An Evaluation of the Cause and Mechanism of Complete Rectal Prolapse

NORMAN D. NIGRO, M.D.

Detroit, Michigan

I appreciate deeply the honor which the American Proctologic Society conferred upon me in electing me President a year ago. I accepted it with gratitude and I have found it a very pleasant and satisfying experience, due entirely to the fine cooperation of the officers and other members of the Society.

The office of the President has many benefits, not the least of which is the privilege of addressing members of the Society under conditions which, from the speaker's point of view, are ideal. He may select his subject, he may say more or less what he pleases without a time limit, and he has a receptive audience. This certainly provides a rare opportunity and I grasp it to discuss certain aspects of the subject, Complete Rectal Prolapse, a condition which poses a challenge to all who undertake its management.

COMPLETE rectal prolapse has attracted the attention of surgeons for a long time, but little progress was made in the understanding of it until the beginning of this century. Since then, it has been of special interest to some of our most oustanding surgeons. A review of their published reports reveals a striking variety of concepts and operations which deal with all phases of the problem.

An important development in the past decade is recognition of the diversity of rectal prolapse. It appears that the deficiency is not always the same and that this results in more than one type of rectal prolapse. This fact alone may well be responsible for a large part of the difference of opinion regarding the cause and mechanism of rectal prolapse, as well as the effectiveness of any single operation.

Some have proposed that the causative factor is a defect in the anatomy and physiology of the prolapsing bowel with a secondary deformation of the pelvic floor. An opposing view is that there is predominantly a defect in the pelvic floor accompanied by development of a peritoneal sac, such as occurs in a hernia. As I shall point out later, both views are correct and it is apparent that prolapse may be due to one of several mechanisms.

Early operations were directed solely at correcting the external portion of the prolapse. Later, procedures were devised to repair the various rectal supports and its musculature. Finally, abdominal operations were utilized to accomplish some of these objectives more effectively. A casual review of medical literature reveals that at least 50 operations have been accorded some degree of acceptance. Many have been discarded, but quite a few are still used. It is a significant fact that there is a tendency for some surgeons to use one operation for all types of prolapse.

It is not unusual to find opinions challenging the rationale and effectiveness of even the most popular operation. Therefore, I believe the subject of rectal prolapse

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is worthy of discussion, and it is apparent that special emphasis should be placed on a consideration of its cause and mechanism because the most rational surgical approach is one which deals with the precise defect presented by each individual patient.

Etiology and Mechanism

Aside from the rectum itself, the principal structure to be considered is the pelvic floor. This is largely a muscular partition, composed of the levator ani muscle which is enclosed in fascia. The levator ani muscle arises from the pelvic aspect of the pubic bone, from the arcus tendineus, and from the ischial spine. It is inserted into the bladder, the vagina (or prostate), the perineal body, the rectum, the coccyx and the sacrum. Although its function has been studied extensively, it is not generally understood.

A brief consideration of the evolution of the pelvic floor may help clarify its function in man.[8-10] Fish do not have a pelvic floor. Neither do they have a thoracic diaphragm or a muscular abdominal wall. These structures are not required by fish for respiration or locomotion, so the distal end of the gut in fish is not subject to strong pressures from muscular walls of the abdomen as are those of vertebrates living out of water. Fish do not have a rectal sphincter, and it is claimed that this is because it is unnecessary. With fish, the hydrostatic pressure of their habitat is ample to prevent emptying of the bowel, except when the gut itself contracts. Therefore, defecation occurs solely by pressure applied by the gut. Continence, if there is such a thing in a fish, is provided by the medium in which it lives.

