SUPPORT OF THE PELVIC VISCERA

AND THE MECHANISM OF PROLAPSE

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INTRODUCTION

IN a previous paper,[1] after reviewing briefly the lack of value of the other ligaments as supporting structures to the uterus, it was shown by histologic section on autopsy and operative material, that there was no ligamentous tissue in the base of the broad ligament around or near the uterine vessels and, extending from the uterus out to the lateral pelvic wall, as described in some texts under the name of the Mackendrodt, the cardinal, or the utero pelvic ligaments.

It was shown that the uterus cannot depend for support on any structures to be found in the base of the broad ligament, any more than it can on any other and all the tissues to which it is attached. Therefore the explanation of the development of prolapse of the uterus can no longer include a consideration of these so-called ligaments. There remain only two other possible causative factors: (1) pressure from above, both intra-abdominal and intrapelvic, and (2) lack of support from below. If attention is directed only to the descent of the uterus it is very likely that, as has happened in the past, much of importance will be missed because the descent of the uterus is merely one symptom of an underlying lesion. Attention should be focused on the fact that there is a bony outlet at the bottom of the pelvis and that unless some adequate means for closing it is provided, the visceral contents of the pelvic and abdominal cavities will be extruded by the combined forces of gravity and intra-abdominal pressure.

The bony outlet is completely closed by the levator ani muscle excepting in its anterior median portion. Here there is a deficiency for the passage of the urethra, vagina and rectum, called the pelvic floor aperture. It is this aperture caused by a deficiency in musculature about which the whole problem of prolapse centers. If there were no such deficiency, there never could be any prolapse. Manifestly then, knowledge of how that aperture is normally kept closed will furnish an important aid in the elucidation of the problem of the prolapse mechanism.

The continued teaching that the pelvic aperture is normally closed by fascial structures, the integrity of which prevents prolapse, betrays an utter disregard of accurate anatomical, physical and pathological data which have been separately accumulated over a long period of years. With logical application of the data a solution can be evolved which is in accord with all known facts, is not theoretical, or supposititious, and offers in turn, a sound explanation for the mechanism by means of which prolapse is prevented normally.

CLOSURE OF BONY OUTLET

The bony pelvic outlet is diamond shaped. The contents of the pelvis are prevented from being extruded through the outlet by a muscular hammock consisting of the levator ani and the coccygei muscles which completely closes the bony outlet except for a slight deficiency anteriorly in the median line.

The levator ani arises from the pubis about ½ inch from the middle of the symphysis, from the fascia covering the obturator internus muscles (the so-called "white line") and from the ischial spines. These fibers pass backward and inward obliquely, the anterior running more directly backward and the posterior ones more nearly inward. They are inserted into the anococcygeal raphe or ligament and into the coccyx and terminal part of the sacrum. The anterior free margins of the levator ani form the lateral boundary of the pelvic floor aperture, the deficiency in the pelvic floor. The pubis forms the anterior boundary and the junction of both levators form the posterior boundary of the deficiency. The posterior margins of the levator ani overlap the anterior free margins of the coccygeus while the posterior free margins of the coccygeus overlap the small sacrosciatic ligaments. Excepting for the deficiency anteriorly in this musculature the opening of the bony pelvic outlet is perfectly closed by these muscles. Through this deficiency the urethera, the vagina and the rectum gain an exit to the surface of the body. This aperture situated medially and anteriorly just behind the pubic arch is obviously a necessity.

If this pelvic floor aperture remained open constantly the pelvic viscera would be in danger of extrusion not only because of the force of gravity but also because of the continuous variation in intra-abdominal and intrapelvic pressure (caused by the contractions of the diaphragm and the abdominal wall by coughing, sneezing, straining, etc.) which would constantly compress and force the pelvic viscera out. There must exist some mechanism which would be able to keep this pelvic floor aperture adequately closed to counteract the extruding force of this pressure and yet provide for the occasional passage through the aperture of the contents of the three canals which traverse it. It must therefore be both resistant and elastic. Consequently, it is inconceivable that such a closure-mechanism could consist of fascial structures which are inherently dense and unyielding or else have no supportive value at all. Certainly no fascial structures whether dense and firmly resisting or loose and yielding could be adaptable to the requirements placed upon this aperture of both closure and relaxation for the passage of visceral contents.

