DEFINITION AND INCIDENCE

A prolapse is downward or forward displacement of one of the pelvic organs from its normal location. Traditionally, prolapse is referred to displacement of the bladder, the uterus, or the rectum. The problem of pelvic organ prolapse excluding true rectal prolapse will be described in this chapter.

Of the common gynecological complaints one comes across in clinical practice, the problems related to pelvic support disorders are common and account for almost 400,000 surgical procedures annually for women in the United States, which is nearly 60 percent of major gynecological surgeries a year and 101,907 hospitalizations due to prolapse and/or stress urinary incontinence in the reproductive age group. The development of effective operations to alleviate uterovaginal prolapse was one of the key factors that led to the establishment of gynecologic surgery as a separate specialty.

CLASSIFICATION

Pelvic organ prolapse can be classified etiologically into congenital (neonatal)—which is a rare event found at birth in some neonates and disappears soon, unless it is associated with spinal cord defects; acquired—due either to childbirth or any kind of injury; and nulliparous—where childbirth injury to the supports is not the cause.

Prolapse is commonly classified on the basis of anatomical structure that is protruded or prolapsed, namely:

- **anterior vaginal wall:**
  - bladder — cystocele
  - urethra — urethrocele

- **central or cuff:**
  - uterus — uterine prolapse

- **posterior vaginal wall:**
  - small bowel — enterocele
  - rectum — rectocele

These descriptive terms have been in use since time immemorial, but they tend to prejudge the true nature of any prolapse by focusing attention on bladder, rectum, or uterus rather than focusing on the specific defects that are responsible for alteration in vaginal support.

Normally, the cervix or vaginal cuff is supported at or above the level of ischial spines. Pelvic organ prolapses have usually been graded on a scale of 0-3 (or 0-4), the grade increasing with increasing severity of prolapse, with 0 referring to no prolapse and 3 (or 4) referring to total prolapse (procidentia) with reference to vagina and introitus (Table 18.1).
Since introitus is an ill-defined and imprecise term, hymen is preferred as landmark to evaluate prolapse, even though plane of hymen is somewhat variable depending on the degree of levator ani dysfunction. In the latter system of evaluation, consideration is given to anterior and posterior vaginal walls descent (Table 18.2).

**Table 18.1: Uterine descent**

<table>
<thead>
<tr>
<th>Degrees</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1°</td>
<td>Cervix below ischial spines</td>
</tr>
<tr>
<td>2°</td>
<td>Cervix up to the introitus</td>
</tr>
<tr>
<td>3°</td>
<td>Cervix outside introitus</td>
</tr>
<tr>
<td>(Proxidens)</td>
<td>All of the uterus outside the introitus</td>
</tr>
</tbody>
</table>

Some workers combine 4° with 3° prolapse.

**Table 18.2: Hallway system for grading each site of pelvic relaxation**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Normal position for each respective site</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal position for each respective site</td>
</tr>
<tr>
<td>1</td>
<td>Descent halfway in the hymen</td>
</tr>
<tr>
<td>2</td>
<td>Descent to the hymen</td>
</tr>
<tr>
<td>3</td>
<td>Descent halfway past the hymen</td>
</tr>
<tr>
<td>4</td>
<td>Maximum possible descent for each site</td>
</tr>
</tbody>
</table>

Notes for using the grading system:
1. Prolapse is graded at each site (cystocele, uterine prolapse, vault prolapse, rectocele, enterocele) with patient straining maximally. The upright position may also be used.
2. When choosing between 2 grades, choose the higher grade.

Data from Baden and Walker.

However, the International Continence Society has adopted a site-specific descriptive system, taking hymen as a fixed point of reference and measurements are taken from that point on the anterior and posterior vaginal walls and to the vaginal apex. In addition, the genital hiatus width of perineal body and total vaginal length are measured and recorded on a grid form. This suggested system is complex and needs to be popularized since it describes patient characteristics, helps to develop effective treatment protocols, and to assess surgical outcomes.