A pelvic floor begins to appear in some advanced forms of marine life when they begin to move about out of the water. A rudimentary pelvic floor appears before the rectal sphincter. The salamander has the beginnings of a good pelvic diaphragm but no rectal sphincter, and, in animals higher in the evolutionary scale, the pelvic musculature is separated from the external sphincter. Even in apes, it is easy to separate the external sphincter muscle from the pubococcygeus. In man this is almost impossible. These facts may provide a clue to the relative importance of these two structures. At any rate, ecologists claim that the developments which occurred in response to the change in environment from water to air accounted for a pressure system in the two main body cavities. To accomplish this, two muscular partitions were evolved, namely the anterior abdominal wall and the thoracic diaphragm. The posterior abdominal wall in vertebrates is a rigid structure. Therefore, to complete the system, it was necessary to have a strong, muscular floor which could act synergistically with the other two walls to create the required degree of pressure. The pelvic floor, therefore, developed in conjunction with the other two muscular partitions to provide a mechanism for creating pressure changes to help such functions as breathing, blood circulation, and locomotion.

An additional factor came into play when the quadruped evolved into a biped. The erect position of man obviously creates new pressures upon the pelvic floor. The stimulus created by the change was substantial because its response included both an increase in the strength of the pelvic floor and a decrease in the load applied to it from above. The function of the pelvic floor was augmented by adaptation of the tail muscles of the quadruped.[13] The impact on the pelvic floor was diminished by a shift in direction caused by development of deep vertebral curves and a forward tilt of the pelvis. The ape has a spine which is much straighter than man's, and his pelvis is almost vertical. Consequently, pressure from above is applied more directly on the pelvic floor, while in man it is applied to the lumbar spine, to the lower abdominal wall, to the pubic bone, and indirectly to the pelvic floor. In newborn infants, this

factor is not fully operative (Fig. 1). The situation is similar to that of the earlier primates and the pelvic floor is more vulnerable. This explains the greater incidence of prolapse in infants and also its spontaneous regression with their growth and development.

If there were no problems other than that of support, the function of the pelvic floor would be relatively simple. But the pelvic floor, like the roof, must have openings for the passage of certain structures and, in this regard, it is obvious that the floor is subjected to by far the greatest demands. It must provide functional openings for three important pelvic organs.

Since we are focusing our attention on the rectum, we will consider how the pelvic floor supports it while, at the same time, allowing it to empty periodically. There are three factors involved and Maes and Rives,[4] long ago, referred to them as passive support, conformation and position, and active support. Passive support is that which is provided by the various rectal connective tissue attachments; conformation is the support based on skeletal factors and the geometric pattern which the rectum assumes as it descends through the pelvis. Active support is that which is provided by the pelvic floor itself.

In the past, considerable importance has been ascribed to the passive supports of the rectum. Some have regarded their strength as the major factor in maintaining the rectum in its normal position. Experience does not support this view. If these supports alone maintain the rectum in place, they must necessarily be under the continuous tension caused by persistent but varying degrees of pressure coming from above. A number of experimental studies have been performed which show that when force is applied continuously for prolonged periods of time, fascia always yields. A good example of this is the linea alba of a pregnant woman. I believe it is safe to say that ligamentous structures alone are not adequate to hold the rectum in position continuously for prolonged periods.

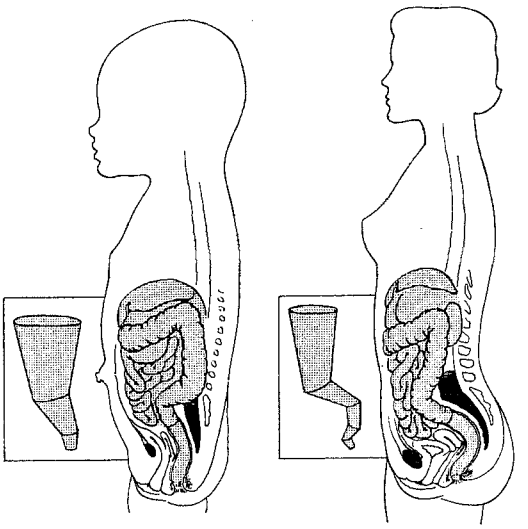

FIG. 1. Influence of skeletal changes on function of pelvic floor. *Left:* Infant with straight rectum, sacrum, and pelvis. *Right:* Adult with rectal and sacral curves; pelvis is tilted forward 45°.

