THE PELVIC FLOOR, RECTOCELE, CYSTOCELE, AND PROLAPSUS UTERI, ETIOLOGY, MECHANISM AND BEHAVIOR.

BY

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(With Four Illustrations.)

A CLEAR conception of the etiologic factors leading to prolapse of the vagina and uterus is necessary in order that the mothers of a nation may receive the benefit of the best prophylactic measures during labor and the most efficient repair (when necessary) after labor is over.

The importance of this knowledge from a sociologic as well as a professional standpoint cannot be overestimated.

Prolapsus of the vagina and uterus may result from changes occurring in (*a*) the perineum and lower third of the vagina (lower segment), (*b*) the paravaginal tissues and upper third of the vagina, (*c*) the parametrium and ligaments of the uterus (upper segment), and (*d*) both upper and lower segments of the pelvic floor together; and demands a careful detailed study of the virginal, marital gestation, and postpartum anatomy of each.

The lower (floor) segment is composed of bones, muscles and fasciæ, and the walls of three canals—anus, vagina and urethra, passing at a certain angle through the same.

The stationary *sacrum*, straight at birth, gradually later becoming curved forward toward the symphysis, both narrows and strengthens the outlet, extending as it does with the mobile *coccyx* as far forward as the axis of the inlet. These two posterior bones with the *ischia* and *pubes* laterally and anteriorly, provide for the strong muscle and fascia attachments so necessary to the pelvic floor anterior to this inlet axis point.

As the central perineal tendon is the key of the pelvic floor, the muscle and fascia attachments should, for gynecologic demonstration, be considered as arising from it, to the more easily demonstrate their (antagonistic) anatomic importance.

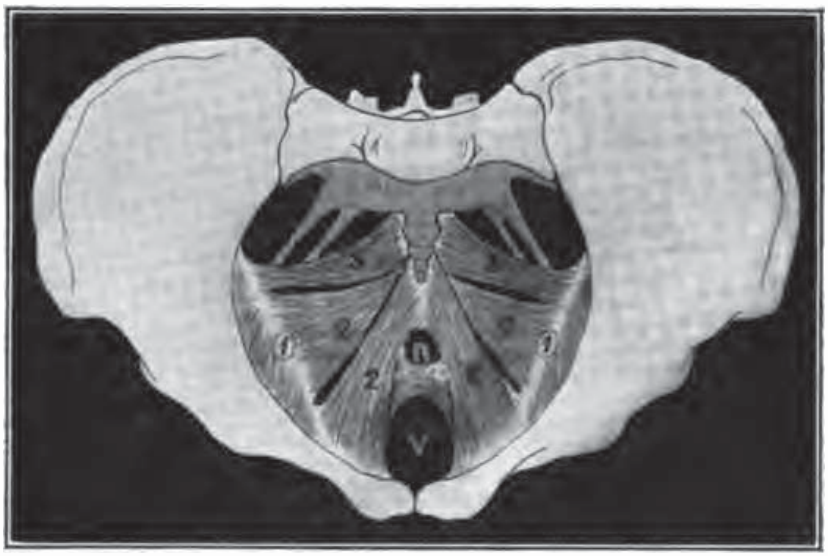

FIG. 1.—The levator ani seen from above. (1), The white line; (2), levator ani; (3), the coccygei; (R), opening for rectum; (P), central perineal tendon portion of levator; (V), opening for vagina.

(1) The two *levator ani* muscles (rectovesical fascia above, levator ani fascia below), derived on either side of the pelvis from the *white splitting-lines* of the obturator internus fasciæ, extend to the spines of the ischia, and from there are reflected to meet at the tip of the coccyx and unite in the midline to form a perforated sling.

This perforated sling (*muscular and fascial*) surrounds the anal opening, passes below and supports the ampulla of the rectum, then unites at the central perineal tendon with the five other floor muscles and fasciæ, then loosely surrounds the lower third of the vagina, passing thence to the pubes 2 1/2 cm. external to, and 3 1/2 cm. below the symphysis upper margin.

(2) The (triangular) *coccygeal* muscles on either side arise from

the spines of the ischia, and unite by their bases to the sides of the coccyx, and the last segment of the sacrum. They serve to help raise and support the mobile coccyx, after defecation and parturition.