**ETIOLOGY**

The etiology of pelvic floor disorders is more likely to be multifactorial. All pelvic prolapse conditions and urethral hypermobility result from pelvic floor relaxation. The etiopathology is often attributed to childbirth injury. Prolonged labor and high forceps delivery, particularly if prolonged traction is required, have always been blamed as etiological factors in the development of genital prolapse. Considerable stress imposed on the pelvic floor during a 'squatting' labor might increase the risk of subsequent prolapse, but return to physical activity soon after labor may be protective. 'Bearing down' before full cervical dilatation puts a tremendous strain on the uterine supports and contribute to an increased risk of prolapse. Fundal pressure was a recommended method of placental delivery, but it could lead to gross stretching of the uterine ligaments and has now been abandoned in favor of patience, maternal effort and judicious controlled cord traction. Neuromuscular damage to the pelvic floor is associated with development of pelvic organ prolapse. The function of the levator ani muscle can be compromised in two ways. First, there can be direct mechanical injury to the muscle. Second, damage to the nerve supply leading to their inability to contract, even though they themselves remain intact. The direct nerve supply from the sacral plexus to the levator ani is placed under great stretch during parturition resulting in transient neuropraxia and repeated birth could further injure such patients and ultimately produce symptoms of prolapse and incontinence as the muscular supports are progressively damaged. A prolonged second stage and heavy fetal weight are associated with neural damage.

Previous pregnancy is not a required precondition for prolapse, because it can occur in nulliparous women, especially when the cervix is congenitally elongated. Women with congenital defects like ventral arched abnormalities such as spina bifida, or those with defects of pelvic floor muscles as in exstrophy of the bladder or those with primary myopathies, such as muscular dystrophy, have a striking propensity to develop uterine prolapse, often before pregnancy and at remarkably early age. Syndromes such as Ehler-Danlos syndrome characterized by fascial and connective tissue weakness have significantly higher prevalence of genital prolapse.

An underlying abnormality of connective tissue in pelvic floor ligaments and fascia is also believed to be
The cause of pelvic support disorders. This may be due to an intrinsic abnormality of collagen synthesis (e.g., abnormal collagen, imbalance between synthesis and degradation, imbalance between collagen types) that leads to pelvic support disorders regardless of outside stressors. A second mechanism might be mildly abnormal fascia and ligaments that can withstand normal pressures, but with excessive strain (e.g., high parity, chronic straining, or loss of pelvic floor contraction) develop genitourinary prolapse. In these individuals, the abnormality might be errors in repair of damaged ligaments and fascia, or lack of remodeling in mature collagen.9

The tendency to prolapse is more likely to manifest after the menopause, when the ovarian steroidogenesis ceases and genital support tissues are no longer affected by estrogen. Although inherent tissue weakness and childbearing are among the most common causes of vaginal prolapse, an additional cause is a prior hysterectomy. If the vaginal vault was not sufficiently resuspended and the cul-de-sac was not obliterated, vault prolapse and/or enterocele are common sequelae.

The greatest forces that affect the pelvic floor come from increased intra-abdominal pressure and the weight of the abdominal organs in a woman with some weakness induced by childbirth trauma or inherent connective tissue weakness or aging and menopausal hypoestrogenism. Chronic intra-abdominal pressure from pulmonary disease or lifting, chronic straining during bowel movements, ovarian tumor, ascites, and fibroid uterus contribute to aggravation of the prolapse condition.

ANATOMY OF THE PELVIC FLOOR

Understanding the anatomy of structural components that support uterus and pelvic floor is essential to understand the problems of the pelvic floor and in planning surgical correction.

The top layer of the pelvic floor is the endopelvic fascia, which attaches the pelvic organs, especially the vagina and uterus to the pelvic walls, thereby suspending the pelvic organs. Support of the pelvic organs is provided by a group of muscles referred to as the levator ani.

Viscero-fascial Layer

The fascial layer of the pelvic floor is a combination of the pelvic visceral and endopelvic fascia. Hence, it is referred to as viscerofascial layer. On each side of the pelvis, the endopelvic fascia attaches the uterus and vagina to the pelvic wall. It forms a continuous sheet-like mesentery. It runs continuously from the uterine artery at its cephalic margin to the point at which the vagina fuses with the levator ani muscle below. The part that attaches to the uterus is called the parametrium, and that which attaches to the vagina, the paracolpium.