It has been the opinion of many that the pelvic fascia which forms a continuous sheet varying in density in certain positions, is the chief support of the pelvic viscera against ordinary conditions of pressure. Studies indicate quite definitely that the development of the fascial sheath of muscles depends on the functional activity of the muscle. The analogy which has often been drawn between the value of the fascial support of the abdominal wall and the fascial support of the pelvis is not based upon proper premise. The fascial support of the abdominal wall is not a fascial investment of muscle; it is an *aponeurosis* of origin or insertion of muscles. The individual muscles have their own fascial coverings, but the aponeurosis of the external oblique muscles, for example, is a very dense, highly resistant broad tendinous fibrous tissue sheet which forms the insertion of the muscle. The fascial sheath of the muscles of the abdominal wall is very much more delicate than any aponeurotic continuation of any of the muscles of the abdominal wall. Such aponeurotic membrane cannot be compared to the very much more delicate, thinned-out, and less resistant fascial sheath of the pelvic floor musculature as a restraining factor in the prevention of prolapse. Goff[2] demonstrated very clearly that in the rectovaginal and the vesicovaginal septum, the only tissue which can be found which might be called a fascial structure is a loose areolar connective tissue. This cannot be dissected as an individual layer. Thus there is no fascia other than this loose areolar tissue available for use in the surgical correction of

either cystocele or rectocele. This has been corroborated by me.[1]

Instead of fascia, however, there is a musculature which, with the exception of the anterior pubic arch, completely circles the pelvic floor aperture and the three canals which pass through it. This musculature, when intact, is capable of increased contraction and great relaxation and forms a very efficient closure-mechanism capable not only of preventing the extrusion of abdominal and pelvic viscera, but also of permitting the proper passage of visceral contents from the pelvis to the exterior. Let us now carefully consider how it effects the closure of the pelvic floor aperture.

This musculature, in the region of the pelvic floor aperture consists of two distinct parts, the pubococcygeus and the puborectalis. The former, is more or less in the same plane as, and continuous posteriorly with, the remainder of the pelvic floor musculature, the ileococcygeus and the coccygeus. The latter, the puborectalis, is placed just below this in a different plane entirely. Both arise from the pubic bone, their innermost border reaching so near the symphysis that the distance from the inner edge of the muscle bands of one side to that of those on the other is 1 inch. The upper, the pubococcygeus, passes backward, inward in a gentle curve and slightly upward to be inserted into the anococcygeal raphe and coccyx forming with its fellow of the opposite side a V-shaped muscle. The apex of this V is at the coccyx while the diverging arms form with the pubis the upper part of the pelvic floor aperture. The lower, the puborectalis, passes backward by the side of the vagina and rectum but instead of being attached to the coccyx or anococcygeal raphe turns around the anorectal junction and becomes continuous with the corresponding muscle layer of the other side. The two muscle arms together form a U-shaped muscle, the *Posterior Commissure of Which Is Not Tendinous or Fibrous but Continuous Striated Muscle Lying Free in Loose Fatty Connective Tissue.* At this point the puborectalis lies above the level of the superficial perineal muscle but below that of the main part of the pelvic floor musculature. Its muscle arms extend forward to just above the anterior border of the pubic arch and form with that part of the arch between the points of origin the lower and more important part of the pelvic floor aperture. It must be remembered that unlike the pubococcygeal muscle the puborectalis has no posterior attachment either to the anococcygeal raphe, the coccyx or to the sacrum. (Figs. 1, 2.)