(3) The *sphincter ani* muscle (backward tractor) passes from the central perineal tendon backward, surrounds the anus, to be attached to the tip of the coccyx.

(4) *The bulbus cavernosi muscles* (forward tractors), two slender muscles arising from the central perineal tendon on either side, pass forward to be inserted in the corpora cavernosi clitoridis, and assist in holding the central perineal tendon in its correct position.

(5) The two layers of *triangular fasciæ* extending from the central perineal tendon forward as two perforated sheets, are attached bilaterally to the tuberosities and rami as far forward as the pubes.

These two important sheets of fascia rigidly hold the central perineal tendon and vaginal introitus in their proper position in the preaxial part of the pelvic floor.

(6) *The transverse perineal muscles*, small in size, arise from the central perineal tendon and pass to either tuberosity and the adjacent rami.

From the foregoing it is apparent that the anatomic perineum extending from the clitoris to the anus might clinically be prolonged to the bony coccyx; it being equally important posteriorly with what one understands by the *perinuem gynecologic* (formed by the meeting of seven muscles and their fasciæ which is in reality the wedge-shaped *central perineal tendon or body.*

(7) *The vagina* is a nonstriated muscular, squamous epithelium lined tube passing obliquely from before backward and upward through the pelvic floor, with its greatest lumen at the fornices near the cervix, and its smallest at the introitus near the hymen, where it is still further narrowed if this latter be intact.

The vaginal walls are collapsed antero-posteriorly in an H-shaped manner. The more loosely attached anterior being at least partially supported by the closely opposed more firmly attached posterior wall.

(*a*) *The virgin vagina* about parallel with the conjugata anatomica (promontory suprapubic line), is practically horizontal when standing, and forms a slightly acute angle with the normal anteverted uterus above.

This "horizontalness" is very necessary to allow the vaginal canal to pass through the pelvic floor with the least possible weakening anatomically, and at the acutest angle to the anteverted uterus above,

to provide the greatest resistance to the intraabdominal pressure by spreading this pressure more evenly over the entire floor when rising and falling with the inspiratory and expiratory increase and decrease. This is especially true during violent exertion when the "horizontalness" of the vagina and the normal anteversion of the uterus are both creased, making the angle still more acute and prolapse consequently more difficult; indeed, with an intact hymen, the normal pelvic floor is almost as strong as that of the boy, so architecturally perfectly is it constructed.

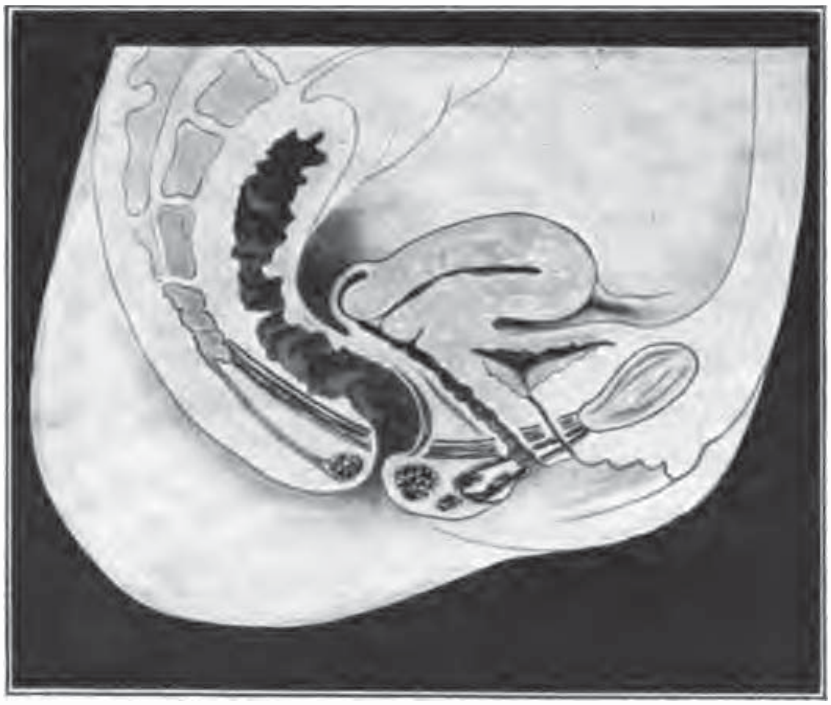

FIG. 2.—FIG. 2.—The normal angle of the pelvic organs to the canals passing through the floor and the normal perineum below.

