought to be the main pathologic change in the disorder. An increase in local venous pressure combined with a localized weakness in the vein wall were thought to result in hemorrhoidal disease.

Contributing factors were said to be the known absence of valves in the portal vein and its tributaries. Add to this the effect of gravity on the upright posture of man combined with the increased intra-abdominal pressure from straining at defecation providing a localized increase in venous pressure. However, further anatomic study established the free communication between the portal and systemic circulations in the anal canal via the middle and inferior rectal veins, which drain into the internal iliac veins and back to the heart via the inferior vena cava. Therefore, any local increase in venous pressure may dissipate via this route. Although portal hypertension may lead to rectal varices in extreme cases, they do not resemble standard hemorrhoid disease and will not be addressed here.

Quenu and Hartmann, in the late 19th century, recognized these anatomic facts and believed a weakness in the hemorrhoid vein wall, perhaps brought about by local infections from the repeated trauma of defecation, was responsible for hemorrhoid disease.[10] However, they recognized that "there was little histological evidence to back up" this theory.[9] Unfortunately, the infectious theory of hemorrhoids was taken up by several prominent surgeons of the 20th century. The absence of any histologic evidence of underlying infection or sepsis in studies from the mid- to late 20th century doomed this theory.[11]

Further anatomic study established dilatations of anal canal veins uniformly found in the fetus, infants, and children, thus establishing that these venous dilatations were, in fact, a normal part of human anatomy and not representative of pathology.

Vascular Hyperplasia Theory

Anatomic findings suggested hemorrhoids were cavernous, erectile tissue or hemangiomatous tissue, thus suggesting the name "corpus cavernosum recti."[9] Arteriovenous communications were established in the anal submucosal, suggesting erectile properties which could conceivably enhance its function as part of a proposed anal continence mechanism by contributing to anal closure. It was proposed that hemorrhoids might result from hyperplasia of the "corpus cavernosum recti." While this theory may not be accurate, the discovery of arteriovenous communications was another important piece of the puzzle, explaining the bright red bleeding typical of symptomatic hemorrhoids rather than dark, venous bleeding which would otherwise be expected if this were purely venous disease.

Thomson's anatomic studies concluded that there is "neither histological nor circumstantial evidence to support the vascular hyperplasia theory."[9] Furthermore, the bleeding from hemorrhoids "seems to arise from capillaries in the lamina propria rather than the venous dilations."[12]

Infection/Inflammation Theory

The idea of an infectious etiology probably dates to Quenu and Hartman in the 19th century who introduced the idea of inflammation as the pathological cause of hemorrhoids.[10] Rankin and coworkers[13] and later Buie noted that "there is always an infectious process associated with hemorrhoids . . . a condition which may properly be termed 'phlebitis'."[14] This infection was thought to "come from outside the lumen of the vessels" to weaken the venous walls.[14] These factors when combined with the gravitational effects of erect posture, first were proposed by Morgagni in the 17th century,[2] "which normally they are able to withstand, but when they are weakened by phlebitis, hemorrhoids results."[14] Buie used findings of "infiltration with round cells, either diffusely arranged or in foci, will be observed in all specimens" with "occasionally large leukocytes" as histologic evidence of underlying inflammatory changes.[14]

Nesselrod confirmed that hemorrhoids are "vascular tumors made up of infected varices" and that "the principal cause of hemorrhoidal disease is anal infection."[15] It was reasoned that "infectious material from the bowel content having gained entrance to the perianal tissues via the anal crypts, the anal ducts, the anal glands, and their associated lymphatics, it can readily attack the thin-walled venous structures comprising the hemorrhoidal plexus."[15] He quotes Buie's idea that the "vulnerability of the anal crypts must be considered as the surgeon seeks the portal through which the infectious process gains admission . . . there always is an infectious process."[15]

Interestingly, it was only after further anatomic studies that this erroneous suggestion, that hemorrhoid disease is due to an infectious process that weakens thinned-out venous walls leading to hemorrhage, was able to be put to rest. To the contrary, pathologic studies exposed an opposing viewpoint of vessel wall thickness in diseased hemorrhoids. In fact, compensatory thickening of the venous walls was revealed in the diseased state.[16] Jackson and Robertson formally concluded that "the infectious theory was unfounded" in a presentation at the joint meeting of the American Proctologic Society (now known as the American Society of Colon and Rectal Surgeons) and the Section of Proctology of the Royal Society of Medicine in Philadelphia in 1964.[11]