When muscles contract they shorten in the long diameter and thicken transversely. If they have bony attachments at both ends, resistance to the shortening results in increased tension of fibers. In the case of the pubococcygeus, a bilateral musculature having bony attachments at both ends is so arranged because of its gentle curve that between the two symmetrical parts there exists an almost elliptical space. Contraction of such musculature with its consequent thickening gives rise to a diminution in the width of the space. Thus, the pelvic floor aperture is diminished in its transverse diameter when that muscle contracts. This contraction results in a bilateral occlusion of the visceral canals placed in the median line between the two symmetrical arms of the musculature. It has been definitely shown that the rectum is constricted in this way.[3]

The result of the contraction of the puborectalis is entirely different. It was emphasized previously that it has a bony attachment anteriorly but no tendinous or bony connections posteriorly. When it contracts, there is no bony resistance to shortening in the longitudinal diameter and the posterior commissure of this muscle is pulled forward towards the pubic arch. In its forward advance it pulls the rectum forward at its flexure and in so doing pushes the vagina firmly against the pubic arch. In this way it acts as a very efficient sphincter mechanism for closing

the pelvic floor aperture, thus compressing and closing the two visceral canals, the vagina and rectum. Budin[4] has shown that

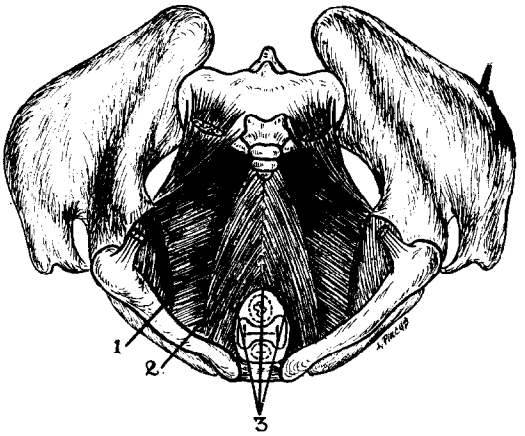

Fig. 1. Muscular closure of bony pelvic outlet. 1, Lateral portion of levator ani. 2, Pubococcygeus portion of levator ani. 3, Pelvic floor aperture where a deficiency in musculature exists.

a cylinder of wax inserted into the vagina during voluntary muscular relaxation receives a very marked circular impression upon the forceful voluntary contractions of the muscles of the pelvic floor. Although the impression is most marked in the antero-posterior direction, particularly on the side toward the pubic arch, the lateral indentations are quite definite. The marked indentations of the wax model are evidence not only of the sphincteric closure of the vagina but also and more important they are further evidence of the value of the contraction of the puborectalis muscle as a means for closure of the pelvic floor aperture by the forward advance of the posterior commissure of the muscle. Even when the external sphincter has been completely lacerated the retention of solid feces within the rectum is made possible by the contraction of the puborectalis portion of the levator ani muscle. Its sphincteric action in closing the vagina plays an important part during coitus, sometimes causing imprisonment of the penis within the vagina. Such cases have been described by Hilderbrandt,[5] Budin[4] and others. Dickinson[6] has noted the particularly good development of this muscle in erotic women.

If this aperture were very large its closure would be very difficult but its anteroposterior diameter measured from

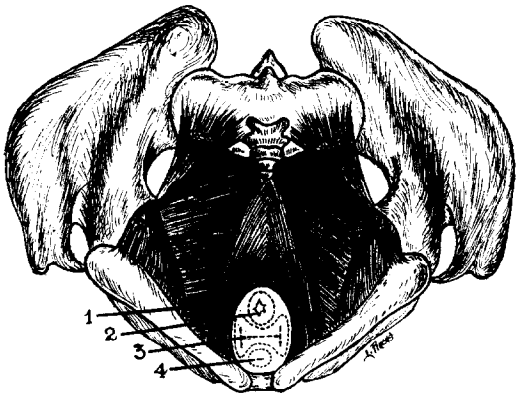

Fig. 2. Muscular closure of pelvic floor. 1, Puborectalis muscle at a lower plane than pubococcygeus and having no bony attachment posteriorly. 2, Rectum. 3, Vagina. 4, Urethra.