(*b*) *The Marital Vagina "Nulliparæ"* with the hymen ruptured has suffered its first traumatic lesion. The introitus is now situated slightly more posteriorly than before (at least as far as the posterior wall is concerned).

The "horizontalness" of the vagina in the pelvis is slightly lessened. The whole vagina throughout becomes longer, wider and looser.

Married nulliparæ frequently show a marked congenital looseness of the para-tissues (-metrium -cystium and -vaginum)

combined with a tendency to undue cervical anteposition, and even corpus retroversion and under sudden and repeated excessive increase of intraabdominal pressure (while lifting weights, etc.), to some primary prolapsus uteri.

(*c*) *The Vagina of "pregnancy"* undergoes increased lengthening, with marked loosening of, and mobility of the parametrovaginum and pelvic floor, a change that is sometimes so marked at term that the vaginal wall in late pregnancy may protrude slightly through the introitus during bearing down even before labor begins.

The simple resorbtion of fat as a cause of this looseness of the lower segment of the pelvic floor in pregnancy as pointed out by Hoffman would be counterbalanced, at least in part, by the elevation of the uterus at this time.

(*d*) *The "Parous" vagina* due to the passage of the term child (especially if large) undergoes an extreme dilatation, and often some submucous tearing, usually much greater in extent in unduly rapid deliveries (due to the pernicious use of ergot, occipto-posterior rotation rapid forceps deliveries, or rapid extraction, not forgetting the lack of moulding in justomajor pelves).

Each succeeding labor can naturally be expected to be the cause of some additional dilatation and perhaps tearing if the children are larger as is frequently the case.

While postpartum involution of both vagina and paravaginum occurs, it perhaps never entirely "involutes" to the shortness and narrowness previous to the last pregnancy, but frequently allows some permanent eversion or prolapsus of the (especially posterior) vaginal wall through the introitus during increased intraabdominal pressure, even though there be no appreciable perineal tear.

This permanent increased vaginal length *per se* is physiologic, for it allows an easier maintainance of the normal elevation of the cervix of the multiparous uterus if anteverted, and prevents undue traction on the cervix in slight vaginal prolapses.

The increased length of the parous uterus also has a prophylactic-value (if normally anteverted) by allowing a longer posterior surface on which the intraabdominal pressure can act.

(8) *The so-called lacerations of the perineum* (*central tendon*) in labor, (levator ani subampulla portion, including the rectovesical and levator ani fasciæ posteriorly; together with the bulbus cavernosus and triangular fasciæ anteriorly) allow the backward traction of the sphincter ani to drag the introitus, *especially the posterior vaginal wall and remains of the perineal tendon* backward toward the coccyx, the tip of which latter also recedes somewhat by pointing

more downward instead of being held firmly in a "near" horizontal direction.

The unopposed sphincter ani muscle also drags the anus more posteriorly and nearer to the receded coccyx, markedly increasing the acuteness of the rectoanal angle, increasing the liability to stagnation of feces in the rectal ampulla, the anterior wall of which becomes atrophic, and in turn causes atrophy, eversion and prolapse of the posterior vaginal wall through the enlarged and retroposited introitus (rectocle).