Conformation refers to the stabilizing effect which the normal human spine and pelvis have on the rectum. The vertebral curves and the tilt of the pelvis cause the rectum to follow an undulating course through the pelvis. All of this is a part of the important skeletal mechanism which reduces the load on the pelvic floor.[15]

Active support refers to the mechanism by which the levator ani muscle provides an effective floor, while, at the same time, providing an adequate opening to permit defecation.

As I have indicated earlier, the levator ani muscle arises from the anterior and lateral walls of the pelvis, is inserted into the pelvic organs and into the lower end of the vertebral column. However, there is a small deficiency in the floor in the midline anteriorly which is triangular in shape. Its base is directed forward and is formed by the pubic arch. The sides converge posteriorly and are formed by the medial, free borders of the levator ani muscles. The muscle forming these medial borders is

thick, and the two sides join posteriorly behind the rectum, suspending it to the pubic arch. Furthermore, there is a firm attachment between the rectum and this muscular sling, owing to an extensive interweaving of the longitudinal fibers of the rectum with the more or less horizontally-placed fibers of the levator ani muscle. This extends over a considerable surface along the long axis of the rectum, thus providing a secure attachment.

The sling portion of the levator musculature, which is called the puborectalis muscle, functions by elevating the lower portion of the rectum in a forward direction toward the pubic arch, compressing the structures in front of it to decrease the aperture in the anteroposterior diameter. This action is supportive. It results in a more complete floor upon which the lower rectum, with its contents, rests.[1] Relaxation of the sling causes a descent of the pelvic floor with an increase in the size of the pelvic aperture. In this position, the rectum is more vertical and its lumen is situated more directly over the opening in the pelvic floor.

We have just described the two extremes of the position of this muscle, but its fibers are in a continual state of reflex activity, undergoing constant changes in all degrees of tension in response to variations in pressure from above. In general, the muscle contracts to provide resistance as it does, for instance, while the subject is coughing or lifting. The exception occurs when the muscle relaxes to permit evacuation of the rectum. A brief description of the mechanism of defecation will serve to illustrate this.

When the sensory cortex is stimulated by a pressure sensation in the lower rectum, a voluntary effort to defecate may result. It is begun by contraction of the anterior abdominal wall and the thoracic diaphragm, which increases the intra-abdominal pressure. At the same time, the levator muscle is inhibited, the levator sling lengthens, and the pelvic floor descends, obliterating the anorectal angle. The external sphincter muscle, which functionally forms a single unit with the puborectalis sling, relaxes at the same time. The rectum now occupies a more or less vertical position and the fecal mass is extruded by pressure from above, combined with contraction of the rectum. At this point, while the sling is open, the rectum is held in position only by its attachment to the relaxed levator ani muscle and by the various ligamentous structures attaching the rectum to adjacent structures. After defecation, the levator sling returns to its usual support position, relieving tension on the ligamentous tissues.

From these observations, it is obvious that an interplay of forces is exerted on the pelvic floor. The pressure from above is the sum total of several factors. One is pressure caused by contraction of the anterior abdominal wall and the thoracic diaphragm. Another factor is the weight of the abdominal and pelvic viscera. As I have pointed out, the abdominal viscera are supported, in part, by skeletal factors. However, there is some effect on the pelvic floor and it probably varies considerably with the build of the patient. Naturally, the weight of the pelvic organs imposes more work on the pelvic floor, and the muscular contractions of the colon itself create an impact upon it of varying degree. In opposition to the pressures from above are the retaining factors, which we have already discussed. There is a balance between these two opposing forces which is very broad because of the wide margin of safety with respect to rectal stability.