Sliding Anal Lining Theory

Original observations on "4000 private patients" in Chattanooga, Tennessee over a 4-year period by Gass and Adams in 1950 helped turn the tide away from the still popular theory of varicose veins toward a new understanding based on microscopic pathology. The "outstanding feature" of these detailed microscopic sections from pathologic specimens taken from 200 consecutive patients revealed the "loose and fragmented nature of the submucosal, collagenous, connective tissue stroma as the fundamental early lesion" in hemorrhoidal disease.[16] This suggested to the authors a "giving away of the supportive structures rather than a weakening and thinning out of the vessel walls as the initial pathologic change." In fact, "many of the vein walls showed a 'compensatory thickening'." They also reported "a striking absence of any inflammatory reaction," thus opposing the infectious theory of hemorrhoids. The findings supported the standard treatment of the era, which "depend on the correction of

herniation or protrusion and not on the obliteration of varicose veins, surgery or injection sclerotherapy."[16]

This short, simple study paved the way for further research and anatomical studies in support of this new direction. Jackson and Robertson confirmed the complexity of hemorrhoids, which includes the "rectal mucosa and submucosa, including muscularis mucosa, terminal branches of the superior hemorrhoidal artery and veins and connective tissue with varying degrees of elastic tissue."[11] This anatomic study showed a "high degree of elastic tissue seen in infants and older children, this supportive type of tissue fragments and gradually degenerates into a state of near 'nonexistence' in hemorrhoidal disease."[11] They "believed the cause of hemorrhoids is due to the gradual reduction of supportive elastic tissues due to aging and abetted by the daily trauma of straining at the time of a bowel movement."[11] This decrease of elastic tissue is said to be "consistent with similar changes occurring during advancing years in the tissues of other organs such as the aorta, skin and so forth."[11] They also implicate "our rich, low-residue diets," which "often necessitate prolonged straining of the abdominal and pelvic musculature," raising intrarectal pressures (from 20 to 200 mm Hg) for prolonged periods of time.[11] Burkitt also pointed out the role of the low-fiber diet in the pathogenesis of hemorrhoid disease.[17] Chronic constipation with the associated prolonged and repeated straining to evacuate hard stools will further exaggerate hemorrhoid protrusion.

The anatomy and function of the perianal connective tissue "system" was further delineated in anatomic studies by Haas and Fox, who labeled the conjoined longitudinal coat as the most important element of this "fibroelastic network."[18] They were unable to find elastic fibers in children under the age of 2, when they begin to appear. In the young, these connective tissue fibers are thinner and less numerous than in adults. They devised a connective tissue (C) to muscle tissue (M) ratio or C/M ratio. Although "there are overlappings in the older age groups and one cannot estimate the age of a person from the C/M ratio, but it is certain that as a person grows older the amount of the muscle element decreases and the amount of connective tissue elements increases."[18]

As in other anatomic studies, they noted that "hemorrhoidal vessels are present in the newborns but the surrounding connective tissue fibers are solid, firm, and unbroken."[18] In adults, the connective tissue fibers were "thick, loose, disintegrated and broken." Blood vessels under the mucosa form the bulk of hemorrhoids and these connective tissue fibers support the "cushions." In the young, the connective tissue fibers are "dense, well organized and parallel to each other."[19] Haas and coworkers found that "around age 30, the connective tissue fibers start to disintegrate; they loosen around the blood vessels, and the veins become distended."[19] In older age groups this anchoring connective tissue system degenerates and the fibers are disintegrated, weak, and broken loose. The hemorrhoid cushions detach from the internal sphincter and slide downward, bulging into the anal canal. This work further supported the findings of Gass and Adams that the "outstanding feature of the sections studied was the loose and fragmented nature of the collagenous connective tissue stroma which showed a loss of its usual compact and even staining architecture."[16] Jackson and Robertson reported similar findings of tissue fragmentation in patients with hemorrhoidal disease and they blamed the daily trauma of straining at defecation for this connective tissue breakdown.[11] Strehler also found that fibers and cells of connective tissue become disorganized with advancing age.[20] This, along with age-related changes in collagen synthesis and collagenase function, may all contribute to the degradation of native collagen over time. According to Haas and coworkers, this aging theory explained the deterioration of the anchoring connective tissue, which allows the eventual slide of the anal cushions and seemed to fit better than a "wear and tear" theory.[19] This theory also explains observations by some that "hemorrhoids are inevitably progressive."[21]

Vascular Anal Cushions

The year 1975 was a watershed year for studies regarding the origin and explanation of hemorrhoids. This was the subject of Thomson's thesis for his Master of Surgery degree. By pooling the data from previous studies along with original anatomic studies, he was able to support and better articulate the "sliding anal lining theory"[16] and used simple terms such as "cushion" to describe the complex nature of hemorrhoids.[9] Also in 1975, Alexander-Williams and Crapp used the term "vascular anal cushions" in a review of management options.[22] The term "cushion" has remained in the lexicon as it is simple yet accurate in its description of a complex intertwining network of arteriovenous communications, muscle, and connective tissue elements.