The parametria are made up of the cardinal (transverse cervical) and uterosacral ligaments, which are two different condensations of a single mass of tissue. Opposite the external cervical os, the sheet of tissue that attaches the genital tract to the pelvic wall arbitrarily changes name from the parametrium to the paracolpium. These supportive tissues contain prominent blood vessels, nerves, and fibrous connective tissue that can be thought of mesenteries that supply the genital tract bilaterally.

The paracolpium attaches to the upper two thirds of vagina and it consists of two portions. The upper portion (level I) consists of a relatively long sheet of tissue that suspends the vagina by attaching it to the pelvic wall. In the midportion of the vagina, the paracolpium attaches the vagina laterally and more directly to the pelvic walls (level II). This attachment stretches the vagina transversely between the bladder and rectum and has functional significance. The structural layer that supports the bladder is pubocervical fascia and is composed of dense fascia from the cervix to the pelvic wall. The suburethral endopelvic fascia is better developed than that higher in the area of bladder; thereby providing better support for the vesical neck.

This layer attaches laterally to the arcus tendineus fascia pelvis and also to the medial border of levator ani muscles. Loss of this normal support at the vesical neck is one of the factors responsible for stress urinary incontinence. The posterior vaginal wall and endopelvic fascia (rectovaginal fascia) form the restraining layer that prevents the rectum from protruding forward. In the distal vagina (level III), the vaginal wall is directly attached to surrounding structures without any intervening paracolpium.9
Damage to the upper suspensory fibres of the paracolpium (level I) causes different type of prolapse from that caused by damage to the midlevel supports of the vagina (level II). Defects in the support provided by level II vaginal supports (pubocervical and rectovaginal fasciae) result in cystocele and rectocele, and loss of the upper suspensory fibers of the paracolpium, and parametrium (level I) is responsible for the development of vaginal vault and uterine prolapse.

**Levator Ani Muscles**

The levator ani consists of two portions: the pubovisceral muscle and the iliococcygeus muscle. The pubovisceral muscle is a thick U-shaped muscle whose ends arise from the pubic bones on either side of the midline and pass behind the rectum, forming a sling-like arrangement. It has several components. The pubococcygeus is the most cephalic portion of the levator and passes from the pubic bones to insert on the inner surface of the coccyx. This portion does not contribute substantially to supporting the pelvic organs. The puborectalis portion of the pubovisceral passes beside the vagina, and lateral vaginal walls are attached to it. The muscle then continues dorsally where some fibres penetrate the rectum between internal and external sphincter, while others pass behind the anorectal junction. Laterally, the iliococcygeus arises from the fibrous band on the pelvic wall (arcus tendineus levator ani) and forms a relatively horizontal sheet that spans the opening within the pelvis and forms a shelf on which the organs may rest.

The opening in the levator ani muscle, through which urethra and vagina pass (and through which prolapse occurs) is the urogenital hiatus. The normal baseline activity of the levator ani muscle keeps the hiatus closed and thereby the lumen of vagina, urethra and rectum. This eliminates any opening in the pelvic floor through which prolapse could occur and forms a shelf on which the pelvic organs are supported.

The interaction between pelvic floor muscles and supportive ligaments is critical to the support of pelvic organs. As long as the levator ani muscles function normally, the pelvic floor is closed and the ligaments and fascia are under no tension. The fasciae stabilize the organs in their position above the levator ani muscles. When the pelvic floor muscles relax or are damaged, the pelvic floor opens and the vagina lies between the high abdominal pressure and low atmospheric pressure. Although the ligaments can sustain the organs in place for short periods of time, if the pelvic floor muscles do not close the urogenital hiatus, then the connective tissue will become stretched or damaged and eventually fail to hold the vagina in place. This situation is likened to a ship in its berth on the water, attached by rope on either side to a dock—where the water supports ship's weight and the moorings simply keep the ship from straying away.

**SECONDARY ANATOMICAL CHANGES**

Uterus gets retroverted before prolapsing through the vagina. The prolapse leads to further kinking of its blood supply, especially the venous drainage and results in congestion. The chronic congestion of the cervix will be responsible for the bulky cervix seen in menopausal women with prolapse.