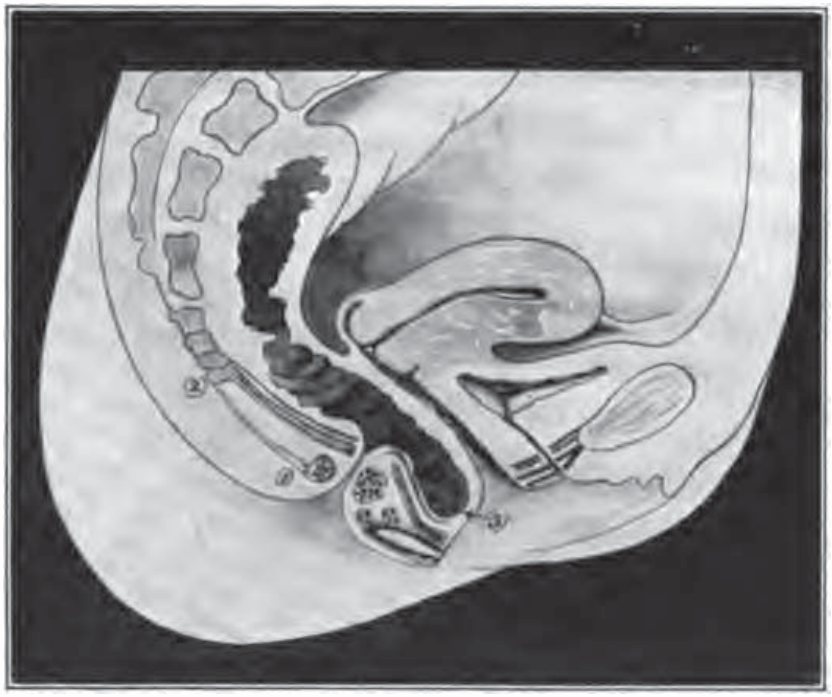

FIG. 3.—Torn central perineal tendon. (1), The unopposed sphincter anus has dragged back the anus and remains of the perineum and posterior vaginal wall nearer to the coccyx; (2), the coccyx itself has also receded somewhat and points more downwards. The ano-rectal angle being more acute increases constipation. The utero vaginal angle is less acute, predisposing to prolapse; (3), the ampulla of the rectum has lost its support and become distended and is protruding more and more through the vaginal introitus as a "*rectocele*" causing traction on the uterus. The support by the posterior vaginal wall to the anterior is also lost, predisposing to cystocele.

The angle between the now "more vertical" vagina and the uterus (even if the latter be normally anteverted) becomes more obtuse and consequently less able to resist the traction on the cervix by the rectocelic vaginal wall which latter becomes more and more prolapsed as the feces increasedly distend the rectal ampulla.

The degree of laceration of the central perineal tendon (submucous or open) usually governs the size of the rectocele which may vary in size from an almond to that of a hen's egg or even larger.

The vagina (at least in the lower third) apparently does not prolapse circularly like a prolapsing rectum, but the anterior and posterior walls prolapse independently, or simultaneously, due to the difference in length and resistance of their independent attachments.

In the dorso-buttocks position, a rectocele till then nonapparent if empty, usually comes strikingly into view when bearing down, showing the characteristically pale smooth vaginal mucosa if of long standing; or if the ampulla be filled with inspissated feces, it may appear at the introitus as a tumor-like enlargement in the posterior vaginal wall.

The little finger inserted in the rectum during bearing down easily passes into the (empty or feces filled) dilated ampulla and causes its further protrusion from the vaginal introitus.

During this manipulation the remaining perineal attachments (if any) of the levator ani muscles and fascia can often be palpated, under the lateroposterior vaginal wall about 3 cm. above the remaining carunculæ myrtiformes by the index-finger of the other hand.

(*b*) *The anterior vaginal wall is said to prolaspse more easily* than the more firmly attached posterior wall, due to its much looser attachment to the bladder (paracystovaginum), and there is no doubt but that the real frequency of *cystocele* is much underestimated because it is more out of sight during examination in the ordinary dorsal-buttocks position.

Cystocele by no means always accompanies nor is accompanied by perineal laceration and rectocele. Some of the worst cystocele occurs in young primiparæ entirely free from posterior vaginal wall or perineal lesion.

By placing the index-finger in the vagina and depressing the posterior vaginal wall, a cystocele (often till then unsuspected) if present will usually come clearly into view, or if large, protrude from the introitus when simply bearing down, as a round (1–3 cm. diameter or longer) enlargement, and is often mistaken by the patient for a prolapsed uterus.

Incontinence on exertion is common if the bladder be full. The incomplete evacuation often present allows the residual urine to become ammoniacal, resulting in catarrhal cystitis, lack of contractility and later still less complete evacuation, and further increase

in the size of the cystocele and consequent increased dragging on the intermediary cervix.

Congenital looseness or lowness or both of the paracystovaginum often has been given as a predisposing cause of cystocele, doubtless aided by the often violent trauma in cases where the thickened anterior uterine labium becomes caught between the descending head and the pubic symphysis with stretching and tearing of the weak parametrovaginocystium.