the midpoint of the summit of the pubic arch at the symphysis to the posterior wall of the anorectal junction is about 1½ inches. At first this seems grossly underestimated, but careful examination reveals it to be an accurate measurement. It must be remembered that this aperture is not a patent foramen with no contents. On the contrary it is completely filled by the tissues forming the urethra, vagina and rectum. That these structures do not fall out is due to the fact that circumferentially they are enclosed by or adherent to the muscle bounding the aperture. Henle[7] has shown that the non-striated muscle of the rectum and vagina interweave with the striated fibres of the puborectalis. Furthermore, these three visceral canals are adherent to each other and their mechanical presence reduces the size of the pelvic floor aperture so that it really exists only potentially during the resting stage.

Support of the Pelvic Viscera

There is no free space in the pelvis. The entire pelvis is occupied by viscera. Each viscus possesses volume and although it may be deformable it is incompressable. The thoracic diaphragm and the anterior abdominal muscular walls are continually

in apposition with the viscera contained within the abdominal cavity and constantly compress them as a result of their muscular tonus. This produces a pressure amongst them which is transmitted to the pelvic viscera and causes the latter to press against each other and in addition upon the walls and floor of the pelvic cavity. Since the bony pelvis is unyielding, the continuous changes in volume of some of the pelvic viscera must be made at the expense of other organs.

Concomitant with breathing, coughing, sneezing and straining movements, changes constantly occur in intra-abdominal and the intrapelvic pressure to which the pelvic viscera are subjected. This pressure, exerted upon the viscera in all directions but especially so in a downward direction wherein it is increased by the force of gravity, tends to cause their protrusion from the pelvis. This forms one component of a delicately balanced mechanism. The intact musculature of the pelvic floor, operating as already described, is the only resistant power so placed as to be capable of preventing such extrusion and it constitutes the second component. The innervation of the musculature allows for rapid reflex accommodation by contraction and relaxation to changes in intra-abdominal and intrapelvic pressure.

The pubococcygeus muscle is present in four-legged animals. There is no puborectalis muscle in such animals. In them the rectum is flattened laterally by the two pubococcygeus muscles, the free borders of which stretch between the symphysis pubis and the mid-line of the root of the tail, and in them the rectum runs a straight course. As a result of the assumption of the erect posture the puborectalis muscle was evolved, to provide a compensatory mechanism to control the increased intra-abdominal pressure which, unobstructed, might bring about an extrusion of the pelvic viscera.

Examination of the puborectalis in the dissecting room where it is seen in a distinctly atrophic state is partially responsible for the old idea that it can have very little value as a restraining influence in the pelvic floor diaphragm. To properly appreciate that it exists, lives and really functions, however, it must be studied in the living subject. This is best done in corpulent or pregnant women in whom there has been a rise in intra-abdominal and intrapelvic pressure which is followed by an adaptive hypertrophy of the muscles. In a well-developed primagravida, this muscle can easily be palpated as a tense mass of tissue constricting the finger against the pubic arch and it can be traced alongside the vagina, from its pubic origin backward. On simultaneous rectal examination with the other hand it can be perceived to come from beside the vagina backward by the side of the rectum and then inwards behind and around the anorectal junction posteriorly where it becomes continuous with a similar mass of tissue on the other side. This in turn can be followed forward to the pubic bone of the other side. It lies at a higher level than the external sphincter of the anus. Straining movements on the part of the individual during examination will cause a marked contraction of the muscle. This will force the fingers against the pubic arch. It is readily distinguishable from the superficial muscles which are inserted into the perineal body. If it be picked up between the finger and thumb, it can be felt to harden, that is, to contract on coughing and straining. This contraction which is felt is live muscle function and not passive fascial action to which so much importance has previously been laid.