The venous congestion over a period of time leads to decreased oxygenated blood in the most dependent portions of the cervix. Decubitus ulceration is the result of such tissue hypoxia. It is a trophic ulceration and heals on reposition of the uterus into the vagina, which restores venous drainage and improves availability of oxygenated blood.

In a patient with prolapse the exposed vaginal skin shows areas of keratinization and pigmentation due to exposure and friction. It looses rigidity from lack of estrogen.

Childbirth injury usually affects pelvic floor and traction from below by the weight of the uterus or any strain imposes pull of parametrium (cardinal and uterosacral ligaments) to keep the cervix and uterus in position. This results in stretching and elongation of softened, congested supravaginal portion of cervix.

Anterior vaginal wall prolapse and the resultant descent of posterior bladder wall near trigone cause stretching of the ureteric openings. It may also result in cystoureteric reflux of urine. The effect of these is hydroureter and hydronephrosis in a case of long-standing prolapse of severe degree.
SYMPTOMS
The degree of prolapse bears little relationship to the presenting symptoms. Someone with a severe prolapse, say procidentia, may have a few symptoms, whereas others with a small anterior wall descent may complain of more symptoms.

Symptomatic prolapse may manifest in several different ways. Majority of the patients referred to hospital with genital prolapse complain of ‘something coming down’. There may be only a feeling of pressure or insecurity ‘inside’.

Low backache may be one of the symptoms due to strain on the periosteal attachment of uterosacral ligaments caused by downward pull of uterine descent. The patient is unable to pinpoint the site of pain. It is felt more at the end of a day’s work and is relieved after taking rest by lying down.

Anterior vaginal wall prolapse leads to urethral hypermobility, loss of urethrovaginal angle and descent of proximal urethra below the pelvic floor. This results in the failure of direct transmission of increased abdominal pressure to urethra whilst it is transmitted to the bladder, which often (but not necessarily) results in stress urinary incontinence. A larger prolapse, prolapsing posterior bladder wall through anterior vaginal wall coming out below the urethra, can produce symptoms of voiding difficulty. Such patients may have the sensation of incomplete voiding and require digital pressure on the prolapsed vaginal wall for completion of the act. They may also have frequency and urgency of micturition due to cystitis.

Protrusion of the posterior vaginal wall by rectum can cause symptoms of inefficient rectal emptying, often described by the patient as constipation, necessitating splitting of the posterior vagina to reduce the pocket of trapped stool.

A sexually active woman may complain of dyspareunia or obstruction to the penetration because of tissues protruding outside the introitus. Lax vagina and introitus could be the reason for decreasing sexual pleasure or there may be diminished frequency of sexual intercourse because of anxiety on the part of her partner.

Congested and hypertrophied cervix by itself or the secondary infection may be the cause for discharge per vagina. The patient with decubitus ulcer on cervix will present with blood-stained discharge.

CLINICAL EVALUATION
Since pelvic support defects are frequently associated with specific alteration in bowel, bladder or sexual function, both the evaluation and management of poor support and abnormal visceral function are important in developing a treatment plan and assessing therapeutic outcome.

There are no universally agreed definitions for normal pelvic support or for pelvic support defects.

The patient is examined in the dorsal lithotomy position with moderate amount of urine in the bladder to help evaluate stress incontinence. Pelvic support is assessed when the patient is straining maximally as each individual site is identified. The defects are based on an imaginary line in the midvaginal axis extending from the midportion of the hymen to the hollow of the sacrum. During maximal strain in women with normal support, the urethra, bladder, cul-de-sac, and rectum will not cross the midvaginal axis. Support defects may occur at any or all of these sites.

Using any of the classification of grading the descensus (vide supra), the urethra, bladder, cervix or vaginal cuff, cul-de-sac, and rectum are described as grade 0 to 3 (or 4). If the patient is not straining effectively, the prolapse may not be apparent. If one is unable to make prolapse protrude when the patient is in lithotomy position, repeat examination may be necessitated while she is standing and straining.