Obviously, some increase in pressure from above can be tolerated and, at the same time, some deficiency of the supporting mechanism of the rectum can be present without the occurrence of prolapse. If the increased pressure applied from above is strong enough, or if the supporting mechanism is significantly weak, prolapse will occur.[7] However, it is probable that pro-

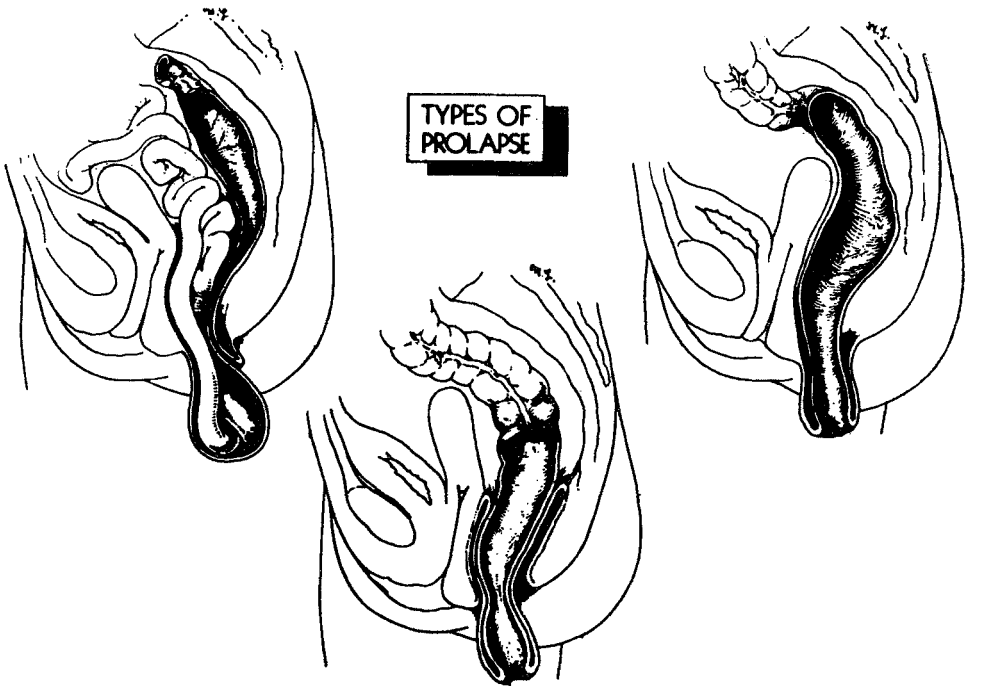

FIG. 2. *Top left:* Prolapse with deep cul-de-sac (anterior defect). *Bottom center:* Colonic intussusception through anus. *Top right:* Prolapse due to simple eversion (posterior defect).

lapse results more commonly from a combination of these two conditions.

Speculation regarding the nature of the changes which may cause prolapse are best made by observation of the appearance of the prolapse and clinical findings.[12] Such studies suggest that there are three varieties (Fig. 2) or types of complete rectal prolapse; namely, prolapse of a colonic intussusception, prolapse containing a deep cul-de-sac, and prolapse due to rectal eversion.

Prolapse of a colonic intussusception occurs rarely. It is a protrusion of a segment of colon, usually a portion of the sigmoid flexure, through the anus while the rectum remains in its normal position. This type is easily identified by a deep sulcus around the protruding mass, which indicates that the rectum is not everted. Often such a prolapse does not occur with every bowel movement and there is a tendency for the prolapsed portion of the bowel to become incarcerated. This is explained by the fact that the pelvic floor is normal or nearly so, and the descent must occur when the pelvic sling is relaxed. If the condition persists, the pelvic floor may become involved secondarily.

Prolapse associated with a deep cul-de-sac is the type described by Moschcowitz[5] as a sliding hernia through the anterior rectal wall. In its early stages, it can be recognized by inspection. The anterior wall of the protruding segment is much longer than the posterior wall and the pelvic sling retains a significant degree of function which can be demonstrated by digital examination or by x-ray studies. If the prolapse persists, the sling may become so weakened secondarily that support of the posterior wall may be lost. The defect then becomes a mixed type with a large protruding mass containing a peritoneal sac and loops of small intestine. The presence of the small intestine in the protruding mass can be shown by x-ray studies after oral ingestion of barium.