Thus the conception is established that the pelvic floor aperture is closed by a living muscular mechanism. The association between increased abdominal pressure and contraction of the circular musculature surrounding the pelvic floor aperture can be voluntarily interrupted as for example during defecation but unless conscious dissociation is created the muscle contracts reflexly as a protective mechanism against extrusion of contents of the

pelvis or of the canals running through the pelvic floor aperture.

In a standing position, the pelvic floor aperture is in the most dependent portion of the pelvic cavity and might seem to be most disadvantageously placed. Such position would invite the assumption that in prolapse it is the weight of the individual organ plus the effect of gravity which causes the extrusion. This, however, is not true because the patient suffering with prolapse, after reposition of the organs, even placed in the lateral position, can voluntarily, by bearing down, extrude the pelvic organs from the pelvic floor aperture.

Considering the position of the pelvic floor aperture in relation to the transmission of increases of intra-abdominal pressure to the pelvic cavity, it would at once seem that the aperture is placed in the most advantageous position against prolapse. A sagittal section of the body shows the outline of the abdominal cavity to be pear-shaped with the large end uppermost. The posterior wall, consisting of the psoas muscle and the fat pad, is inclined backward from below upward at an angle of about 50° and it forms a padded shelf which helps to support the organs of the upper abdomen. Pressure from above would be directed not downward in a straight direction but downward and forward toward the anterior abdominal wall at an angle of about 50°. It would strike the anterior abdominal wall at its lower portion whence it would be reflected backward toward the sacral hollow. (Coughing produces marked bulging of the anterior abdominal wall at its lower portion thus confirming the conception.) Because of the natural lumbar lordosis there is a recession of the pelvic cavity from that of the abdomen. This is rendered more conspicuous by the promontory of the sacrum. When there are marked and sudden rises of pressure within the abdomen as the result of strain or cough the visceral thrust from above is reflected by the lower abdominal wall into the pelvis and is directed downward and backward so that it impinges upon the superficial surface of the organs of the pelvic cavity far behind the aperture.

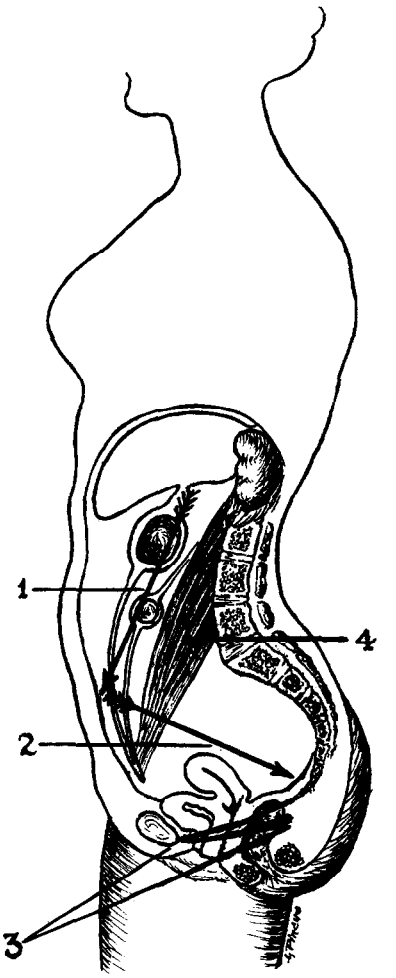

Fig. 3. Sagittal section demonstrating (1) direction of intra-abdominal pressure thrust; (2) deflection from abdominal wall into sacral hollow; (3) lower limit of pelvic floor aperture guarded by puborectalis muscle situated in most dependent portion of pelvis but anterior, out of direct line of deflected intra-abdominal pressure; (4) psoas muscle.

Thus, the pelvic floor aperture because of its anterior position (situated as it is immediately behind the symphysis pubis) is out of reach of the direct visceral thrust. In the erect posture, such a pressure wave can reach the aperture only by further reflection and diffusion. An added safeguard is the plasticity of the visceral mass. This further dissipates the force of the pressure wave causing it to diffuse. There-

fore despite its dependent situation in the pelvis, the aperture is really in an advantageous position. (Fig. 3.)