Urethra
Urethral support defects are associated generally with paraurethral loss of support. As the patient with urethral hypermobility strains, the urethra rotates posteriorly, and its junction with the bladder straightens. The meatus rotates anteriorly. The paraurethral support defect can be confirmed by using an open curved ring forceps in a position lateral to the urethra to provide support paraurethrally as the patient bears down. As lateral support
is applied, one should look to see if there is any correction in the rotational descent. The cotton swab (Q-tip) test objectively quantifies the degree of mobility. When an individual is straining, the urethra usually does not rotate more than 30° from the horizontal plane, but some continent multiparas may have loss of urethral support with no stress incontinence.

Bladder
Support defects involving the anterior vaginal wall may occur in the midline, laterally or paravaginally, superiorly, or in any combination of these sites. Defects can be identified clinically by evaluating each individual area. Using the sponge forceps with the curve pointing posteriorly toward the ischial spines, the lateral aspects of the anterior vagina and pubocervical fascia can be returned to their normal point of attachment along the arcus tendineus fasciae pelvis. The forceps then is placed laterally, and the patient is asked to strain maximally; if there are no evident anterior defects, she has lateral or paravaginal loss of support. If, when she strains, there is some improvement in anterior support, but she continues to have a midline bulge through the open arms of the forceps, she also has a midline defect in pubocervical fascia. The forceps may be closed and used to support the base of the bladder centrally. When the patient strains and she has no midline descent, the support defect is central or midline.

Superior loss of support is characterized by several clinical clues. When the patient strains, if the anterior vaginal epithelium appears thin and, with loss of rugae from the vaginal cuff along the base of the bladder, and the anterior vaginal wall is longer than the posterior vaginal, the patient is likely to have superior loss of support of her pubocervical fascia. Superior defects are usually associated with midline defects.

Cervix/vaginal Cuff
Descents of the cervix or that of cuff as made out by dimples seen at 3- and 9-o'clock areas from the level of ischial spines signify inadequate cardinal uterosacral ligament (level I) supports.

An attempt should be made to evaluate the length of the cervix.

Cul-de-sac
With the loss of support in the cul-de-sac, the epithelium overlying it generally becomes thin, shiny and distended by intestines. Placing a speculum over protruding posterior vaginal wall and asking the patient strain will result in cul-de-sac gliding over it confirming the enterocele.

Rectum
Defects in the perirectal fascia occur most commonly in the midline but may occur laterally or transversely near the perineum or the vaginal cuff. The curved ring forceps may be placed posteriorly and laterally in an effort to reduce the posterior defect. If there continues to be a bulge between the open arms of the forceps, the defect is midline. The forceps may be closed and used to support the midline. If there is no loss of support when the patient strains, the defect is in the midline.

Perineum
The distance between the anal orifice and posterior fourchette should be noted. With a finger in the rectum and the thumb pressing against the perineum, the thickness of the perineum can be felt.

Loss of support at cul-de-sac and at perineal body is best identified intraoperatively. A full general and abdominal examination is necessary. It is necessary to exclude chronic chest problems, gross obesity and abdominal masses. Bimanual vagino-abdominal palpation should always be carried out to exclude pelvic masses such as ovarian cysts or fibroids.

DIFFERENTIAL DIAGNOSIS
Diagnosis of prolapse is not difficult, but the correct identification of site-specific defect in the pelvic floor may pose a problem.

Descent of anterior vaginal wall along with portions of bladder or hypermobile urethra may at times have to be differentiated from vulval cyst, tumor, Gartner’s duct cyst, or urethral diverticuli.

Congenital elongation of cervix needs to be considered while diagnosing uterine descent or vault prolapse. Cervical fibroid polyps or chronic inversion of uterus are rare conditions that may be reported as a uterine descent and need to be recognized.
Rarely, the patient complains of vaginal prolapse, but in fact she will be suffering from true rectal prolapse.

**TREATMENT OF PELVIC ORGAN PROLAPSE**

The modalities of treatment include conservative follow-up, surgical repair, or use of a vaginal pessary.

**Expectant Management**

Prolapse may be discovered during a routine gynecological examination. Such patients may or may not have symptoms due to pelvic support defect. One should keep in mind the axiom of medicine that “the asymptomatic patient cannot be made to feel better by medical or surgical therapy”. The patient should be informed of the physical examination finding and the problems that could make the condition worse over time. The importance of buttock squeezing exercises in improving perineal muscle tone should be emphasized and in some vaginal cones of increasing weight are prescribed for use in vagina for the same purpose. It is to be remembered that pelvic muscle exercises are virtually never harmful, but they are not likely to correct the problem if there is neuromuscular damage as the underlying pathology. In postmenopausal women use of estrogens as replacement therapy may help strengthen the supports and alleviate trivial symptoms. Periodic examinations while on follow-up will provide comparisons regarding the status of pelvic support defect, changes in patient’s physical condition or symptoms.