In my experience the third type is the most common, and it appears to be a simple eversion of the rectal wall. There is no peritoneal pouch. From its inception the protruding mass is completely circular, the walls are equal on all sides, and there is no sulcus around the prolapsed portion. The levator sling is defective early, a fact that can be demonstrated by digital examination of the rectum while the patient contracts the sphincteric mechanism. X-ray studies are difficult in these patients because many are incontinent.

This brings us to the question of incontinence in rectal prolapse. Often the two are associated and there has been much speculation as to whether incontinence is a cause or the result. Experience suggests that it is the result. Infants with rectal prolapse recover completely with development and show no signs of incontinence. Patients who have a prolapse with a deep peritoneal sac have good control during the early stages of the condition. However, as the prolapse increases in size, continence becomes progressively less adequate. In the type of prolape due to rectal eversion, incontinence is an early feature and its degree is proportional to the severity of the prolapse. In many patients, depending on the severity of the prolapse, continence improves to some degree after successful fixation of the rectum. Efforts to discover neurological changes have failed. Therefore, evidence supports the idea that incontinence is due primarily to muscle insufficiency.

I would like to return now to the question of the specific etiology of rectal prolapse. As I have indicated, rectal prolapse occurs when there is insufficiency of the pelvic floor, due either to a marked increase in its work load or to a reduction in its support capacity. Apparently it is often a combination of the two, and it is obvious that precise changes in any given patient may be difficult or even impossible to determine. Significant questions are: Why do only a few infants have rectal prolapse when it is acknowledged that their pelvic floors are vulnerable? Do some infants cry much more than others, or is the supporting mechanism weaker in some? Is it a combination of the two? Admittedly, answers to such questions are not easily obtainable.

Congenital anomalies contribute significantly to the causation of insufficiency of the pelvic floor. There may be anomalous connective tissue attachments of the rectum —an anomaly which I, as well as others, have seen is the suspension of the rectum by a free mesentery extending all the way to the anus. Skeletal anomalies involving the vertebral column and the tilt of the pelvis may be very important contributing factors. This is a subject which I intend to investigate. Anomalies of the levator ani muscle may be important causes of rectal prolapse. It is well known that the muscle is complicated and it is derived from several sources so that opportunities for anomalous development are prevalent.

The high incidence of prolapse in mentally ill patients is well recognized. It is known that they have a tendency to strain excessively, for long periods, and often this is done while forcibly relaxing the levator sling. When this occurs in a person who has only a minor degree of weakness of rectal supports, rectal prolapse may ensue.

Colonic protrusion through the normal rectum may be initiated by a marked increase in peristaltic activity in the sigmoid flexure, due to a large polyp or to severe degrees of constipation. Such causes are not in themselves sufficient to produce a prolapse, but in infants, or in the aged, this may happen. This is because the supporting mechanism may be deficient enough to be incapable of coping with the added burden.

There are two known causes of pelvic-floor inadequacy which result in prolapse. One is an extensive injury to the puborectalis sling, which often is surgical, and the other occurs in the cauda equina syndrome.

The neurologic defect causes paralysis of the levator muscle and prolapse almost always follows. As a matter of fact, prolapse can be produced in animals by sectioning the cauda equina.[14]

Many operations are available for the management of complete rectal prolapse. All are deficient to some extent, if a successful result requires full restoration of both structure and function. However, although such a degree of success may be unobtainable, from a practical point of view, acceptable results are nearly always possible with treatment having a more limited objective. One reason is that many patients are old, and demands are minimal as far as long-term results are concerned. Consequently, simple, palliative measures often may provide a satisfactory measure of relief. Often infants require only minimal therapy since the condition resolves spontaneously as they grow older. However, the problem is quite different in middle-aged patients who are physically active and have a significant life expectancy.