MAINTENANCE OF UTERUS IN ITS NORMAL POSITION

The uterus is maintained in its normal position by two factors. One is the interaction of the pressure of the intestinal coils from above exerted on its broad posterior surface against the supporting value of the pelvic floor musculature and the second is the volume of the connective tissue in which the pelvic viscera are embedded. An increase in the volume of the pelvic connective tissue such as occurs by the deposition of fat as part of a general obesity causes a relatively high position of the uterus in the pelvis.

Against the conception that the uterus is maintained in its position by the thickening of the connective tissue to which the cervix is attached and that relaxation of these connective tissue thickenings results in prolapse, are massed a group of facts. In the first place the normal mobility of the uterus and the restraining influence of the connective tissue are irreconcilable. If they fix the position of the uterus then the cervix should have less than its normal mobility. Since the cervix is mobile these tissues do not cause its fixation. If the mobility is due to slackness of these thickenings what causes the recession of the cervix to its normal position after release of the tension of a tenaculum by means of which it was drawn down to the vulva?

No matter how high the intra-abdominal pressure be raised voluntarily by straining, the cervix never descends to as low a level as it can be pulled by a tenaculum. When the uterus descends following straining, it does not do so alone. The downward movement is manifested in all its attachments, the other pelvic viscera, the embedding connective tissue and the pelvic floor itself. The reason why the uterus does not descend as far as it can be pulled by a tenaculum is that with the descent due to strain there is a synchronous, reflexly produced, increase in the tone of the levator preventing too much displacement and a more forceful closure of the pelvic floor aperture preventing extrusion.

These connective tissue thickenings are said to reach full development in adult life. Yet prolapse occurs in adult life and not in childhood. Infants are subjected to great rises of intra-abdominal pressures with frequent, sudden, down-thrusts of viscera, as in crying, but prolapse does not occur. Yet in infants with spina bifida in which the lesion involves the lower sacral nerves supplying the muscles of the pelvic floor, prolapse of the uterus is not an uncommon occurrence following the strain of crying even as early as the second or third day after birth.

DEVELOPMENT OF PROLAPSE

It becomes evident, then, that two interrelated causes are important in the production of prolapse. The first is an increased pressure in the pelvis, and the second, an insufficient resistance of the pelvic musculature. The exciting cause, the increased pressure in the pelvis, is one which is produced frequently, and often with marked variation in degree. Not infrequently it is the result of corpulency. This necessarily affects the pressure in the pelvis. When corpulency exists alone without the predisposing insufficient resistance of the pelvic floor musculature, the uterus rises high in the pelvis. When, however, it is found in conjunction with an incompetent resistance of the pelvic floor musculature, prolapse occurs.

The predisposing cause, the insufficient resistance of the pelvic floor musculature, is usually the result of childbirth injury. Such an injury affects most frequently the sphincteric mechanism through which the child passes, namely, the puborectalis muscle. The injury to the puborectalis muscle is most likely to occur in the anterior portion near the pubic attachment in the region of the bladder support

and not posteriorly where greater extensibility is possible. The result is a permanent enlargement of the aperture particularly in the transverse direction. As may be expected, the closure of the aperture in the anteroposterior diameter by approximation to the symphysis pubis is likewise interfered with.

Not every pregnancy is complicated by an injury to the puborectalis muscle. In many instances where it is injured the amount of injury is insignificant. In others, sufficient repair takes place during the lying-in period so that with the return to active life there is a resumption of functional activity sufficient to maintain adequate pelvic floor resistance to the intra-abdominal and intrapelvic pressure.