The lateral traction of the transverse perineal muscles on perineal tears undoubtedly adds to the gaping of the posteriorly drawn introitus vaginæ thereby allowing still easier prolapse of the vaginal walls.

This gaping condition of the vaginal introitus which so often assists in bringing about that distressing condition known as *"Garulitus Vaginalis"* due to the entrance and exit of air on change of intraabdominal pressure when sitting, standing or walking is probably much worse when extensive or complete tears of the triangular ligament occur.

By the accompanying schematic drawing of the "organless pelvis" the author has tried to depict the rectal, uterine, vaginal and bladder (upper floor segment) supporting tissues only.

(6) *The parametrium* is a continuation of that loose connective tissue of the pelvis which extends from the anterior preperitoneal connective tissue to the cavum Retzii, then around the bladder as the *paracystium* then around the cervix and up between the uterus and peritoneum and the leaves of the broad ligaments as the *parametrium*, and down around the upper part of the vagina as the *paravaginum* and backward to the rectum as the *paraproctium*.

It is on the integrity of this loose para-tissue in which the blood and lymphatic vessels and nerves lie and its overlying peritoneum and ligaments that the uterus relies for its correct position in the pelvis.

By virtue of its physiologic looseness the normal mobile retroversion of the corpus and anteposition of the cervix occurs on filling of the bladder and rectum.

In pregnancy this tissue also becomes markedly looser, undergoing involution after parturition. It also contains a certain quantity of loose muscle fiber derived from the external longitudinal layer of the uterus.

Together with the overlying peritoneum it forms (with the exception of the muscular ligamentum teres) the most part of the so-called ligaments of the uterus, *e.g.*, and uterovesical anteriorly;

the broad laterally, and the uterosacral (ligamentum retrahens) posteriorly.

The latter uterine ligaments are in reality the "mesometrium" and very akin to the mesentery of the intestine.

Congenital weakness may be present in the peritoneum itself or in the paratissues or the attachment of the paratissues to the overlying uterus, and underlying vagina and bladder; indeed any

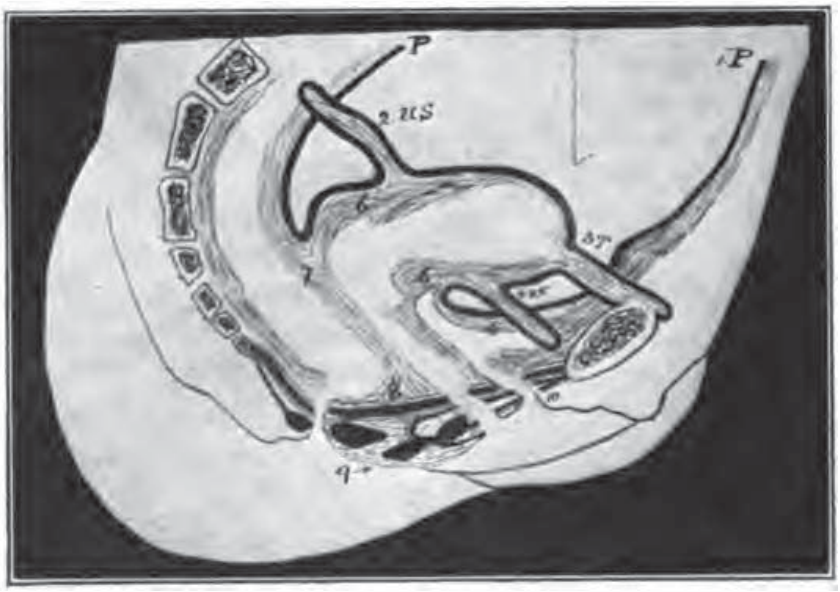

FIG. 4.—The schematic "organless" pelvis and pelvic floor and ligaments in sagital section with only the peritoneum and para-tissues remaining showing spaces originally occupied by the pelvic organs and canals with the normal intact floor muscles and fasciæ. 1 *P*, Parietal peritoneum ligaments; 2 *U.S.*, utero-sacral; 3 *T*, round; (4) *U.V.*, utero-vesical, para-tissues; 5 *V*, vesicum; 6 *M*, metrium; 7 *Pro.*, procteum; 8, levator ani muscles with recto-vesical fascia above and levator fascia below; 9, central perineal tendon meeting place of seven muscles and five layers of fasciæ; 10, the triangular ligaments extending to the central perineal tendon.

or all of these may be illy fitted to withstand abnormal pressure or traction; as is evidenced by the deeper Douglas' sac in one case than in another, as well as by those cases where the anterior culdesac remains at the same level while the cystocelic bladder is entirely stripped off from its uterine and vaginal attachments.