Among other findings, Thomson was able to dispel the notion that the three cardinal locations of hemorrhoids (right anterior, right posterior, and left lateral) were due to the termination of the superior hemorrhoidal (rectal) artery. "In summary the anal lining derives a rich blood supply from the superior, middle and inferior rectal arteries, whose branches reach the anal submucosa in a most varied way."[9] Specimens injected with red latex from the arterial side under microscopy were found to fill "some of the venous dilatations by way of tiny direct arteriovenuous communications."[9] These findings proved the existence of "arterio-venous anastomoses . . . previously postulated by the histological studies of Staubesand et al (1963)."[23] It also supported the work of Thulsesius and Gjores who in 1973 studied the oxygen content of hemorrhoids in normal subjects and found that "the consistent finding of high oxygen contents in anal blood (mean 98%) clearly indicates that AV-shunts are a normal feature, thus supporting previous anatomical evidence in favour of this view."[24]

Besides these arteriovenous communications, he was able to demonstrate free communication between the tributaries of the superior, middle, and inferior rectal veins in all 25 injected specimens. These "free communications" were noted both through and below the anal sphincter to join the subcutaneous tributaries of the inferior rectal vein, thus demonstrating portosystemic communications.[9] This system allows a natural "escape valve" for an abnormal elevation in portal vein pressure (portal hypertension). This explains the findings that hemorrhoid disease is not a more frequent finding in patients with portal hypertension.[1]

The anatomical dissections demonstrated that the thickened submucosa (cushions) on histology were indeed "composed largely of blood vessels (the venous dilatations making the main contribution), smooth muscle (Treitz's muscle) and elastic and connective tissue."[9] The clinical portion of the article revealed that "the majority of patients (60%) claimed that prolapse had been the first symptom." Only eight patients (12%) thought that "bleeding had started first."[9] He was also able to dispel the notion of vascular hyperplasia as there was no sign of this in any specimen. In fact, the normal cadaver specimens were similar to the hemorrhoidectomy specimens.

Furthermore, the study demonstrated "that the submucosa does not form a continuous ring of thickened tissue in the anal canal but a discontinuous series of cushions, the three main ones being found constantly in the left lateral, right anterior and right posterior positions."[9]

He invalidates the varicose vein theory by revealing normal venous dilatations in cadaver specimens along with the fact that anal varices appear quite different from hemorrhoid disease. He finds "neither histological nor circumstantial evidence to support the vascular hyperplasia theory."[9]

Thomson reasons that if the cushions contribute to the mechanism of anal closure, then this may explain "the high percentage of people who experience an impairment of continence after haemorrhoidectomy."[9] His proposal that "Treitz's muscle might become stretched and disrupted if repeatedly subjected to such forces, intermittent and then permanent prolapse of the cushions resulting"[9] was later confirmed by

the work of Haas and coworkers.[19] The final conclusion that "the available evidence suggests that the theory that piles are nothing more than a sliding downwards of part of the anal canal lining is probably the correct one" remains simple, direct, and to the point.[9]

Contributions of Anorectal Physiology

The "French schools" are credited with advocating rectal bouginage for hemorrhoids and early practitioners of this art included Copeland and the founder of St. Mark's hospital, Frederick Salmon.[25] They believed that a hypertonic anal sphincter was present in these patients and that a degree of anal stenosis was at least part of the cause of hemorrhoids so bouginage was advised as conservative treatment.

Abnormalities of anorectal physiology can be demonstrated in patients with hemorrhoid disease. It is of interest that the extensive contributions of the article by Gass and Adams to the understanding of the nature of hemorrhoids also included an opinion of the role of sphincter tone in hemorrhoid disease that ran counter "to the popular opinion of both the profession and the laity. The more atonic the anal sphincter the greater the protrusion, herniation or hemorrhoids."[16] They go further and point out that "in our experience hemorrhoids seldom develop in the constipated individual with a spastic or hypertonic sphincter muscle."[16] It took the advancement of technology to accurately measure such parameters as anal canal pressures to prove these statements wrong. Almost every published anorectal physiology study shows an increase in resting anal pressures in patients with hemorrhoid disease.[26]