In the comparatively few instances where the pelvic floor resistance is insufficient as a result of atony of the muscle or general weakness, the whole musculature of the pelvic floor is involved. In those instances, the coccygei or the ileococcygei suffer as much as the puborectalis muscle. The greater the injury to the pelvic floor, the less will be the intrapelvic pressure rise necessary to produce prolapse. While hard physical manual labor would be insufficient to produce a prolapse in one with slight damage, even ordinary exercise may be responsible for extrusion of the uterus at the vulva in another with severe impairment and in the borderline cases where manual labor produces symptoms of prolapse it is easy to understand how a less arduous life often relieves them. Then, again, a woman in whom the injury to the pelvic floor, in relation to the amount of intrapelvic strain that she ordinarily undergoes is insufficient to produce prolapse, can develop a prolapse as time passes and the intra-abdominal pressure strains are increased by the gradual development of obesity.

When the pelvic floor aperture is enlarged as a result of injury to the sphincter mechanism, the bladder which rests above the aperture anteriorly and which is subjected to the forces of intrapelvic pressure is pushed down into the aperture everting the anterior vaginal wall in front of it. Thus, at first, it dilates the aperture and then it is extruded through it. In a similar manner dilatation of the anterior rectal wall because of increased pressure within, as a result of straining at stool occurring repeatedly in an individual in whom the aperture is larger, ultimately results in extrusion of the posterior vaginal wall overlying the rectum through the aperture with the consequent production of a rectocele.

Whether the bladder comes through the aperture first or the rectal wall begins to protrude first, matters but little. Ultimately, with the continuation of visceral thrust from above and an insufficient resistance on the part of the pelvic musculature, the cervix and uterus begin to slip through the pelvic floor aperture and finally present at the vulva. Thus, the anterior position of the cervix of which so much is made in the supposed antecedent retroversion is not the cause of prolapse. The uterus and cervix together are more or less non-deformable. As a result of intra-abdominal and intrapelvic pressure the anterior cul-de-sac becomes dilated with intestines and the uterus is pushed away from its anterior position where it rests on the bladder, back toward the sacral hollow and the cervix assumes a corresponding anterior position. It is to be expected that because the cervix is immediately over the aperture it frequently is the first organ to present itself at the vulva when the factors which produce prolapse have been active.

It is obvious that as the uterus becomes retroverted, the cervix anteverted and prolapse begins, elongation of the supposed supporting ligaments must occur. The attached connective tissue, the uterosacral and the pubovesical ligaments become greatly lengthened. The absence of elongation of these ligaments in normal individuals is due to the negligible tension to which they are subjected.

It is not an uncommon thing to find patients in whom the cervical connective tissue attachments are so loose that the uterus can be moved very freely in all directions on bimanual examination, yet who, leading active lives, have no signs or symptoms of prolapse because of a competent pelvic floor musculature. On the other hand it is inevitable that an active woman with an incompetent musculature and a considerably enlarged pelvic floor aperture will develop prolapse. Laceration or insufficiency of the pelvic floor aperture by itself cannot bring about prolapse. This in itself is but the predisposing cause. It is only when the patient begins to move about, undergoing varying degrees of strain, and performing long arduous manual labor that prolapse may arise, for then the exciting factors come into play.

The great mobility of the uterus in the normal individual is not due to the looseness of the connective tissue attachments but rather to the pliability of the visceral mass about the uterus. An increase in the volume of the visceral mass occasioned by distention of the bladder, the enlargement of the pregnant uterus, the growth of tumors in the pelvis leads to diminution in the mobility of the uterus. Another factor which becomes effective because of such diminished deformability of the viscera is the concomitant increase of the intra-abdominal and pelvic pressure. The increased intra-abdominal pressure factor tends to restrict the mobility of the viscera in their normal relationship.

In the normal individual, straining with marked increase of intra-abdominal and pelvic pressure results in a descent of the pelvic floor. There is no enlargement of the aperture however, because, reflexly, the tonicity of the muscles guarding the aperture is correspondingly increased so that with this exaggerated activity, no extrusion of pelvic viscera can occur. This synchronous muscular contraction can best be felt by the examining finger in a primipara at about the forth or fifth month because there the accommodation to the increasing size of the fetus and uterus with the concomitant increase in intra-abdominal pressure produces a hypertrophy of the pelvic floor musculature which lends itself more readily to the palpatory investigation of contractions. In an individual in whom there is a weakened, injured, dilated pelvic floor a similar strain would not only cause a descent of the floor but also an extrusion of the pelvic viscera with prolapse.