**Surgical Management**

Traditionally, prolapse has been treated by surgery, the nature of which depends on degree and type of prolapse, patient’s general health status and the need for preservation of menstrual, reproductive or coital function. The goal of surgery should be to relieve the patient of her symptoms by repairing each aspect of abnormal pelvic support in a durable and long-lasting manner. A detailed description of the various operations for managing pelvic support defects is beyond the scope of this chapter, however, a few general remarks are in order.

Removal of the uterus is not the surgery for prolapse. It is the repair of support tissues of uterus and vagina, which is the corrective surgery for prolapse. The surgical approach for each patient needs to be tailored to the specific symptoms, objective physical findings, and tests of visceral function. Most patients with prolapse have defects in more than one location, so attention should be paid to correcting all defects during the same operation. Operations for prolapse are generally, but not always, carried out through the vaginal rather than through the abdominal surgical route. Some conditions, such as stress urinary incontinence, are most reliably handled by an abdominal operation.

**Repair of Anterior Vaginal Wall Defects**

Patients with a central defect are best treated by anterior colporrhaphy, which reapproximates the pubocervical fascia in the midline under the bladder neck. Lateral defects require a different approach in which the vaginal attachments to the pelvic sidewall are reconstituted. These defects are commonly corrected either with a paravaginal repair via the abdominal or vaginal approach where endopelvic fascia is reattached to the arcus tendineus fasciae pelvis, or with a 4-corner bladder neck suspension.

**Operations for Uterovaginal Prolapse**

**Vaginal hysterectomy and repair:** Uterine prolapse is generally treated with vaginal hysterectomy and repair, which may be accomplished by several different techniques. It is preferred in a woman who has desired number of children and is not particular about preserving menstrual function. The advantage of vaginal hysterectomy is that it allows other vaginal surgery (viz., anterior and posterior colporrhaphy or enterocoele repair) to be performed at the same time, without the need for a separate incision or for repositioning the patient. At the time of hysterectomy for prolapse, special attention should be paid to closing the cul-de-sac using a McCall culdoplasty and to reattaching the endopelvic fascia and the uterosacral ligaments to the vaginal cuff to provide additional support.

**Manchester/Fothergill repair:** An alternative to hysterectomy for patients with uterine prolapse who wish to
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Manchester/Fothergill repair: An alternative to hysterectomy for patients with uterine prolapse who wish to
retain the uterus is the Manchester operation. In this operation, the bladder is dissected off the cervix and cervix is amputated. The cardinal ligaments are sewn to the anterior of cervical stump. Anterior colporrhaphy and colpopereineorrhaphy form the essential components of the repair.

Shirodkar's modification of Manchester repair: In a patient desirous of retaining childbearing function where amputation of cervix could compromise fertility, Shirodkar's modified Manchester repair may be a better option. In this operation, uterosacral ligaments are divided close to their attachment to cervix; the stumps are brought in front of the cervix, crossed and stitched to the cervix. High closure of the peritoneum of the pouch of Douglas is carried out. The cervix is not amputated. The rest of the operation is similar to Manchester repair.

Uterocervicopexy and sling operations: Occasionally, marked uterine prolapse may develop in a young nulliparous patient due to inherent weakness in suspensory supports. The basic principle behind these operations is to fortify the supporting ligamentary structures. Abdominal round ligament (Gilliam) uterine suspension with uterosacral plication and culdoplasty may be helpful. There have been efforts to use ribbons of rectus sheath brought out retroperitoneally between leaves of broad ligaments to be attached to isthmus of uterus. As a modification, some surgeons have used Marsilene/nylon tapes instead, to be attached between urethra and external oblique aponeurosis. A fascial strap or Marsilene/nylon tape can also be interposed between the cervix and the sacrum. Another attempt is to fix the mesialene tape to isthmus posteriorly and bringing the free ends out retroperitoneally to emerge laterally through anterior oblique abdominis for anchoring to anterior superior iliac spines on either side. There is anecdotal evidence of success in such patients using sacropinous ligament fixation or retroperitoneal abdominal uterosacropexy, suturing mesh or fascia to the uterosacral ligaments and then to the anterior longitudinal ligament of the sacrum. There is little information available regarding long-term follow-up of such patients.