Many have pondered whether this increase in resting pressure is the cause or the result of pathological hemorrhoids. Rubber band ligation results in only an insignificant decrease in anal pressure without a return to normal pressures. This is in contrast to a prompt return to normal anal canal pressures usually 3 months following hemorrhoidectomy. Since the pressures return to normal after surgery, it is reasoned that the increase in resting anal canal pressures initially must be a result of the hemorrhoid disease and not the cause of it.[12] In a typical study, Sun and coworkers blame "hypertensive anal cushions" as the cause of the high anal canal pressures in hemorrhoid patients.[27]

Anorectal manometry has provided the means to investigate the role of sphincter pressure in the pathogenesis of hemorrhoids. El-Gendi and Abdel-Baky documented significantly high anal pressures in all patients with symptomatic internal hemorrhoids. One week following surgery, the high pressure was significantly reduced.[28] Ho and coworkers also noted a significantly higher resting anal pressure. Following hemorrhoidectomy, they found significant decreases in both maximum resting and maximum squeeze pressures to within normal values 3 months after the operation. Rectal compliance also increased significantly to that of normal controls.[29] They concluded that, since these hemorrhoid patients had anorectal physiologic abnormalities which reverted to normal within 3 months of hemorrhoidectomy, these physiological changes are more likely to be an effect, rather than the cause of symptomatic hemorrhoid disease. Furthermore, ultraslow wave activity found in patients with hemorrhoid disease was no longer identified following hemorrhoidectomy. Moreover, "since the physiologic abnormalities returned to normal after operation, attempts to lower anal sphincter pressure by internal sphincterotomy as an adjunct to hemorrhoidectomy are unnecessary and carry the risk of incontinence in susceptible patients."[29]

In an early study of sphincter pressure in 1977, Hancock found "greater activity of the internal sphincter" in hemorrhoid patients with a highly significant increase in maximal anal pressure and presence of ultraslow waves. There was also a trend to lower anal pressures with increasing age in normal controls and this trend was greater in hemorrhoid patients reaching statistical significance.[30]

Lin also finds "high basal anal sphincter pressure" in a Chinese study but found that the squeeze pressure of the hemorrhoid patients was not significantly higher than that of the control population, drawing the conclusion that the high pressure was "mainly due to overactivity of the internal sphincter muscle."[31] Sun and coworkers blame the "increased vascular pressure in the anal cushions" for the abnormally high pressure in the anal canal in hemorrhoid patients.[27]

To the contrary, Waldron and coworkers find "evidence of increased external sphincter function during waking and sleep" that have implications in the pathophysiology of hemorrhoids.[32] They also demonstrated a greater degree of sampling response, irregular slow wave activity, increased ultraslow and giant ultraslow waves, normal rectal contractile activity, and normal anal sphincter response to a prolapsing mass in the anal canal.[32]

Normal Function of the Anal Cushions

In 1928 Stieve[33] showed that, on its own, the internal sphincter cannot completely close the anal canal.[34] The addition of mucosal folds is still not enough: filling of the vascular spaces is also necessary and contributes 15 to 20% to resting anal pressure.[35] Therefore, the anal cushions "act as a compliant and comfortable plug"[26] and "help to preserve continence by forming an expansile anal seal."[27] Haas and coworkers agree that the cushions "contribute to the mechanism of anal closure."[19] They assist in "anal closure, providing perhaps the capacity for fine adjustment of continence," thus contributing to the "continence mechanism."[36] Moreover, this may explain why surgical removal of these cushions (eg, hemorrhoidectomy) "is often associated with minor degrees of incontinence and seepage."[37]

Factors Predisposing to Hemorrhoidal Disease

The following are factors which may or may not predispose to the diseased state of hemorrhoids.

Heredity

Given the explosion of genetic research which has provided inroads for explaining many diseases and disorders of the human condition, it would not be surprising that a "hemorrhoid gene" or multiple "hemorrhoid genes" may one day be localized somewhere in the genome. However, there has been no such breakthrough, to date, to prove such a genetic component. Loder and coworkers note that "a positive family history is common."[26] There is clearly a predisposition in some families where members can recite a lengthy list of relatives with hemorrhoid disease.[3] However, such a strong family history may be explained by more important shared factors such as "dietary, cultural, behavioral and other environmental factors rather than genes."[26] Burkitt and Graham-Stewart note that a "similar situation in black and white Americans in contrast to that in Africa and the evidence of increasing prevalence following a change in life patterns, indicate that heredity is not a primary causative factor."[38]

Anatomic Factors

As much as the absence of valves in the portal venous system together with gravitational pressures would lead one to conclude that the upright posture of humans predisposes to hemorrhoid disease, this is not a cause-and-effect relationship. The hemorrhoids are richly vascularized by multiple vessels and similarly there is a corresponding vast venous outflow back to both the portal and the systemic venous circulation. There is a rich communication between the portal and systemic circulation even allowing a safety valve or vent for elevated portal vein pressures through the systemic veins via the middle and inferior rectal veins. Rectal varices do indeed exist (although very rare); however, they have a much different appearance from hemorrhoid disease.