This paratissue being also changed in character as a result of infection especially via the cervical abrasions and tears of labor undergoes acquired changes the after results of which are often firm indurated patches or strong bands, which if on one side and high up in the broad ligament are liable to drag the corpus down in latero-version, or if low down in lateroposition, if behind in retroposition, often elevating the cervix and increasing the anteversion, if anteriorly

in anteposition with an increased tendency to retroversion, or any combination of these.

These infectious parametric changes vary in character and are by no means always Dupuytren-Palmar band-like and strong in every case but may leave behind a very weak extensile tissue entirely devoid of elasticity and invite purely passive cervical anteposition or corpus malversion or indeed prolapse.

The uterus though not strictly a part of the pelvic floor, yet when it normally lies in mobile anteversion at a right or slightly acute angle to the horizontal vagina is undeniably an important factor in spreading the intraabdominal pressure more evenly over the whole pelvic floor and directing it to the sides and bony retroaxial portion.

From the foregoing it is apparent that prolapse of the uterus can be divided according to its etiology and mechanism into three varieties, viz., primary, secondary and combined or mixed.

(1) *Primary prolapsus* alone is prolapse entirely independent of vaginal traction.

(a) In young nulliparæ it occasionally occurs consequent on sudden violent, or oft-repeated increase in intraabdominal pressure, as from lifting weights, or falls.

The vagina in these cases is apparently in the normal horizontal in its lower third, but the upper third of the vagina may often be found bimanually to be situated apparently too far anteriorly for the cervix to be in the interischial-spines line.

Intraabdominally in young nulliparous women one often encounters parametric slackness and long thin uterine ligaments, accompanied by a short vagina and retroversion.

This ligamentous laxity is possibly congenital, or acquired during the exanthemata of childhood, or perhaps due to the ascending infection at the period described by Schultz or an infantile or adult gonorrhea, or infection following an early unrecognized abortion; not forgetting the possibility also of infections, especially from the rectum, less often from the bladder to the parametrium causing weakening of the so-called uterine ligaments.

Whether the infection be peri- or parametric, or both, the resultant condition may be such that immediate restitution to retroposition of the cervix and anteversion of the corpus after the anteposition and retroversion due to an overfilling of the rectum and bladder may be delayed; and the uterus remain longer in temporary retroversion, thereby exposing it unduly to the influence of sudden or frequently repeated increase of intraabdominal pressure on its fundus and anterior surface.

Again the filling of Douglas' sac by the often very long feces-filled, long mesenteried sigmoid in chronically constipated women, undoubtedly presses the cervix forward in anteposition and favors retroversion even though the bladder be regularly emptied, and may indeed gradually stretch even healthy uterosacral ligaments.

The fact that kyphotic girls often suffer from prolapse, would make it appear as if the lessened intraabdominal area and presumable increase in the intraabdominal pressure was a very important factor in the etiology.

The author of this paper has noted many nulliparæ in the Presbyterian and Cook County Hospitals with retroversion and a certain tendency to prolapse of the uterus that easily on unduly violent exertion might cause such a rapid lowering of the cervix as to give the idea that it was of sudden, instead of gradual occurrence.

(*b*) *In the parous woman* a slight physiologic primary prolapsus uteri is very common. Every woman who has borne a child, is liable to have a slight physiologic anteposition of the cervix, and some physiologic lowering of the uterus, compared to the normal nulliparous condition.

In many multiparæ the portiovaginalis tends to point more downward in the axis of the vagina, even though the corpus uteri is still maintained in anteversion.

Possibly the larger multiparous corpus is more easily maintained in anteversion by the action of the intraabdominal pressure on its longer posterior surface, or by the better developed (parous) teres and uterovesical ligaments anteriorly, or both.

Later the uterus may become retroverted, and possibly the prolapse increase, but examination of these cases month after month, and indeed year after year, often shows the anteversion still well maintained.