Specific operations performed, with the objective of obtaining long-term results, may be divided into two groups depending on whether they are designed to reduce the pressure on the pelvic floor or repair the floor itself. A careful appraisal of the principal deficiency in any given case should lead to selection of the appropriate procedure or combination of procedures. For example, pressure on the pelvic floor may be lessened by removing a markedly redundant portion of the colon, excision of a large peritoneal sac, and occasionally by removal of a large polyp of the sigmoid flexure. This pressure may also be decreased by attaching the rectum to the sacrum as illustrated by the operation of Ripstein and Lanter,[11] or by resection of the unstable portion of the rectum either from above or below. Correction of a relaxed uterus may also help.

Surgical repair of the pelvic floor has been used for years and, in this manner, small or moderate deficiencies can be repaired effectively. On the other hand, larger defects cannot be closed successfully by suturing the muscle because of insufficiency of muscle tissue. Opportunities to repair the levator muscle effectively are encountered frequently in patients with small prolapses through the anterior rectal wall. Usually this defect is situated chiefly anterior to the rectum and the muscle can be repaired either from above[3] or from below. In general, this type of repair is only one step in the complete procedure, but it is important in patients with this type of prolapse. If the prolapse is far advanced and the defect is very large, this step is of no avail.

In patients with prolapse caused by eversion of the lower rectum, the principal defect is in the puborectalis sling. Often this occurs early and is so extensive that repair by simple suture is not ordinarily successful. In this instance, utilization of a fascial or Teflon sling to suspend the lower end of the rectum forward to the pubic bone provides an effective means of support.[6] A piece of fascia lata or Teflon, measuring 3 by 25 cm., is placed across the lower portion of the rectum through a transcoccygeal incision. The midpoint of the graft is attached securely to the posterior rectal wall, just above the external sphincter. The two ends of the graft are placed around the rectum as far forward as possible. A retropubic extraperitoneal incision is made, and the two ends are sutured to the posterior aspect of the pubic bone. This procedure fixes the rectum and, at the same time, angulates its lower end upward and forward to support the rectum in a manner similar to that of the normal puborectalis sling. However, the artificial sling is rigid and can provide support only. It cannot elongate, as does normal muscle, to allow easy defecation. These patients, therefore, have difficulty initiating defecation, but this can be managed satisfactorily and they are pleased with the feeling of support provided by the sling.

A Thiersch wire should be inserted whenever the external sphincter muscle is markedly relaxed. Since this happens frequently, the wire is a useful adjunct in many operations performed to relieve prolapse. I use it routinely in conjunction with placement of a sling around the lower rectum. It provides support at a lower level, just as the sphincter muscle adds support to the puborectalis sling. Application of the Thiersch wire alone is an incomplete procedure. Nevertheless, it is useful in controlling prolapse in very old patients who are in poor general condition. It is a procedure which provides a surprisingly good palliative effect.[2]

The muscles supporting the rectum can be strengthened to some degree by exercising the sphincteric mechanism after rectal fixation. Improvement, however, is significant only when the prolapse is small. When the muscle deficiency is large, it does not improve function.

Recently, electrical stimulation has been used and, while there have been conflicting reports regarding its effectiveness, the method, as an adjunct to surgery, deserves further consideration.

Summary and Conclusions

Complete rectal prolapse is caused by insufficiency of the pelvic floor which may be due to an increase in the force applied to it from above, to a defect in the floor, or to a combination of the two. Both may be either acquired or congenital. Apparently prolapse is often caused by a combination of both stress and weakness of the pelvic floor. The anatomic defect is not always the same. In fact, I believe that there are three types of insufficiency of the pelvic floor and this is of major importance in planning surgical procedures.

Because of the age and condition of the patient, treatment of rectal prolapse is sometimes restricted to simple measures of palliation. Utilization of a Thiersch wire around the anus is a simple and effective method for such cases.

The objective in patients with acceptable surgical risks should be to correct the particular kind of deformity encountered in each individual patient. Today a number of successful surgical procedures are employed to repair the various types of defects.

Finally, it appears that incontinence in patients with rectal prolapse is due to a progressive muscular failure. Therefore, it is obvious that restoration of function is dependent not only on adequate fixation of the rectum, but also on the degree of muscle destruction encountered. Consequently, complete functional and anatomic restoration depends, to a considerable degree, upon early treatment.

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