Straining at Stool

It would seem reasonable to assume, given the present understanding of breakdown and disintegration of support structures leading to hemorrhoid disease, that either the trauma of extended straining to pass a hard, fecal bolus (constipation) or the repeated trauma of passing explosive stools several times a day (diarrhea) may predispose to hemorrhoid disease. Given enough time, breakdown of supporting structures with progressive prolapse can be expected.

Age

Contributions from various authors have demonstrated that support is lost over time as muscle mass (M) diminishes and is replaced by connective tissue (C), allowing sagging of the cushions and eventual protrusion. Haas and Fox referred to a C/M ratio, and so an increase in the C/M ratio occurs with age and downward displacement of hemorrhoids or prolapse becomes more prevalent over time.[18]

Occupation

Paradoxically, Gabriel notes that both "men who do heavy manual work" and "sedentary workers who take unaccustomed exercise while on holiday, or may have been too enthusiastic in their garden" are liable to develop hemorrhoid symptoms.[39] In other words, there is no known association with a given occupation. Rankin and coworkers note that "few led sedentary lives."[13]

Socioeconomic Status

According to Ebstein, "hemorrhoids are particularly frequent among the better classes, less frequent among people of a sober and active life."[3] The increased prevalence in more industrialized, "sophisticated" societies may merely represent the Western, "refined" diet, which is a low-residue diet often contributing to known predisposing factors such as constipation and straining to stool.

Nutrition

There is no evidence to suggest a relationship between hemorrhoid prevalence and nutritional status.[38]

Sex

Some studies suggest a predisposition among men. Gabriel notes "women not infrequently develop an attack of hemorrhoids from spring-cleaning, moving house, or the prolonged efforts required in the nursing of an incapacitated husband or an aged relative."[39] Although studies exist to suggest either a male or a female predominance, the condition may be seen in either sex in equal proportions.[13]

Level of Activity

Both active exercise and sedentary occupations have been blamed. "Neither suggestion gains epidemiological support."[38]

Prolonged Standing

Occupations including "drivers of trams, conductors of public vehicles, postoffice sorters, shop-walkers, and the like seem very prone to develop haemorrhoids after reaching middle age."[39] Again, "middle age" is more likely predisposing to the condition than the implied effects of prolonged standing.

Sphincter Relaxation

Gabriel notes "in old age or accompanying the debility after any severe shock or illness, there may be some loss of sphincter tone, and any previous slight degree of haemorrhoidal prolapse may become marked."[39] Most studies reveal an increase (not decrease) in sphincter tone as predisposing to hemorrhoid disease. Once again, "old age" is probably the most influential component in this scenario.

Abdominal and Pelvic Causes

Physiologic causes of hemorrhoid disease include pregnancy. In women, pathological causes of hemorrhoid disease may include uterine fibroids, uterine retroversion, large abdominal tumors, or ovarian cysts. Rankin and coworkers stated that the proposed role of malposition of the uterus in causing hemorrhoid disease was "probably unfounded."[13] Men with prostate enlargement or urethral stricture may be predisposed to hemorrhoid disease as a result of "habitual straining."[39] The typically advanced age of these patients will certainly contribute to progressive symptoms of prolapse.

Rectal Congestion

Gabriel used this term to suggest that "the person of plethoric build, the habitual over-eater, the subject of portal congestion, and increasing adiposity—these are all liable to haemorrhoids. People who do much riding on horseback, and men engaged in the tailoring trade, who habitually sit for many hours a day in the cross-legged position, are very prone to develop haemorrhoids."[39] There is no known correlation between body habitus, occupations with prolonged sitting, and especially horseback riding to support these claims.

Vascular Conditions Elsewhere (Varicosities)

Cleave, a surgeon captain of the British Royal Navy, suggests that the "mechanism" causing varicose veins of the leg and femoral vein thrombosis "is not fundamentally different that already accepted in the case of haemorrhoids."[40] He questions "is the body built wrongly, or is it built rightly but being used wrongly"? He goes on to implicate the "refined" low-residue diet as the cause of these maladies.[40] While this may be true, there is no demonstrable connection with these various forms of diseased veins and hemorrhoidal disease.