LeFort repair: This is reserved for the very elderly menopausal women who are poor medical risks. It is not suitable for a sexually active woman. In this operation, rectangular flaps of the vagina from anterior and posterior walls are excised, the raw areas apposed with absorbable sutures. The repair converts vagina into a double barrel where uterus sits atop the midline adhesions.

Operations for Vault Prolapse

Among the most challenging cases are those involving complete eversion of the vagina in patients who have had a previous hysterectomy. Vault prolapse requires surgical correction because of the large size of the prolapse, its propensity to increase over time because of increase in intra-abdominal pressure, and the possibility of vaginal evisceration if it is not treated.

a. Colpectomy and Colpocleisis: For some patients, particularly elderly women who are not sexually active and who lead a sedentary lifestyle, surgically removing the vagina and closing off the space is a suitable option.

b. Colpopexy: An alternate procedure is required for younger women and women who wish to retain sexual function. For these women, the condition can be managed transvaginally or transabdominally. With the transvaginal approach, vaginal eversion is corrected by suturing one side of the vaginal apex (usually the right side) to the sacropinous ligament with one or two sutures—a transvaginal sacropinous colpopexy. In the transabdominal approach, the vaginal apex is suspended from the anterior longitudinal ligament along the sacrum using a graft of fascia or artificial mesh that is sutured to the vagina and to the sacrum in a retroperitoneal position—a transabdominal sacral colpopexy. Both the operations are highly successful in re suspending the vaginal apex.

Repair of Posterior Vaginal Wall Defect

Repair of posterior vaginal prolapse for rectocele and enterocele is performed vaginally using posterior colporrhaphy. In a rectocele repair, the posterior vagina is opened, the rectum is dissected away from the
pararectal fascia, and the levator ani muscles are plicated over the rectum in the midline, after which the vaginal epithelium is closed. It is important to note that a rectocele is a defect of the vaginal supporting tissue and not a defect of the rectum. An enterocele is a peritoneal hernia over the rectum, often seen as a second bump higher up in the vaginal canal on examination. The peritoneal sac should be carefully identified, opened, and closed with several purse-string sutures of permanent material; the sac should be excised, and the vagina should be closed once more over the defect. If the perineal body is noted to be deficient and the patient has a gaping introitus, this defect may be repaired by a perineorrhaphy or a perineoplasty, in which the vaginal fourchette is opened and the base of the levator ani muscle is pulled together in the midline, providing renewed support for the lateral vagina at its outlet. The latter step (perineorrhaphy) is better avoided in the sexually active woman for the fear of causing dyspareunia, unless gaping introitus and related sexual dissatisfaction are the symptoms.

**Conservative Management**

Conservative management of prolapse usually involves fitting the patient with a pessary. It should be emphasized that pessary will not cure prolapse but relieves the symptoms by stretching the urogenital hiatus. The patient who is using a pessary should have a well-estrogenized vagina. For women who are postmenopausal, it is preferable to use intravaginal estrogen cream 4-6 weeks before the pessary is inserted, because this makes the pessary more comfortable to wear and dramatically increases compliance and promotes long-term use. Pessaries are advised not only to test as to whether the low backache or urinary stress incontinence is due to prolapse condition, but also as an interim therapy to avoid and/or postpone surgery in early pregnancy, puerperium, patients unfit for surgery with limited life expectancy, or while awaiting surgery. Postmenopausal women should use intravaginal estrogen cream on a regular basis if they are not on hormone replacement therapy.

**SUMMARY**

Pelvic organ prolapse is not due to single etiology. Normal support of the pelvic organs depends on a combination of fascial and muscular support. The specific type of prolapse that exists in an individual corresponds with specific defects in the anatomic structures responsible for normal support. Hence, surgical management needs to be individualized.

**REFERENCES**