The normal respiratory rise and fall of pressure within the abdominal cavity is manifest on the peripheral surface of the body by a rhythmic motion downward and upward corresponding to the contraction and relaxation of the diaphragm. This can easily be observed on any patient when placed in the lithotomy position. When the diaphragm contracts and the intra-abdominal and intrapelvic pressure rises the pelvic floor (the levator ani muscle) is forced downward and with the relaxation of the diaphragm, it returns to its normal position again. During coughing, straining, sneezing, when there is a very marked rise in pressure in the abdomen produced not only by diaphragmatic contraction but also by marked contraction of the abdominal muscles, the normal non-lacerated pelvic diaphragm would be expected to bulge tremendously, and it would so bulge, were it merely an elastic structure without the possibility of contracting and without the regulatory reflex mechanism which insures its simultaneous contraction. It does not bulge to any greater extent because it does contract simultaneously with the abdominal wall and the thoracic diaphragm. This contraction can easily be palpated during coughing or sneezing and this contraction forms a very efficient mechanism for successfully withstanding the down-thrust occasioned by the sudden rise of intra-abdominal and intra-pelvic pressure. Were there not such a mechanism for withstanding the down-thrust, the pelvic viscera would certainly be extruded from the pelvis. It is not difficult to understand how such a mechan-

ism would be a much more effective restraint against visceral extrusion than would the radiating cervical connective tissue strands which can come into play only as their tension is increased by visceral descent and which even then are only capable of passive resistance. When such a patient is given spinal anesthesia vomiting movements may produce an extrusion of the cervix from the vagina because now the pelvic diaphragm no longer acts as a restraining mechanism because the anesthesia has temporarily interrupted the nervous mechanism which normally reflexly produces contractions of the levator simultaneously with intra-abdominal rises in pressure.

Where the pubococcygeus or puborectalis muscle is damaged, the visceral parts immediately adjacent to the pelvic floor aperture bulge into the aperture during the period of straining. The larger the aperture, the greater is the initial bulging, the greater the descent, and the more these parts can enter the aperture, the more is the expanding pressure exerted circumferentially on the boundaries of the aperture tending to enlarge it according to the amount of pressure transmitted to it from the pelvis. On the contrary, straining, while it affects those organs immediately over the pelvic floor aperture so as to attempt to extrude them, in the normal individual also causes equal pressure downward on the lateral boundaries of the aperture and because of the equality of the pressure transmission in all directions the tendency toward extrusion through the aperture is nullified by the pressure on the sides of the aperture which tends to close it.

In the case of a damaged pelvic floor musculature, when descent and bulging through the aperture occur, the aperture becomes secondarily dilated, allowing more and more visceral contents to come through. As the prolapse continues and the prolapsing organs descend they are grasped circumferentially by the contractions of the remainder of the pelvic floor. This continues until an equilibrium is established. This equilibrium depends especially upon the condition of the puborectalis. In the development of prolapse the puborectalis muscle hypertrophies as an expression of the attempt on the part of the individual to compensate for the original injury, that is, as an adaptive response. Occasionally, such a response may be sufficient to close the aperture and prevent further prolapse.

Besides prolapse due to obstetrical injury and to spina bifida in infancy, prolapse occurs in a very small group of nulliparous women (intravaginal hypertrophy of the cervix is not included in this group). These women are found to have spina bifida occulta with trophic disturbances in the pelvic floor musculature or constitutional pelvic floor musculature dystrophy. Thus the mechanism described fits the development of prolapse in all its phases.

Grateful acknowledgment is made to Dr. Irving Gordon for his aid in editing this manuscript.

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