Acute Thrombosed Hemorrhoids

As much as the search for the truth regarding the nature and pathophysiology of hemorrhoids has been slowed by myths and legends, the special situation of acute thrombosis is even more shrouded in mystery.

The first hurdle to overcome was the "misconception of the pathological process."[41] In the early 20th century, infection was believed to be the cause of both the chronic diseased state of internal/external hemorrhoids and also the acute state of thrombosis. Nesselrod notes that "an external varix is one of several manifestations of anal infection. When the varix becomes thrombosed rupture of its wall frequently follows, allowing blood to extravasate into the adjacent subcutaneous tissue. Blood outside a blood vessel is highly irritating, and an acutely painful inflammatory condition is quickly established."[15]

Similar to the investigations of chronic internal/external hemorrhoid disease, histopathology proved the absence of an infectious process and also provided a glimpse of the actual underlying pathophysiology.

The next hurdle was the actual portrayal of the basic pathologic process occurring underneath the painful, swollen mass. Gross examination would suggest that this acute condition is "caused by

extravasation of blood into the tissue from a ruptured vein."[42] Nesselrod describes a "painful, bluish colored swelling. Due to edema and the extravasated blood, the overlying skin is tense and glossy."[15] This was initially considered a "perianal hematoma"; however, histologic evidence of a "true intravascular thrombosis" eventually emerged.[43]

In a 1971 retrospective study of 100 consecutive patients with pathologic diagnoses of thrombosed hemorrhoids, Ganchrow and coworkers discovered that "no 'perianal hematomas' were identified and that "all thrombi were intravascular ... and when the vein wall could not be clearly demonstrated in sections stained with hematoxylin and eosin, Mason's trichrome showed the smooth muscle outline of a vessel wall." They also concluded that "thrombosis of hemorrhoids has been recognized for years, but the thrombogenesis is still unsolved," although "it seems reasonable to conclude that the common denominators in these patients are stasis and local trauma."[43]

Regarding "trauma" as a predisposing factor, "approximately half of the patients in the prospective group were able to identify the dates of their initial thromboses by their association with acts such as dancing, lifting heavy objects, or straining."[43] Nesselrod and Oh find that the left side is more often involved than the right side of the anus.[15,44]

In 1979, Hamish Thomson also discussed "the well known but misnamed 'perianal haematoma' (or 'thrombosed external pile')" and suggested that "subpecteneal thrombosis might be a better name."[36] He recognized "the shiny intimal lining of the venous saccule in which it arose."[36] A few years later, in 1982, he stated, "I wish to challenge the usual pathological explanation attributing the lesion to a burst blood vessel. First, there is no bruising but a discrete 'berry' under the skin. Second, evacuation reveals a glistening white compartment. Neither characteristic is compatible with vessel rupture and haemorrhage" and he coined yet another new catch phrase "clotted venous saccule" and proposed that it be used as a substitute name for the condition.[45]

In 1989, Oh found acute thrombosis to be "relatively common in young active persons" between the ages of 20 and 40 with a preponderance of men (2:1).[44] While constipation is a "preceding condition in the majority of patients," diarrhea may also exist, although some patients will have no predisposing factors.[44] According to Nesselrod, "it may occur during or following a bout of acute diarrhea from any cause, following a drastic purgation, following the passage of a hard stool, or it may occur for no apparent reason."[15] There is agreement that the mechanism of thrombogenesis seems to be "stagnation of blood and trauma to the anal vessels due to strain."[44]

Not only is etiology obscure, but even surgical treatment options have evolved only over the published objections of respected and revered surgical specialists of the 20th century. In the first edition of his textbook, "Goligher (1961) sums up the position thus: 'The consensus of surgical opinion in this country enjoins a strictly nonoperative and expectant approach to prolapsed thrombosed piles. Treatment entails rest in bed, hot baths, soothing local applications, sedatives, aperients to secure easy motions, and antibiotics to control infection.'" Surgery is deemed inadvisable because of the alleged dangers of disseminating infection by operative interference in a septic field, thereby precipitating pylephlebitis, secondary hemorrhage, and stricture formation.[46] Although its origins were in a pre-antibiotic era (with the attendant fear of unrelenting/untreatable sepsis), "it is not clear how the fear of septic complications of operations of strangulated piles has become established."[46] Smith explains, "we have been taught over the years of the great danger of surgery at these times, and deaths from ascending infection and portal pyaemia have always been hinted at. The supposed dangers of surgery in these acute cases, however, exist largely on hearsay evidence, and a search of the literature does not bring to light many actual cases."[47] He defends emergent surgical intervention by pointing out that a case report from Lockhart-Mummery in 1934[48] "also, was a death from portal pyaemia, but it followed the conservative management of acute hemorrhoids."[47] This was one of many reports to rebuke the "classic, but unsatisfactory, conservative management" of acute disease.[47]