The etiology of this slight physiologic primary prolapsus of multiparæ with anteversion, is easy to attribute to a subinvolution of the whole posterior parametrium, and is not due to a slackening of the uterosacral ligaments alone, causing a loss *of the "centre basculer."*

Besides the integrity of the anterior ligaments, doubtless adhesions or tumor or other cause, may also assist in maintaining the anteversion in some of these cases.

The faith placed in the round ligaments alone as supports capable of preventing cervical anteposition, corpus retroversion and prolapsus uteri, and the amount of ingenuity shown in the invention of round ligaments (shortening) operations is absolutely unwarranted.

The author believes that more attention should be paid to combining with it, uterosacral ligament shortening and other procedures to restore the cervix to its normal distance from the sacrum as demonstrated by Dudley, of Chiago, especially when we consider the complex etiology.

(2) *Secondary prolapse* of the multiparous uterus perhaps erroneously considered by many authors as the commoner form, *i.e.*, secondary to (lower third) vaginal prolapsus and traction on the cervix by the vaginal wall consequent on "perineal" tears and retroposition of the remains of the perineum and enlarging rectocele, or cystocele or both.

The prolapsing lower vaginal walls (rectocele and cystocele) gradually drag the uterus down unaided or aided by intraabdominal pressure especially if the uterine ligaments and parametrium be relaxed and the uterus be retroverted and free from adhesions.

Though cases of prolapsus uteri do occur with the uterus at the introitus vaginæ still in anteversion, it by no means minimizes retroversion as a predisposing factor but accentuates the greater need of considering the parametrovaginum and uterosacral and other ligaments.

The size of the introitus is important, but prolapse occurs where the introitus vaginæ is narrow and often no prolapse where it is wide and gaping. If the introitus is narrow, the anterior vaginal wall and cystocele is often found to be the one making the traction; where the perineum is torn and rectocele is present and the introitus gaping the posterior vaginal wall often either acts alone, or in conjunction with the anterior.

(3) *Combined Primary and Secondary Prolapse of the Uterus.*—From the foregoing remarks both the causative factors (primary and secondary) can easily be understood to be present in the majority of the cases formerly considered to be entirely secondary to vaginal prolapse. That the uterus in cases with an enlarging rectocele causing vaginal traction on the cervix is frequently but poorly held up in position by a congenitally weak parametrium and ligaments must be conceded by all clinicans.

Again the frequency of acquired prolapse of the uterus among the poor and hardworking multiparæ is, not without some reason, often attributed to too early rising (third or fourth day) after childbirth and the early violent increase of intraabdominal pressure and vaginal traction while the enlarged puerperal uterus is still in postpartum retroversion and while the parametric (ligamentous) involution is still incomplete.

One cannot but feel that the application of forceps, (so common in this country) if applied before the os is dilated, is to blame for much irreparable tearing and weakening of the parametric tissues as well as the lacerations of the cervix, opening up the way for an "infection" weakening of these tissues as stated under primary prolapse.

The author believes from intraabdominal observation that in many multiparous women with very long intestinal mesenteries the intraabdominal pressure as a causative factor is far in excess of that generally conceded.

Indeed with a weakened parametrovaginum the increased action of the intraabdominal pressure in these cases is probably a not uncommon factor in pushing the entire upper portion of the pelvic floor as a whole down to a lower level even when no prolapse has as yet occurred.

Again it is not uncommon after the menopause to find the senile atrophied and shortened uterus till then anteverted, apparently dragged down by the senile atrophy of the paratissues and shortening of the vagina.

It would also appear as if the senile shortening of the uterus allowing less length of surface for intraabdominal pressure to act upon, was at least in part responsible.

Lastly the power of vaginal traction as a factor in prolapse is well shown in those cases where the uterus is fixed in the pelvis, *e.g.*, by adhesions or tumor or other cause; by the traction on the cervix by the "celic" vagina resulting in a ***true elongation of the cervix*** while the corpus and fundus uteri remain at, or nearly at, the normal level in the pelvis.

In these latter the anterior vaginal wall traction has its effect mostly upon the intermediary cervix with but little deepening of the vesicouterine culdesac while the ***posterior vaginal wall*** traction being principally on the supravaginal portion of the cervix drags the Douglas' peritoneal sac down with it.

PEOPLE'S GAS BUILDING.