By 1964 the tide was turning, although published acceptance for emergency surgery lagged, "the practice of immediate operation for prolapsed piles is not original, but it continues to be ignored in standard textbooks and condemned on grounds that do not seem substantiated. It deserves to be more widely known and advocated."[46] Indeed, by 1984 Goligher had changed his stance and pronounced that "my own practice for over 20 years has been to advise immediate haemorrhoidectomy to the majority of these cases ... as for the risk of portal pyaemia, this seems to be largely a myth."[49]

There have been accounts of historical figures who probably suffered from some form of acute hemorrhoidal disease. One such account is from Martin Luther, at age 45, in a letter to Justus Jonas on January 6, 1528:

> "My affection was such that upon bowel movement the swollen lip of the anus protruded in the size of a wall-nut. On it was a small itching swelling (scabies minutula) of the size of a small hemp-seed. The troubles were more intense when the stools were soft. Whenever clotted blood was discharged, I was much comforted and relieved, yea, the act of evacuation was a pleasant sensation. The more clots were passed, the more pleasure I had, so much so that this pleasant feeling induced me to pass bowels several times a day. On pressing with my finger I had an exceedingly pleasant sensation and blood began to flow. This is why I thought that the bloody stools should not be stopped or diminished. This affection is called the golden vein (vena aurea) and golden it is indeed. For they say that with this cruor all the unsound substances are removed from the body, as if this here were the porta sterquilinii (heap of dung) for all evils, and that people with such an affection have a long life, as if here in this part were enclosed the entire pharmacy and all the doctores medicinae. The discharge takes place by its own will and at its proper time."

This would seem to be a very accurate description of the natural history of an acute thrombosed hemorrhoid. Indeed, Luther went on to spontaneous and "natural healing" with full recovery from his hemorrhoid troubles.[3]

References

1. Bernstein WC. What are hemorrhoids and what is their relationship to the portal system? *Dis Colon Rectum*. 1983;26:829–834.
2. Holley CJ. History of hemorrhoid surgery. *South Med J*. 1946;39(7):536–541.
3. Ebstein E. A contribution to the history of hemorrhoids and their treatment from the time of Hippocrates to the modern bacteriological era. *Med Life*. 1928;35:5–18.
4. Pruess J. Biblisch-talmudische Medizin. *Berlin S* 1911;212–214.
5. Smith M. Hemorrhoidectomy: past and present. *Dis Colon Rectum*. 1961;4:442–444.
6. *First Book of Samuel, Chapters 4-6. The New American Bible*. Charlotte, NC: CD Stampley; 1971:242–244.
7. Hyrtl I. *Die alten deutschen kuntsworte der Anatomie*. 1884 Wien S. 70f.
8. Johnson FP. The development of the rectum in the human embryo. *Am J Anat*. 1914;16:1–53.
9. Thomson WHF. The nature of haemorrhoids. *Br J Surg*. 1975;62:542–552.
10. Quenu E, Hartman H. Chirurgie du rectum. Paris: G. Steinhil; 1895:335–373.
11. Jackson CC, Robertson E. Etiologic aspects of haemorrhoidal disease. *Dis Colon Rectum*. 1965;8:185–189.
12. Hulme-Moir M, Bartolo DC. Hemorrhoids. *Gastroenterol Clin North Am*. 2001;30(1):183–197.
13. Rankin FW, Bargon JA, Buie LA. Hemorrhoids, in The colon, rectum and anus. Philadelphia: WB Saunders; 1932:624–666.
14. Buie LA. Hemorrhoids and rectal prolapse, in Practical proctology. (ed 2)Springfield, IL: CC Thomas; 1937:218–275.
15. Nesselrod JP. Hemorrhoidal disease, in Proctology in general practice. Philadelphia, PA: WB Saunders; 1950:69–100.
16. Gass OC, Adams J. Hemorrhoids: etiology and pathology. *Am J Surg*. 1950;79(1):40–43.
17. Burkitt DP. Varicose veins: DVT and haemorrhoids: epidemiology and suggested aetiology. *Br Med J*. 1972;2:556–561.
18. Haas PA, Fox TA. Age-related changes and scar formations of perianal connective tissue. *Dis Colon Rectum*. 1980;23:160–169.
19. Haas PA, Fox TA, Haas GP. The pathogenesis of hemorrhoids. *Dis Colon Rectum*. 1984;27:442–450.
20. Strehler BL. Time, cells, and aging. New York, NY: Academic Press; 1962:138.

21. Miles WF. Observations upon internal piles. *Surg Gynecol Obstet*. 1919;29:497–506.
22. Alexander-Williams J, Crapp AR. Conservative management of haemorrhoids. *Clin Gastroenterol*. 1975;4:595–601.
23. Staubesand J, Stelzner F, Machleidt H. Uber die "Goldenen Adern"—ein Beitrag zur Histophysiologie der sogenannten Glomerula venosa haemorrhoidalia. *Morph Jb*. 1963;104:405–419.
24. Thulsesius O, Gjores JE. Arterio-venous anastomoses in the anal region with reference to the pathogenesis and treatment of haemorrhoids. *Act Chir Scand*. 1973;139:476–478.
25. Parks AG. De haemorrhois: a study in surgical history. *Guy's Hosp Rep*. 1955;104:135–156.
26. Loder PB, Kamm MA, Nicholls RJ, et al. Haemorrhoids: pathology, pathophysiology and aetiology. *Br J Surg*. 1994;81:946–954.
27. Sun WM, Read NW, Shorthouse AJ. Hypertensive anal cushions as a cause of the high anal canal pressures in patients with haemorrhoids. *Br J Surg*. 1990;77:458–462.
28. El-Gendi MAF, Abdel-Baky N. Anorectal pressure in patients with symptomatic haemorrhoids. *Dis Colon Rectum*. 1986;29:388–391.
29. Ho YH, Seow-Choen F, Goh HS. Haemorrhoidectomy and disordered rectal and anal physiology in patients with prolapsed haemorrhoids. *Br J Surg*. 1995;82:596–598.
30. Hancock BD. Internal sphincter and the nature of haemorrhoids. *Gut*. 1977;18:651–655.
31. Lin J-K. Anal manometric studies in hemorrhoids and anal fissures. *Dis Colon Rectum*. 1989;32:839–842.
32. Waldron DJ, Kumar D, Hallan RI, et al. Prolonged ambulant assessment of anorectal function in patients with prolapsing hemorrhoids. *Dis Colon Rectum*. 1989;32:968–974.
33. Stieve H. Uber die Bedeutung des venoser Wunderneize fur den VerschluB einzelner Offnungen des menschlischen Korpers. *Dtsch Med Wochenschr*. 1928;54:87–90 130-133.
34. Stieve H. Uber den VerschluB des menslichen Afters. *Z Mikrosk Anat Forsch*. 1930;21:642–653.
35. Lestar B, Penninckx F, Kerremans R. The composition of anal basal pressure*: An in vivo and in vitro study in man*, *Int J Colorectal Dis*. 1989;4:118–122.
36. Thomson H. The anal cushions—a fresh concept in diagnosis. *Postgrad Med J*. 1979;55:403–405.
37. Gibbons CP, Trowbridge EA, Bannister JJ, et al. The role of the anal cushions in maintaining continence. *Lancet*. 1986;1:886–887.
38. Burkitt DP, Graham-Stewart CW. Haemorrhoids—postulated pathogenesis and propsed prevention. *Postgrad Med J*. 1975;51:631–636.
39. Gabriel WB. Haemorrhoids, in The principles and practice of rectal surgery. (ed 5) Springfield, IL: CC Thomas; 1963:110–164.
40. Cleave TL. Varicose veins: nature's error or man's? *Lancet*. 1959;2:172–175.
41. Stern W. Thrombosed haemorrhoids: immediate surgical treatment. *Med J Aust*. 1964;2:635–636.
42. Jackman RJ. The management of hemorrhoidal thrombosis. *GP*. 1956;14:82–86.
43. Ganchrow MI, Bowman HE, Clark JF. Thrombosed hemorrhoids: a clinicopathologic study. *Dis Colon Rectum*. 1971;14:331–340.
44. Oh C. Acute thrombosed external hemorrhoids. *Mt. Sinai J Med*. 1989;56(1):30–32.
45. Thomson H. The real nature of "perianal haematoma". *Lancet*. 1982;2:467–468.
46. Tinckler LF, Baratham G. Immediate haemorrhoidectomy for prolapsed piles. *Lancet*. 1964;2:1145–1146.
47. Smith M. Early operation for acute haemorrhoids. *Br J Surg*. 1967;54(2):141–144.
48. Lockhart-Mummery P. *Diseases of the rectum and colon*. London, Bailliere: Tindall and Cox; 1934.
49. Goligher J. Haemorrhoids or piles, in Surgery of the anus rectum and colon. London: Bailliere Tindall; 1984:98–149.